International Journal of
Environmental Research and Public Health

Special Issue

Substance and Behavioral Addictions: Co-Occurrence and Specificity

2012

Steve Sussman (Ed.)

MDPI
OPEN ACCESS

Cover picture: Ihr zu Füßen!
Author: Gustav Imlauer
In: *Über Land und Meer. Allgemeine illustrirte Zeitung*, H. 13, 1883, S. 265
Available online at:
simple.wikipedia.org/wiki/File:Imlauer_Ihr_zu_Fuessen_1883.jpg

ISBN 3-906980-32-4

Table of Contents

Preface

Researchers and practitioners have discussed the existence of several types of substance and behavioral addictions. That is, in general, they have agreed that individuals may fall victim to maladaptive, repetitive patterns of behavior involving recreational drugs (e.g., tobacco, alcohol, illicit drugs), other substances (e.g., binge eating), and other behaviors (e.g., gambling, the internet, shopping, workaholism, exercise addiction, love, or sex), that reflect attempts at achievement of appetitive physiological outcomes but result eventually in negative outcomes to self or others. Research over the last two decades suggests that a wide range of substance and behavioral addictions may serve similar functions. Overall, 12-month prevalence of one or more of the 11 addictions just listed above among adults in the U.S. has been estimated to be 46% based on an exhaustive review of the literature [1]. As such, it may be useful to think of the addictions in terms of problems of lifestyle as well as of a person. Yet, “co-occurrence” of addictions has been reported among only a minority of sufferers, and is estimated to be approximately 23% [1]. “Addiction specificity” pertains to a phenomenon in which one pattern of addiction may be acquired whereas another is not [2]. Differential patterns of addiction may be a function of such variables as accessibility, intrinsic appetitive effects, differential socialization, and specific outcome expectations. The present Special Issue examines addiction co-occurrence and addiction specificity across several addictive behaviors. The goal of the Issue is to comprehensively describe notable addictive behaviors, and elaborate on what the associational pathways might be of addiction co-occurrence and specificity.

A total of 11 papers are included. The first paper by Sussman *et al*. [2] introduces the topic of the Special Issue and presents a model that might help explain addiction specificity (the “PACE” model). Pragmatics (e.g., access), attraction (e.g., subjective effects), communication (e.g., addictive behavior jargon), and expectation (e.g., number of experiential solutions provided by a given addictive behavior) variables are described, and examples in the development of different addictions are derived from this model. The second paper by Blum *et al.* [3] presents literature on the Reward Deficiency Syndrome, and how it might apply to various addictions and other problem behaviors as a nonspecific reward phenotype. Further, these authors present results of a genotype analysis which indicates extremely high presence of certain gene alleles (e.g., TacA1) across two families with high prevalence of the phenotype. Their article highlights more an underlying potential mechanism of addiction co-occurrence which might fall mostly within the “A” (attraction) facet of the PACE model.

The third paper by Griffin *et al.* [4] examines the prospective relationships of alcohol use and sexual behavior among urban minority women from the beginning of high school to young adulthood, eight years later. Self-control was found to serve as a protective factor of latent class membership ― those who would end up exhibiting low levels on both behaviors, high levels of alcohol use-low levels of risky sexual behavior, or high levels on both behaviors. This paper

highlighted both the operations of addition co-occurrence and specificity and one potential determinant (lack of self-control; also an “A” variable). The fourth paper by Freimuth *et al.* [5] provides one of the first reviews of exercise addiction, how it might develop, and its developmental specificity (e.g., reliance on exercise for its mood altering effects) and co-occurrence (e.g., with eating disorders, alcohol use). Treatment implications of exercise addiction also are discussed.

The next three papers pertain to internet addiction. The fifth paper by Bergmark *et al.* [6] provides data from a large Swedish survey on internet use (mean age of sample = 45 years). It is observed that while face-to-face contact is decreased due to internet use, total number of social contacts appears to increase. Addiction-type problems pertaining to internet use appear to be a function of engaging in time intensive activities such as online gaming. Internet addiction may be best understood as being a modality in which other types of addictions (e.g., gambling, sex) are expressed. The sixth paper by Sun *et al.* [7] presents data from Chinese and U.S. adolescents on the bidirectional one-year prospective relationships between compulsive internet use and drug use (cigarette smoking and alcohol binge drinking). Each behavior predicted itself over time. In addition, compulsive internet use positively predicted later smoking or binge drinking among females but not males, in both countries. The authors speculate that females that take on male gender roles, possibly through on-line gaming, are at relatively greater risk for drug use (illustrating both addiction co-occurrence and specificity). The seventh paper by Kuss *et al*. [8] is the first review paper on social network sites (SNS) addiction. While SNS are used mainly for maintenance of offline pre-established social networks, relatively high levels of use are related to decrease in real life social participation, relationship problems, and academic achievement, suggestive of addiction. There may be co-occurrence of SNS addiction with drug misuse and overeating.

The next three papers pertain to playing videogames. The eighth paper by Pentz *et al*. [9] examines the relations among high calorie, low nutrient (HCLN) intake and videogaming among a large sample of initially 9-to-10 year olds over an 18 month period. Male gender, free/reduced lunch, low perceived physical safety and low inhibitory control predicted both behaviors, whereas ethnicity and low stress predicted HCLN intake. Possibly, staying indoors (that may characterize some poor areas), may reflect a “P” (pragmatics) variable conducive to both behaviors. The ninth and tenth papers, both by Ream, Elliott, and Dunlap [10,11] were drawn from a nationally-representative survey of U.S. adults who regularly or occasionally played videogames and had played at least one hour in the past week. These authors found that problem videogame playing and substance use problems involved the same types of motivations: to pass time/regulate negative emotion, enhance positive emotion, or balance each type of behavior out (videogame play *versus* use of a substance). In their other paper, they found that (controlling for the shared variance of problem substance use (caffeine, tobacco alcohol, or marijuana) and problem videogame playing) videogame playing while concurrently feeling the effects of a drug

was uniquely associated with problem substance use, but not with problem levels of videogame playing. That is, it appeared that concurrent activity was fundamentally related to substance use difficulties but not the converse.

The final paper in this Special Issue, by Sussman and Sussman [12] revisited the definition of addiction. These authors suggested that cycles of engaging in addictive behaviors to attempt to satiate an appetitive effect that become associated with preoccupation, loss of control and negative life consequences, were the criteria that defined addictive behaviors. They also suggested that there were philosophical concerns regarding the relations of these criteria with each other and with the concept of addiction. Taken together, it is hoped that these 11 papers assist in the arduous task of moving the study of addictive behaviors forward through considering pathways and associations of the many types of behaviors that might encompass, at least in part, the same types of underlying mechanisms.

Steve Sussman, Ph.D.
Guest Editor

References

1. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the Addictions: A Problem of the Majority or the Minority. *Eval. Health Prof.* **2011**, *34*, 3-56.
2. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A Framework for the Specificity of Addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
3. Blum, K.; Chen, A.L.C.; Oscar-Berman, M.; Chen, T.J.H.; Lubar, J.; White, N.; Lubar, J.; Bowirrat, A.; Braverman, E.; Schoolfield, J.; *et al.* Generational Association Studies of Dopaminergic Genes in Reward Deficiency Syndrome (RDS) Subjects: Selecting Appropriate Phenotypes for Reward Dependence Behaviors. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4425-4459.
4. Griffin, K.W.; Scheier, L.M.; Acevedo, B.; Grenard, J.L.; Botvin, G.J. Long-Term Effects of Self-Control on Alcohol Use and Sexual Behavior among Urban Minority Young Women. *Int. J. Environ. Res. Public Health* **2012**, *9*, 1-23.
5. Freimuth, M.; Moniz, S.; Kim, S.R. Clarifying Exercise Addiction: Differential Diagnosis, Co-occurring Disorders, and Phases of Addiction. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4069-4081.
6. Bergmark, K.H.; Bergmark, A.; Findahl, O. Extensive Internet Involvement—Addiction or Emerging Lifestyle? *Int. J. Environ. Res. Public Health* **2011**, *8*, 4488-4501.

7. Sun, P.; Johnson, C.A.; Palmer, P.; Arpawong, T.E.; Unger, J.B.; Xie, B.; Rohrbach, L.A.; Spruijt-Metz, D.; Sussman, S. Concurrent and Predictive Relationships Between Compulsive Internet Use and Substance Use: Findings from Vocational High School Students in China and the USA. *Int. J. Environ. Res. Public Health* **2012**, *9*, 660-673.
8. Kuss, D.J.; Griffiths, M.D. Online Social Networking and Addiction—A Review of the Psychological Literature. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3528-3552.
9. Pentz, M.A.; Spruijt-Metz, D.; Chou, C.P.; Riggs, N.R. High Calorie, Low Nutrient Food/Beverage Intake and Video Gaming in Children as Potential Signals for Addictive Behavior. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4406-4424.
10. Ream, G.L.; Elliott, L.C.; Dunlap, E. Patterns of and Motivations for Concurrent Use of Video Games and Substances. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3999-4012.
11. Ream, G.L.; Elliott, L.C.; Dunlap, E. Playing Video Games While Using or Feeling the Effects of Substances: Associations with Substance Use Problems. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3979-3998.
12. Sussman, S.; Sussman, A.N. Considering the Definition of Addiction. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4025-4038.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A Framework for the Specificity of Addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415; doi:10.3390/ijerph8083399.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

A Framework for the Specificity of Addictions

Steve Sussman [1,*], Adam Leventhal [1], Ricky N. Bluthenthal [1], Marilyn Freimuth [2], Myriam Forster [1] and Susan L. Ames [3]

[1] Departments of Preventive Medicine and Psychology, University of Southern California, California 90032, CA, USA; E-Mails: adam.leventhal@usc.edu (A.L.); rbluthen@usc.edu (R.N.B.); myriamforster@msn.com (M.F.)

[2] Clinical Psychology, Fielding Graduate University, Santa Barbara, California 93105, CA, USA; E-Mail: mfreimuth@fielding.edu

[3] School of Community and Global Health, Claremont Graduate University, Claremont, California 91711,CA, USA; E-Mail: susan.ames@cgu.edu

* Author to whom correspondence should be addressed; E-Mail: ssussma@usc.edu; Tel.: +1-626-457-6635; Fax: +1-626-457-4012.

Received: 24 July 2011 / Accepted: 5 August 2011 / Published: 18 August 2011

Abstract: Research over the last two decades suggests that a wide range of substance and behavioral addictions may serve similar functions. Yet, co-occurrence of addictions has only been reported among a minority of addicts. "Addiction specificity" pertains to a phenomenon in which one pattern of addictive behaviors may be acquired whereas another is not. This paper presents the PACE model as a framework which might help explain addiction specificity. Pragmatics, attraction, communication, and expectation (PACE) variables are described, which may help give some direction to future research needs in this arena.

Keywords: addiction specificity; PACE model

Int. J. Environ. Res. Public Health **2011**, *8*

1. Introduction: Addiction as a Biopsychosocial Phenomenon Involving a Range of Different Behaviors

For many years, researchers and practitioners have discussed various seemingly irrational behaviors that exhibit patterns of self-destruction similar to drug abuse [1-8]. Indeed, the concept of "addiction" has broadened in scope from referring to only physiologic processes related to drug misuse (pharmacodynamic tolerance and withdrawal) to a more elaborate biopsychosocial syndrome with commonalities across several behaviors. An overarching feature of the addictive process includes compulsively performing a behavior, for example, continuous drug taking, binge eating, gambling or working [2,4,9-14].

During the "addictive process" [2,4,5] initially one may pursue some course of action for appetitive effects such as pain reduction, affect enhancement, arousal manipulation, or fantasy. Over repeated engagement in the behavior, the individual becomes intensely preoccupied with the behavior despite diminishing appetitive effects [15,16]. Subsequently, the individual, if desiring to control or stop the behavior, experiences subjective loss control over when the behavior is initiated, how it is manifested, or when it will stop. Finally, one incurs negative consequences (e.g., social, role, physical, emotional) while continuing to engage in the self-defeating behavior. Stopping the behavior becomes difficult for several reasons, including having a lack of awareness of the "stimuli" or triggers that influence the behavior and the cognitive salience of immediate gratification relative to delayed adverse effects. That is, the behavior becomes increasingly more automatic and less under one's control-ability [17-20]. At this point, the individual also may fear having to cope with day-to-day perceived stress and other life experiences upon cessation (possibly due to accumulation of addiction-related consequences, or having to endure "raw" emotional experiences without concurrent self-medication [5]), as well as having to suffer withdrawal-related phenomena [4,11,14]). Various substance and process/behavioral addictions appear to be intricately connected in terms of etiology, patterns of behavior, and consequences [12,21].

1.1. Patterns of Addiction Co-Occurrence

While it is not entirely clear what differentiates addictive-prone from non-addictive prone behaviors [22], Sussman, Lisha and Griffiths [14] identified 11 relatively common behaviors that appear to have addiction propensity (tobacco use, alcohol use, illicit drug use, binge eating, gambling, internet use, love, sex, exercise, work, and shopping). That article reported the prevalence and co-occurrence of these behaviors based on a systematic review of the literature. Data from 83 studies (each study *n* equal to or greater than 500 subjects) was presented and supplemented with smaller-scale data. The authors noted a 23% average co-occurrence among the 11 addictions (with a range from 10% to 50% overlap among 110 pairs of co-occurrence examined), and determined that approximately 50% of the U.S. adult population has suffered from signs of some type of addictive behavior over a 12-month period, controlling for co-occurrence. Although there are some methodological limitations [14], their findings suggest that there is marked variability in the pattern of addiction co-occurrence. As examples, addictions to cigarettes, alcohol, and illicit drugs are highly associated with each other and with sex and love addiction, which also are highly associated with each other. In addition, gambling addiction is strongly associated with cigarette smoking addiction but not as much with illicit

drug abuse or dependence. Exercise addiction is moderately associated with eating, gambling, work, and shopping addictions but is more weakly associated with cigarette, alcohol, and illicit drug addictions [14].

One approach to classifying patterns of addiction co-occurrence purports three categories of individuals: (1) people who experience multiple addictions concurrently [11,14,23,24]; (2) people who experience substitute addictions; that is, where one addiction takes the place of a previously terminated addictive behavior in order to serve the same functions [11,25]; and (3) people who experience only one addiction in their lifetime [14]. However, even within the broad categories of multiple and substitute addictions, there is likely to be considerable differentiation; that is, individuals differ in the functions of addictions they suffer. For example, as was found among a sample of 543 mostly adult consecutive admissions to an addictions treatment center, addiction clusters appear to divide most generally into "hedonistic" (excitement/dominance motives, such as drug use, sex, love/relationship, gambling) and "nurturance" (providing for self or others motives, such as food, shopping, work, exercise) types of addictions [23].

Also, there has been some focus in the literature towards identifying the factors that explain a tendency for some individuals to develop co-occurring addictions [12,21,23,26,27]. For example, Carnes, Murray and Charpentier [21] presented ten different models of co-occurring addictions that they labeled "Addiction Interaction Disorder," based on self-reported experiences of 1604 adult sex addicts. They identified several processes that account for co-occurring addictions, such as "cross tolerance" and "masking." Cross tolerance occurs when one addiction causes a pre-existing tolerance to a second addiction such that the effects of the new addiction are dampened. "Masking" is where one addiction provides an alibi for another addiction (e.g., such as getting drunk prior to engaging in anonymous or casual sex). By contrast there has been relatively little attention directed towards explaining why some individuals are prone to some addictions, but not others (*i.e.*, addiction specificity).

1.2. Addiction Specificity and the PACE Model

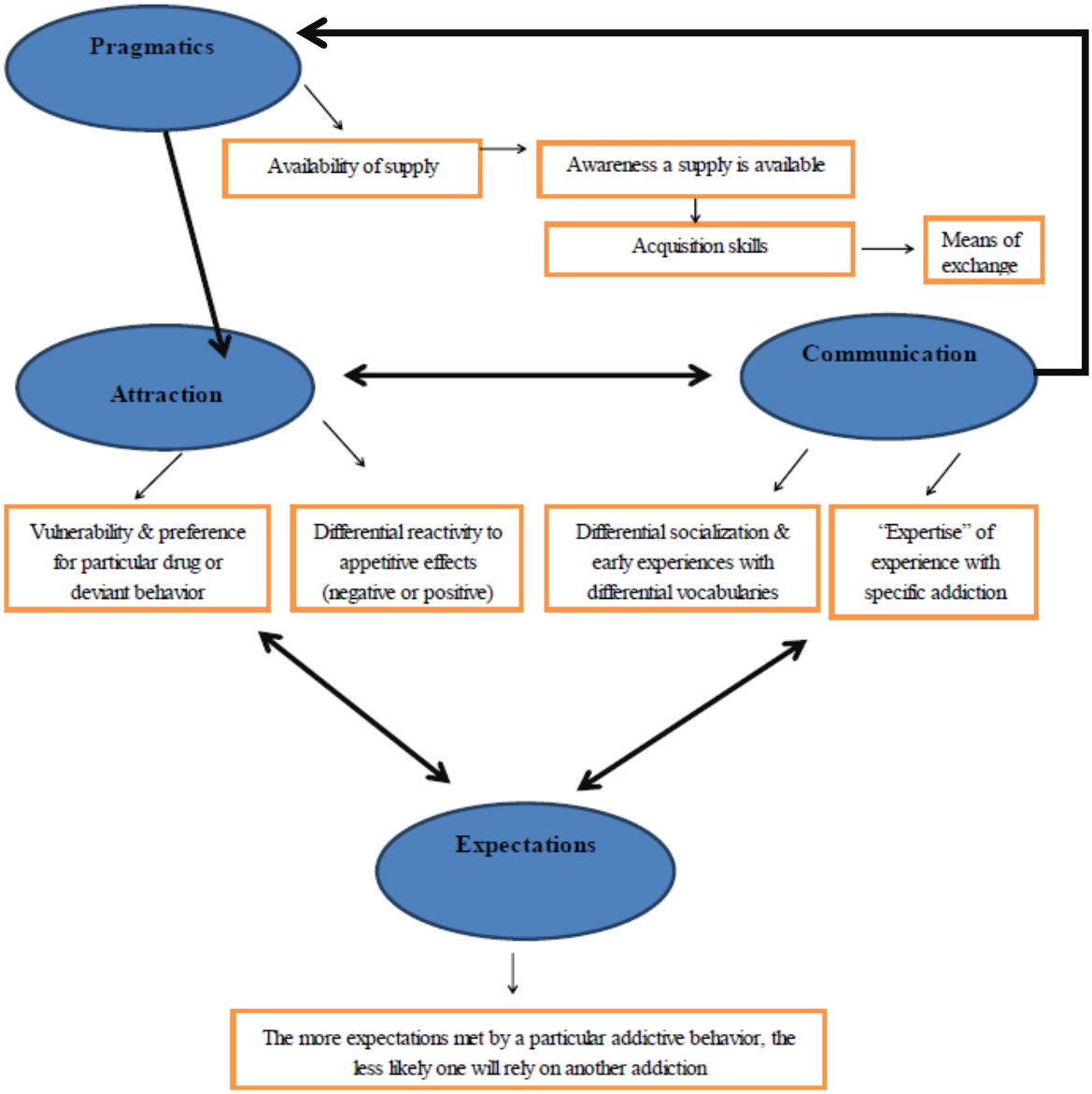
A concept that pertains to why some addictions may not co-occur within individuals may be labeled as "addiction specificity." Addiction specificity is a phenomenon complementary to addiction co-occurrence. Overall, different people appear to show unique patterns of addiction and, while they struggle with one or more addictive behaviors, they may not have difficulty with other potentially addictive behaviors. There are people, for example, who develop problems with drugs and sexual behavior but who never experienced difficulties with gambling (e.g., never lose much money when gambling, do not gamble long hours or lose control of their gambling behavior.) The epidemiology of addiction specificity across substance and process addictions has not been conclusively quantified, though some good work on addiction clusters is now being examined and may pertain to addiction specificity as well as co-occurrence, see [23]. As previously mentioned, Sussman, Lisha, and Griffiths' [14] review on addiction co-occurrence provides emerging support that patterns of addiction specificity are relatively prevalent.

To date, no one model has been utilized to explain addiction specificity that considers the interplay between biological, environmental, situational, and learning factors. Although any individual may be

Int. J. Environ. Res. Public Health **2011**, *8*

susceptible to developing an addiction, it is unlikely that one's genetic profile alone could determine specificity of addiction. Nevertheless, genes influence neurobiological systems and, in turn, responses to reinforcers, and play a role in susceptibility to various appetitive behaviors, see [1,3,28,29]. For example, an individual may inherit a susceptibility to feel shy and then feel much better in social situations while engaging in some appetitive behavior, like alcohol use, possibly leading eventually to alcoholism [30]. However, acquisition of specific addictions *versus* others also involves exposure to unique social environmental experiences, and associative learning and memory processes resulting from those experiences, possibly leading to different behavioral phenotypes [18,31-33]. A comprehensive model of addiction specificity should consider the interplay among these variables, all which impact one's tendency toward one addiction or set of addictions *versus* another.

Figure 1. Diagram of the PACE model.



Int. J. Environ. Res. Public Health **2011**, *8*

The PACE model was originally proposed by the lead author to explain the development of intimacy in relationships between two people from within the interpersonal attraction literature in social psychology [e.g., 34]. The variables that were formed from the interpersonal attraction/relationship development literature include residential propinquity and other practical variables which permit access to relationship development ("pragmatics"); physical attractiveness, speech tone, and personal habits which comprise how initially appealing persons will be to each other ("attraction"); sharing a common understanding of each other and of everyday experience ("communication"); and having cooperative expectations of each other ("expectation" [34]). Later on, the model was adapted and published as a means to describe the development of a relationship of a person with drug use [35] and, indeed, there are several similarities between the development of an entrenchment in an addictive behavior and the development of an intimate relationship [36]. As detailed below, this model provides a useful framework for understanding general processes that underlie specificity in the initiation and maintenance of addictive behaviors. Figure 1 depicts the current conceptualization of the PACE model.

2. Results and Discussion

2.1. Pragmatics

Pragmatics variables operate to discern whether or not one can access a particular addictive behavior and then engage in this behavior regularly. Pragmatics involves four aspects. First, there must be a *supply* of the object of the addiction available in the environment (e.g., drug distribution point, gambling casino, brothel, potential love partner, workplace, gym). If not, no relationship with the "addiction object [behavior]" can develop. Case in point, addiction to the internet was not possible prior to wide availability of the internet [14]. Objects of addiction tend to be available along distribution routes, which permit easiest passage from a manufacture/product/service origin point and where (consequently) there tends to be higher consumer demand [8]. Changes in availability of an addiction object can increase or decrease prevalence of addictive behavior. At a macro-geographical level, the explosion of crack cocaine use in the late 1980s in the United States or decline in heroin supply and use in Australia and the west coast of Canada are but two examples of this common phenomenon [37-39]. At a micro-geographical level, distance from an addiction source or supply is associated with overall prevalence of the behavior as well as disordered forms of the behavior (e.g., regarding alcohol use and abuse [40]; regarding gambling and problem gambling [41]). Nevertheless, if the addiction object is available, then other pragmatics aspects must be considered.

Second, one needs to be *aware* that there is a supply of the addiction object [service] available. In fact, perceived availability of the addiction object may be a more important predictor of behavior than objective measures of availability [40]. Promotion of the addiction object reaches the potential consumer by way of any number of channels (e.g., word of mouth, observation of sales, public venues such as clubs or bars, television advertisements, provider web sites, or even early evening news stories). "Channels of introduction" to the addictive behavior likely contain cues specific to that behavior and begin a process of differential exposure to and learning of information related to the context of the addiction [42], this perhaps being the earliest aspect of addiction specificity. For

example, beer advertisements and packaging may indicate where to purchase the product, suggest that when one drinks beer one drinks multiple beers on a drinking occasion, and suggest how much of the addiction object to purchase [8].

Third, an individual must have *acquisition skills*; that is, one needs to know how to obtain the addiction object from the source. An individual needs to be able to converse appropriately with people who possess the addiction object (e.g., drug, sex), how to bring up topics without being threatening (e.g., cost, location, type of service), and how to arrange an exchange (usually money for the object). Finally, an individual needs to have a *means of exchange*; that is, possess money or services to offer in return for the addiction object. For example, one can pay for a drug, provide a service as a drug transporter, or offer sexual favors, as means to procure one's drug of choice.

The means by which pragmatics influence addiction specificity are relatively intuitive—if the pragmatic variables are favorable to a trial of a potentially addictive behavior, the behavior is more likely to be initiated. The few current statements on specificity of addiction emphasize the importance of access and exposure to the addiction [12,27]. Of course, for some objects of addiction, such as food/binge eating, the pragmatics involved may render the behavior as a relatively easy one in which to engage, whereas some objects of addiction, such as heroin use, may be a relatively difficult one in which to engage [38,39]. However, many people have tried a variety of objects of addiction at least once. For example, by 12th grade 72% of youth in the U.S. have tried alcohol (55% have reported ever being drunk), 45% have tried cigarette smoking, and 47% of youth have tried an illicit drug (25% have tried an illicit drug other than marijuana [43]). A vast majority of the U.S. adult population (over 86%) have tried gambling at some time in their lives [44]. Most people have purchased shopping items "on impulse," roamed the internet for a substantial amount of time, and looked at an erotic photo.

Situational opportunity and curiosity predict that a particular addictive behavior will be engaged in at least once. However, it is doubtful that pragmatics *per se* is the critical factor that leads to addiction specificity, particularly if multiple channels of addiction are readily available. It is likely that other processes are critical in channeling the transition from initiation of behavior to escalation, maintenance, and excessive or compulsive engagement in a specific addictive behavior or set of behaviors.

2.2. Attraction

Attraction plays an important role in addiction specificity by impacting whether someone is likely to initiate and then continue engaging in an addictive behavior. Numerous variables can shape what determines if a behavior is attractive. These include individual difference variables that may influence selection of the addictive behavior. For example, some addictive behaviors (e.g., heroin, involving needle use) may be more normatively stigmatized [45] and, hence, less attractive to many persons. However, those relatively vulnerable to engage in such behavior may prefer relatively stigmatizing addictive behaviors as a *prima facie* expression of defiance [14,27]. More specifically, persons attracted to relatively stigmatized behaviors such as heroin injection may initially intensely enjoy the reputation they obtain (e.g., deviant peer group credibility), or the reactions to their behavior that they observe from others, as being beyond the chains of social restraint, expressed in the addiction [45,46]. These individuals also may be less attracted to addictive behaviors that are more socially

acceptable (e.g., shopping, internet). Conversely, those who are attracted to relatively deviant addictive behaviors may be interested in engaging in relatively deviant manifestations of other addictions. As examples, they may favor shoplifting as a form of shopping addiction or may become a workaholic sex worker (*i.e.*, work long hours at a relatively "extreme" job). Of course, those individuals who are relatively less enticed by deviance might be attracted to fewer types of addictive behaviors [1,3,47].

Individual differences in the initial acute reinforcing effects of addictive behaviors can shape one's attraction to these behaviors [1,3,15,16,48,49]. Indeed, there is marked between-person variability in the acute effects of a variety of addictive behaviors [50,51]. That is, for some individuals a behavior can result in extremely pleasurable experiences (e.g., high, rush, relaxation, stimulation, social and performance enhancement). For others the same behavior can result in severe aversive effects (e.g., anxiety, undersired sedation, social and performance impairment, dysphoria), or relatively few or weak acute effects (neither positive nor negative). For example, some East Asians have a gene variant that produces an enzyme that inadequately breaks down alcohol's initial metabolite, aldehyde dehydrogenase, and hence, they tend to experience uncomfortable physiological reactions such as a flushing response, nausea, and headaches when drinking alcohol. Thus, East Asians may be less attracted to using alcohol in comparison to other substances, such as marijuana or nicotine [27].

Certain intrapersonal traits may impact initial sensitivity to specific addictive behaviors. Anhedonia—the incapacity to experience pleasure in response to natural rewards—is unlikely to increase propensity for behavioral/process addictions that produce relatively less positive reinforcing effects (e.g., sex, shopping [52]). By contrast, anhedonia is associated with increased sensitivity to the euphorogenic effects of stimulant drugs (e.g., amphetamine and cocaine [53,54]). This variation in enjoyment of different drugs or of other addictive behaviors may be similar to notions about using drugs as a means of self-medication [55,56] or for satisfying a biologically-based desire for stimulation as in sensation seeking [57,58].

Attraction also involves the experiential pleasantness ascribed to addictive behavior-related stimuli and context. That is, one may feel attracted to the sight, smells, sounds, tactile stimulation, or social stimuli inherent in the context of the addiction [10]. For example, researchers and practitioners have noted that drug addicts appear to become addicted to the routine of preparing and administering the drug, and contextual cues associated with the drug [6]. Overtime, and through associative learning and memory processes, contextual stimuli may come to represent appetitive effects associated with the behavior [18,33,59], affecting attraction to the behavior itself. Interestingly, accidental circumstances may lead to avoidance of or preference for that addictive behavior [60]. For example, acting in a highly shameful way or experiencing pain following an accidental fall while using marijuana for the first time may cue one to avoid its use, though not the use of other drugs. It is important to note that external cues for an addictive behavior may be unique to that addictive behavior, and, hence, related behavior-specific urges would be elicited in response to those external cues as well as the addiction object [6,61].

In some instances, the shaping of addiction specificity may involve extended access and involvement with a particular addictive behavior during a critical point in childhood or adolescence which may facilitate an intense attraction toward the behavior. Neural adaptations may be especially likely when one is most neurobiologically vulnerable during adolescence. It is at this time that there exists relatively few higher-level inhibitory functions monitoring relatively greater motivational drive

for novel experience and this may affect the course of an addictive behavior [62]. During adolescence some subcortical structures mature earlier and are more able to support the acquisition of appetitive-type behaviors [63,64]. Thus, the behavior may be maintained by early maturation of brain structures able to support the behavior without executive inhibitory control processes overriding the behavioral tendencies [20].

Between-person differences in disliking cessation of an addictive behavior also may be important for explaining the specificity in whether or not one maintains an addictive behavior after a habitual pattern is already established. For example, there are reports of marked individual differences in the severity of withdrawal symptoms following discontinuation of an addictive behavior [65]. It is possible that an individual has a greater propensity to experience severe withdrawal after abstaining from one addictive behavior compared to another behavior [66]. In this case, he or she is likely to continue one type of behavioral pattern to avoid severe withdrawal, and as a result, manifests addiction specificity.

2.3. Communication

People tend to select social and physical environments that are similar to earlier experienced environments, which may shape life experiences in part by repetition of learned patterns of communication (e.g., Life Course Theory [67]). For example, youth who early-on have learned to express anger-related words or cuss words are relatively likely to expose themselves to persons and situations that involve risky behaviors including addictive behaviors such as drug misuse, gambling, or sexual behavior [8,68]. Further, it is possible that earlier life experiences, by perpetuating differential communications associated with addictive behavior, may prepare people for which types of addictions they pursue [42]. That is, early experiences with differential vocabularies can direct behavior toward specific addictive behaviors. For example, observing older siblings engaging in marijuana use may teach one the language associated with marijuana use (e.g., lighters, matches, bongs, rolling papers, pipes, or head highs *versus* body highs, inhaling), preparing one for how to use marijuana when one is older [69,70]. At the same time, if one does not learn the language associated with another addictive behavior and, hence, does not tend to think in terms of the language of the other addictive behavior (e.g., gambling addiction: bet, action, call, payout, all-in, ante, an arm, wad; [71]), then communication becomes engrained specific to one addiction (marijuana) but not another (gambling).

There are several avenues by which communication processes may contribute to addiction specificity. Some people may originate from cultural backgrounds that cause them to feel comfortable or uncomfortable with taking part in the communication processes pertaining to a particular addiction, or lead them to be potentially unaware of words associated with the addiction. For example, Latter Day Saint or Baptist church members tend to avoid alcoholism or tobacco addiction and, in general, may be relatively likely to avoid discussion of these drugs [72].

As one continues to engage in an addictive behavior, a relationship develops that involves seeking, experiencing, and recovering from the effects of the addictive behavior. A system of communication about these aspects of the addiction may develop, encompass important features of one's daily life, and call upon quite distinct personal and intergroup communication styles and techniques. For example, buying a drink in a bar requires different interpersonal communication skills than purchasing an ounce of cocaine from a dealer. The interactions among drinkers occur within the continuum of accepted

social practices where both distributor and consumer often operate within the law (depending on other variables such as if the customer is "cut off" at some point in drinking, drugs are permitted at a bar, or whether drinking and driving are involved). In contrast, cocaine use may place users in jeopardy of physical aggression or theft from peers, and both users and dealers may incur legal consequences, facilitating perhaps a different interactional style including "code words" to arrange a buy (e.g., someone may request buying a "cup of soup" to indicate one "rock" of crack [73]). In general, "insider speech" may develop to serve as a symbol of commonality and group identification pertaining to specific addictions within specific contexts [74].

As one becomes differentially socialized, one may become an "expert" in the language of the addiction and feel like a "regular" or someone who belongs in that context. One may comprehend addictive behavior-specific words that associate the behavior with life experiences and show an understanding of the language of the behavior (e.g., "4:20" is jargon that refers to marijuana use in the United States by many experienced users: the time of day to use, marijuana appreciation day; "hand release" refers to a sex worker bringing a client to orgasm by using a hand, whereas "half and half" refers to engagement in a combination of oral and vaginal sex). Interaction with agents of an addiction (e.g., card dealers, sex workers) or other addicts becomes embedded with a commonality of terms that refer to the behaviors, associated objects or paraphernalia, or subjective experience. The person may self-identify with addiction-related groups or activities (e.g., running clubs, pertaining to exercise addicts). Additionally, individuals with one addiction may communicate disparagingly about another addiction. For example, some methamphetamine users may operate within social contexts that ridicule people who drink alcohol or engage in other behaviors that are sedating or result in certain types of performance impairment (e.g., slurring words). Communication about the addiction, therefore, can be a way of forming or solidifying exclusive social relationships with other addicts or addictive object providers [75].

2.4. Expectations

Various conceptualizations of the expectancy construct have been applied to research on addictive behaviors since Rotter [76] initially proposed expectancy theory. Expectancy as a construct relevant to addiction involves the anticipated consequences of behavior or beliefs held about the likelihood of appetitive effects [77-80]. In general, expectancies are subjective probabilities regarding the likelihood of achieving various outcomes by engaging in some behavior. In terms of the PACE model, addiction expectancies or expectations are beliefs regarding the likelihood that or extent to which an addictive behavior is providing solutions to experiential requests. One may expect or anticipate that the addictive behavior will provide specific outcomes such as helping one live life more comfortably in the immediate present (e.g., to lift self-esteem, complement well other daily activities, or provide a social lubricant effect [7,26,81]).

There are several factors that contribute to development of specific expectancies for particular addictive behaviors. These include one's genetically inherited sensitivity to the behavior [82], emotional disposition (e.g., individuals with social anxiety tend to hold expectancies that alcohol facilitates social performance [83]), or motivational state (e.g., those with weight concerns may hold positive expectations regarding the appetite suppressing effects of tobacco [84]). Importantly, though,

specific expectancies develop through the interplay of individual difference variables with vicarious social learning, as well as with direct experience. For example, hearing comments relevant to expectancies for reinforcement from alcohol predate teens' first drinking experiences, and predict drinking onset [85].

Direct experience may refute, confirm, or enhance pre-use expectancies. The learned expectations and experiences of specific outcomes as they occur with a specific appetitive behavior likely play an important role in addiction specificity. For example, heavier drinkers differ from light drinkers on activation of expectancies of positive arousing effects *versus* sedating effects of alcohol [80,86,87]. Additionally, research suggests that individuals with a single addictive behavior (e.g., alcohol only) differ from those who engage in multiple addictive behaviors (e.g., alcohol and marijuana) in the degree to which they hold positive expectations about the second behavior, suggesting the possibility for an uncoupling of expectations across addictive behaviors [87]. For example, there may be positive-sedating marijuana use expectancies that would be inconsistent with positive-arousing alcohol use expectancies. Some persons may prefer one drug over the other due to these different expectancies, with a preference for sedation or arousal. Others may use both drugs with the expectation that they can use them to fluctuate or balance out their level of arousal [8]. Experiences with addictive behaviors thus may create subjective physiological expectancies that are addiction-specific.

Expectations associated with an addiction also may involve one's perceptions of the social images (or general lifestyle characteristics) associated with participation in the behavior. For example, gambling or shopping addictions may be associated with social images of living luxuriously, love or sex addictions may be associated with social images of intimacy or social power, and marijuana addiction may be associated with living a countercultural lifestyle [e.g., 88]. In addition, perceptions of the gradient of reinforcement value functions portraying different addictive behaviors may vary in steepness, leading to selection of one addiction with a steeper gradient (more reinforcement value per unit time) over another [89]. Through any number of determinants of expectancy differentiation (e.g., mass media impact, family or peer social learning, experiences with an addictive behavior), social image expectations may take shape and impact addiction specificity. As an individual's social activities begin to increasingly involve the addiction and other addicts or providers of the addiction, it may become possible to convince oneself that the addictive behavior does not interfere with and may even actually facilitate one's daily activities. One may come to rely on a specific addiction, avoiding all others, if this addiction is perceived to meet many of one's expectations for their life (e.g., there are people who might say that their life is "all right" as long as they have their marijuana).

3. Future Research Needs and Conclusions

While it is clear that addiction specificity is a phenomenon prevalent in the majority of the U.S. adult population who experience addiction problems [14], at least five research directions should be undertaken to better understand the parameters of addiction specificity or the PACE model as an explanatory device. First, hard epidemiologic data are lacking on addiction specificity [14]. That is, this empirical phenomenon is grossly understudied. Data need to be collected and examined across multiple addictive behaviors (and from diverse populations) to adequately assess the development of addiction specificity [14]. In particular, it would be informative to obtain data on the trajectories of

addiction specificity. That is, data comparing the age of onset, duration, recurrence, or recovery from different constellations of addictions would be beneficial for understanding how different patterns of addiction specificity might occur as well as assist in clinical intervention development. It is plausible that the developmental trajectories for different addictions vary. For example, it is possible that addiction to exercise develops quite slowly because it can takes years for one to get in good enough shape to be able to exercise excessively. On the other hand, addiction to cigarette smoking may occur rather quickly. Different steepness in trajectories may provide one reason why more people may become addicted to one behavior (e.g., cigarette smoking) *versus* another (e.g., exercise). In addition, possibly, people who become addicted to a lower trajectory addiction (e.g., exercise) may become addicted to other lower trajectory addictions (e.g., workaholism), at least more so than persons who tend to become addicted to high trajectory addictions. Treatment implications may vary in terms of focus on fear of loss of slowly gained "expertise" (e.g., exercise and work) *versus* instruction in delaying gratification (e.g., drug use). Certainly, these ideas are speculative [90, 91].

Second, empirical evidence further supporting or refuting operation of the PACE variables on addiction specificity is necessary. In particular, research is needed to discern separability of the four dimensions of the PACE model, and the interplay between them (e.g., interaction effects) in an effort to better understand how, and to what degree each make a contribution to addiction specificity. Empirical testing of the PACE model will require psychometrically sound measures for each dimension, which are not yet available. However, we suggest that measures of pragmatics should quantify the degree of accessibility of the supply of the addictive agent, the perceived awareness of supply sources, the level of acquisition skill that an individual possesses or perceives possessing, and the efficacy of the means of exchange used in the pursuit of the addictive behavior. Measures of attraction should allow for the differentiation between individuals with high preferences for a specific behavior from those with low preference towards the behavior. For example, one might be asked on rating scales how much they like the social context of the addictive behavior, the rituals involved in engaging in the behavior, or the way the behavior feels. Measures of communication should assess familiarity with an addiction-specific language. For example, one might be asked how much slang pertaining to an addictive behavior they think they know, how much slang they know that non-participants in the behavior would be unlikely to know, or to what extent they tend to communicate differently with others who engage in the behavior *versus* those who don't. The development of communication-type items is likely to require extensive qualitative and quantitative research to accurately gage communication regarding particular targets of addiction.

Expectation variables will need to be operationalized carefully to establish clear parameters that differentiate it from the other PACE variables. For example, one may be asked to what extent the behavior met their expectations, or how likely it was that the behavior would result in specific outcomes (e.g., degree to which the behavior helps one achieve a desired social image, fits well within one's daily activities). Such items would need to be differentiated from ones, for example, that asked to what extent the behavior felt good, or was liked (aspects of the attraction variable). Possibly, items that request subjective probability information and, in particular, information that is relatively cognitive (*versus* affective) in nature, would best delineate the expectancy dimension. Figure 1 is an attempt to provide one means of conceptualizing how the PACE model components may relate to each other, but its function is heuristic at this point. Arguably, it is possible that overlap among some of the

dimensions may exist and require some refinement in order to effectively differentiate the components (e.g., measurement of attraction *versus* expectations). This could potentially complicate understanding of the role each dimension serves in discriminating unique patterns of addiction.

Third, assuming the usefulness of separating the four dimensions, the operation of the PACE variables may differ in relative importance across different addictive behaviors, which may or may not reflect the reinforcement valence of these behaviors. For example, pragmatics may be a relatively important determinant of relatively hard-to-locate addictive behaviors (e.g., injection drug use, perhaps regular alcohol use among preteens), but may not be as important a determinant of easy-to-locate behaviors (e.g., eating, alcohol use among adults). Some addictions may be attractive to a relatively small percentage of the population (e.g., exercise), whereas other behaviors may have wide appeal (e.g., food). It is highly likely that each addiction is associated with specialized words or slang. However, is also possible that relatively socially acceptable addictive behaviors (eating, working, exercise) have fewer words associated with them to disguise their manifestations from non-participants. Finally, it is possible that different addictive behaviors are associated with different expectations (e.g., hedonism *versus* nurturance [23]). Examination of the relative importance of different PACE variables with different patterns of addiction specificity will require much work.

Fourth, while the current paper generally focused on addiction specificity from an individual differences perspective, it is important to note that there may be different patterns of addiction specificity within individuals over time. That is, when an addiction or finite set of addictions is terminated, a second addiction or set of addictions may or may not emerge. Longitudinal studies that assess the chronicity and/or fluidity of addictions within individuals will provide valuable information regarding how effectively the model delineates specificity *versus* co-occurrence. The PACE model as presented herein does not address the temporal stability of one' addiction specificity propensity, but we nonetheless acknowledge that within-person variability in cross-addiction tendencies is certainly possible. This is an important issue that should be addressed in future work.

Finally, if the PACE model is useful in explaining addiction specificity then there also may be some clinical research implications. Understanding the association of PACE constituents with different individual addiction specificity trajectories may be useful for assessment research planning and treatment tailoring. For example, PACE information could be tested to potentially identify those who would benefit from interventions that target a single addictive behavior with a steep trajectory (e.g., nicotine replacement for tobacco) *versus* interventions which would be more useful for those prone to co-occurring addictions with a slower trajectory (e.g., learning new ways to manage anhedonia could benefit many different addictions).

In summary, addiction specificity may be a complementary concept to addiction co-occurrence, identifying reasons for non-overlap among different patterns of addictive behaviors. We propose a PACE model, which delineates pragmatics, attraction, communication and expectation components as being a useful framework for investigation on the determinants of addiction specificity. Further research applying the PACE model to addiction specificity may eventually yield clinical applications that help reduce the public health burden associated with addiction.

Int. J. Environ. Res. Public Health **2011**, *8*

Acknowledgements

This paper was supported by grants from the National Institute on Drug Abuse (#sDA020138 and K08-DA025041).

References and Notes

1. Brewer, J.A.; Potenza, M.N. The neurobiology and genetics of impulse control disorders: Relationships to drug addictions. *Biochem. Pharmacol.* **2008**, *75*, 63-75.
2. Goodman, A. Addiction: Definition and implications. *Br. J. Addict.* **1990**, *85*, 1403-1408.
3. Goodman, A. Neurobiology of addiction: An integrative review. *Biochem. Pharmacol.* **2008**, *75*, 266-322.
4. Hatterer, L.J. The addictive process. *Psychiatr. Q.* **1982**, *54*, 149-156.
5. Jacobs, D.F. A general theory of addictions: A new theoretical model. *J. Gambl. Behav.* **1986**, *2*, 15-31.
6. Marks, I. Behaviour (non-chemical) addictions. *Br. J. Addict.* **1990**, *85*, 1389-1394.
7. Marlatt, G.A.; Baer, J.S.; Donovan, D.M.; Kivlahan, D.R. Addictive behaviors: Etiology and treatment. *Annu. Rev. Psychol.* **1988**, *39*, 223-252.
8. Sussman, S.; Ames, S.L. *Drug Abuse: Concepts*, *Prevention and Cessation*; Cambridge University Press: New York, NY, USA, 2008.
9. Faber, R.J.; Christenson, G.A.; de Zwaan, M.; Mitchell, J. Two forms of compulsive consumption: Comorbidity of compulsive buying and binge eating. *J. Consum. Res.* **1995**, *22*, 296-304.
10. Pelchat, M.L. Of human bondage: Food craving, obsession, compulsion, and addiction. *Physiol. Behav.* **2002**, *76*, 347-352.
11. Schneider, J.P.; Irons, R.R. Assessment and treatment of addictive sexual disorders: Dependency relapse. *Subst. Use Misuse* **2001** *36*, 1795-1820.
12. Shaffer, H.J.; LaPlante, D.A.; LaBrie, R.A.; Kidman, R.C.; Donato, A.N.; Stanton, M.V. Toward a syndrome model of addiction: Multiple expressions, common etiology. *Harv. Rev. Psychiatry* **2004**, *12*, 367-374.
13. Sunderwirth, S.G.; Milkman, H. Behavioral and neurochemical commonalities in addiction. *Contemp. Fam. Ther.* **1991**, *13*, 421-433.
14. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority. *Eval. Health Prof.* **2011**, *34*, 3-56.
15. Robinson, T.E.; Berridge, K.C. Mechanisms of action of addictive stimuli. Incentive-sensitization and addiction. *Addiction* **2001**, *96*, 103-114.
16. Robinson, T.E.; Berridge, K.C. The incentive sensitization theory of addiction: Some current issues. *Philos. Trans. R. Soc. B* **2008**, *363*, 3137-3146.
17. Bechara, A. Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nat. Neurosci.* **2005**, *8*, 1458-1463.
18. Stacy, A.W.; Ames, S.L.; Knowlton, B. Neurologically plausible distinctions in cognition relevant to drug abuse etiology and prevention. *Subst. Use Misuse* **2004**, *39*, 1571-1623.

19. Tiffany, S.T. A cognitive model of drug urges and drug-use behavior: The role of automaatic and non-automatic processes. *Psychol. Rev.* **1990**, *97*, 147-168.
20. Wiers, R.W.; Ames, S.L.; Hoffmann, W.; Krank, M.; Stacy, A.W. Impulsivity, impulsive and reflective processes and the development of alcohol use and misuse in adolescents and young adults. *Front. Psychopathol.* **2010**, *1*, 1-12.
21. Carnes, P.J.; Murray, R.E.; Charpentier, L. Bargains with chaos: Sex addicts and addiction interaction disorder. *Sex. Addict. Compuls.* **2005**, *12*, 79-120.
22. Lesieur, H.R.; Blume, S.B. Pathological gambling, eating disorders and the psychoactive substance use disorders. *J. Addict. Behav.* **1993**, *12*, 89-102.
23. Haylett, S.A.; Stephenson, G.M.; Lefever, R.M.H. Covariation in addictive behaviors: A study of addictive orientations using the shorter PROMIS Questionnaire. *Addict. Behav.* **2004**, *29*, 61-71.
24. Hirschman, E.C. The consciousness of addiction: Toward a general theory of compulsive consumption. *J. Consum. Res.* **1992**, *19*, 155-179.
25. Sussman, S.; Black, D.S. Substitute addiction: A concern for researchers and practitioners. *J. Drug Educ.* **2008**, *38*, 167-180.
26. Freimuth, M.; Waddell, M.; Stannard, J.; Kelley, S.; Kipper, A.; Richardson, A.; Szuromi, I. Expanding the scope of dual diagnosis and co-addictions: Behavioral addictions. *J. Groups Addict. Recover.* **2008**, *3*, 137-160.
27. Iacono, W.G.; Malone, S.M.; McGue, M. Behavioral disinhibition and the development of early-onset addiction: Common and specific influences. *Annu. Rev. Clin. Psychol.* **2008**, *4*, 325-348.
28. Ames, S.L.; McBride, C. Translating genetics, cognitive science and other basic science research findings into applications for prevention of substance use. *Eval. Health Prof.* **2006**, *29*, 277-301.
29. Nestler, E.J.; Landsman, D. Learning about addiction from the genome. *Nature* **2001**, *409*, 834-835.
30. Ducci, F.; Enoch, M.-A.; Funt, S.; Virkkunen, M.; Albaugh, B.; Goldman, D. Increased anxiety and other similarities in temperament of alcoholics with and without antisocial personality disorder across three diverse populations. *Alcohol* **2007**, *41*, 3-12.
31. Ames, S.L.; Grenard, J.; Thush, C.; Sussman, S.; Wiers, R.W.; Stacy, A.W. Comparison of indirect assessments of association as predictors of marijuana use among at-risk adolescents. *Exp. Clin. Psychopharmacol.* **2007**, *15*, 204-218.
32. Stacy, A.W. Memory association and ambiguous cues in models of alcohol and marijuana use. *Exp. Clin. Psychopharmacol.* **1995**, *3*, 183-194.
33. Stacy, A.W. Memory activation and expectancy as prospective predictors of alcohol and marijuana use. *J. Abnorm. Psychol.* **1997**, *106*, 61-73.
34. Huston, T.L. *Foundations of Interpersonal Attraction*; Academic Press: New York, NY, USA, 1974.
35. Sussman, S.; Unger, J.B. A “drug abuse” theoretical Integration: A transdisciplinary speculation. *Subst. Use Misuse* **2004**, *39*, 2055-2069.
36. Sussman, S.; Reynaud, M.; Aubin, H.-J.; Leventhal, A.M. Drug addiction, love, and the higher power. *Eval. Health Prof.* **2011**, doi: 10.1177/0163278711401002.

37. Cornish, J.W.; O'Brien, C.P. Crack cocaine abuse: An epidemic with many public health consequences. *Annu. Rev. Public Health* **1996**, *17*, 259-273.
38. Jiggens, J. Australian heroin seizures and the causes of the 2001 heroin shortage. *Int. J. Drug Policy* **2008**, *19*, 273-278.
39. Wood, E.; Stolz, J.-A.; Li, K.; Montaner, J.S.G.; Kerr, T. Changes in Canadian heroin supply coinciding with the Australian heroin shortage. *Addiction* **2006**, *101*, 689-695.
40. Bluthenthal, R.N.; Cohen, D.A.; Farley, T.A.; Scribner, R.; Beighley, C.; Schonlau, M.; Robinson, P.L. Alcohol availability and neighborhood characteristics in Los Angeles, California and southern Louisiana. *J. Urban Health* **2008**, *85*, 191-205.
41. LaBrie, R.A.; Nelson, S.E.; LaPlante, D.A.; Peller, A.J.; Caro, G.; Shaffer, H.J. Missouri casino self-excluders: Distributions across time and space. *J. Gambl. Stud.* **2007**, *23*, 231-243.
42. Akers, R.L.; Krohn, M.D.; Lanza-Kaduce, L.; Radosevich, M. Social learning and deviant behavior: A specific test of a general theory. *Am. Sociol. Rev.* **1979**, *44*, 636-655.
43. Johnston, L.D.; O'Malley, P.M.; Bachman, J.G.; Schulenberg, J.E. *Monitoring the Future National Survey Results on Drug Use*, *1975-2008*; National Institute on Drug Abuse: Bethesda, MD, USA, 2009; Volume I, pp. 40-44.
44. Potenza, M.N.; Fiellin, D.A.; Heninger, G.R.; Rounsaville, B.J.; Mazure, C.M. Gambling: An addictive behavior with health and primary care implications. *J. Gen. Intern. Med.* **2002**, *17*, 721-732.
45. Le Bon, O.; Streel, E.; Tecco, J.; Hanak, C.; Hansenne, M.; Ansseau, M.; Pelc, I.; Verbanck, P.; Dupont, S. Personality profile and drug of choice: A multivariate analysis using Cloninger's TCI on heroin addicts, alcoholics, and a random population group. *Drug Alcohol Depend.* **2004**, *73*, 175-182.
46. Moffitt, T.E.; Caspi, A.; Dickosn, N.; Silva, P.; Stanton, W. Childhood-onset *versus* adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Dev. Psychopathol.* **1996**, *8*, 399-424.
47. Freimuth, M. *Addicted? Recognizing Destructive Behavior before It's Too Late*. Rowman & Littlefield Publishers, Inc.: Lanham, MD, USA, 2008.
48. Di Chiara, G. Nucleus accumbens shell and core dopamine: Differential role in behavior and addiction. *Behav. Brain Res.* **2002**, *137*, 75-114.
49. Haertzen, C.A.; Kocher, T.R.; Miyasato, K. Reinforcements from the first drug experience can predict later drug habits and/or addiction: Results with coffee, cigarettes, alcohol, barbiturates, minor and major tranquilizers, stimulants, marijuana, hallucinogens, heroin, opiates and cocaine. *Drug Alcohol Depend.* **1983**, *11*, 147-165.
50. De Wit, H. Individual differences in acute effects of drugs in humans: Their relevance to risk for abuse. *NIDA Res. Monogr.* **1998**, *169*, 176-187.
51. Pepino, M.Y.; Mennella, J.A. Factors contributing to individual differences in sucrose preference. *Chem. Senses* **2005**, *30*(Suppl 1), i319-i320.
52. Leventhal, A.M.; Chasson, G.S.; Tapia, E.; Miller, E.K.; Pettit, J.W. Measuring hedonic capacity in depression: A psychometric analysis of three anhedonia scales. *J. Clin. Psychol.* **2006**, *62*, 1545-1558.
53. Tremblay, L.K.; Naranjo, C.A.; Cardenas, L.; Herrmann, N.; Busto, U.E. Probing brain reward system function in major depressive disorder: Altered response to dextroamphetamine. *Arch. Gen. Psychiatry* **2002**, *59*, 409-417.

54. Tremblay, L.K.; Naranjo, C.A.; Graham, S.J.; Herrmann, N.; Mayberg, H.S.; Hevenor, S.; Busto, U.E. Functional neuroanatomical substrates of altered reward processing in major depressive disorder revealed by a dopaminergic probe. *Arch. Gen. Psychiatry* **2005**, *62*, 1228-1236.
55. Khantzian, E.J. The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. *Am. J. Psychiatry* **1985**, *142*, 1259-1264.
56. Suh, J.J.; Ruffins, S.; Robins, C.E.; Albanese, M.J.; Khantzian, E.J. Self-medication hypothesis: Connecting affective experience and drug choice. *Psychoanal. Psychol.* **2008**, *25*, 518-532.
57. Zuckerman, M. P-impulsive sensation seeking and its behavioral, psychophysiological and biochemical correlates. *Neuropsychobiology* **1993**, *28*, 30-36.
58. Zuckerman, M. *Behavioral Expressions and Biosocial Bases of Sensation Seeking*; Cambridge University Press: New York, NY, USA, 1994.
59. Wiers, R.W.; Stacy, A.W. *Handbook of Implicit Cognition and Addiction*; SAGE Publications: Thousand Oaks, CA, USA, 2006.
60. Kensinger, E.A.; Garoff-Eaton, R.J.; Schacter, D.L. How negative emotion enhances the visual specificity of a memory. *J. Cognit. Neurosci.* **2007**, *19*, 1872-1887.
61. Carter, B.L.; Tiffany, S.T. Meta-analysis of cue-reactivity in addiction research. *Addiction* **1999**, *94*, 327-340.
62. Chambers, R.A.; Taylor, J.R.; Poetnza, M.N. Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *Am. J. Psychiatry* **2003**, *160*, 1041-1052.
63. Giedd, J.N. The teen brain: Insights from neuroimaging. *J. Adolesc. Health* **2008**, *42*, 335-343.
64. Giedd, J.N.; Lalonde, F.M.; Celano, M.J.; White, S.L.; Wallace, G.L.; Lee, N.R.; Lenroot, R.K. Anatomical brain magnetic resonance imaging of typically developing children and adolescents. *J. Am. Acad. Child Adolesc. Psychiatry* **2009**, *48*, 465-470.
65. Leventhal, A.M.; Waters, A.J.; Boyd, S.; Moolchan, E.T.; Heishman, S.J.; Lerman, C.; Pickworth, W.B. Associations between Cloninger's temperament dimensions and acute tobacco withdrawal. *Addict. Behav.* **2007**, *32*, 2976-2989.
66. Stewart, D.G.; Brown, S.A. Withdrawal and dependency symptoms among adolescent alcohol and drug abusers. *Addiction* **1995**, *90*, 627-635.
67. Elder, G. Life Course and Human Development. In *Handbook of Child Psychology*; Lerner, R.M., Ed.; John Wiley & Sons: New York, NY, USA, 1998; Volume 1, pp. 939-991.
68. Snyder, J.; Schrepferman, L.; Oeser, J.; Patterson, G.; Stoolmiller, M.; Johnson, K.; Snyder, A. Deviancy training and association with deviant peers in young children: Occurrence and contribution to early-onset conduct problems. *Dev. Psychopathol.* **2005**, *17*, 397-413.
69. Bahr, S.J.; Hoffmann, J.P.; Yang, X. Parental and peer influences on the risk of adolescent drug use. *J. Primary Prev.* **2005**, *26*, 529-551.
70. Hyde, J.P. The language game: “Rat” talk: The special vocabulary of some teenagers. *Engl. J.* **1982**, *71*, 98-101.
71. Available online: www.ildado.com/casino_glossary.html (accessed on 9 December 2010).
72. Nace, E.P. Epidemiology of alcoholism and prospects for treatment. *Annu. Rev. Med.* **1984**, *35*, 293-309.
73. Moreno, J.A. Strategies for challenging police jargon testimony. *Crim. Justice* **2006**, *20*, 28-37.

74. Tong, V.; McIntyre, T.; Silmon, H. What's the flavor? Understanding inmate slang usage in correctional education settings. *J. Correct. Educ.* **1997**, *48*, 192-197.
75. Dalzell, T.; Victor, T. *Vice Slang*; Routledge: New York, NY, USA, 2008.
76. Rotter, J.B. *Social Learning and Clinical Psychology*; Prentice Hall: Englewood Cliffs, NJ, USA, 1954.
77. Goldman, M.S. Expectancy and risk for alcoholism: The unfortunate exploitation of a fundamental characteristic of neurobehavioral adaptation. *Alcohol. Clin. Exp. Res.* **2002**, *26*, 737-746.
78. Goldman, M.S.; Reich, R.R.; Darkes J. Expectancy as a unifying construct in alcohol-related cognition. In *Handbook of Implicit Cognition and Addiction*; Wiers, R., Stacy, A., Eds.; Sage Publications: Thousand Oaks, CA, USA, 2006; pp. 105-121.
79. Goldman, M.S.; Brown, S.A.; Christiansen, B.A.; Smith, G.T. Alcoholism and memory: Broadening the scope of alcohol-expectancy research. *Psychol. Bull.* **1991**, *110*, 137-146.
80. Reich, R.R.; Goldman, M.S. Exploring the alcohol expectancy memory network: The utility of free associates. *Psychol. Addict. Behav.* **2005**, *19*, 317-325.
81. Goldman, M.S.; Darkes, J. Alcohol expectancy multiaxial assessment: A memory network-based approach. *Psychol. Assess.* **2004**, *16*, 4-15.
82. McCarthy, D.M.; Brown, S.A.; Carr, L.G.; Wall, T.L. ALDH2 status, alcohol expectancies, and alcohol response: Preliminary evidence for a mediation model. *Alcohol. Clin. Exp. Res.* **2001**, *25*, 1558-1563.
83. Eggleston, A.M.; Woolaway-Bickel, K.; Schmidt, N.B. Social anxiety and alcohol use: Evaluation of the moderating and mediating effects of alcohol expectancies. *J. Anxiety Disord.* **2004**, *18*, 33-49.
84. Cavallo, D.A.; Smith, A.E.; Schepis, T.S.; Desai, R.; Potenza, M.N.; Krishnan-Sarin, S. Smoking expectancies, weight concerns, and dietary behaviors in adolescence. *Pediatrics* **2010**, *126*, e66-e72.
85. Smith, G.T. Psychological expectancy as mediator of vulnerability to alcoholism. *Ann. N. Y. Acad. Sci.* **1994**, *708*, 165-171.
86. Kramer, D.A.; Goldman, M.S. Using a modified Stroop task to implicitly discern the cognitive organization of alcohol expectancies. *J. Abnorm. Psychol.* **2003**, *112*, 171-175.
87. Simons, J.S.; Dvorak, R.D.; Lau-Barraco, C. Behavioral inhibition and activation systems: Differences in substance use expectancy organization and activation in memory. *Psychol. Addict. Behav.* **2009**, *23*, 315-328.
88. Holtgraves, T.M. Gambling as self-presentation. *J. Gambl. Behav.* **1988**, *4*, 78-91.
89. Herrnstein, R.J.; Prelec, D. Melioration: A theory of distributed choice. *J. Econ. Perspect.* **1991**, *5*, 137-156.
90. Albrecht, U.; Kirschner, N.E.; Grusser, S.M. Diagnostic instruments for behavioral addiction: An overview. *GMS Psycho Soc. Med.* **2007**, *4*, 1-11.
91. Lacy, J.H.; Evans, C.D.H. The impulsivist: A multi-impulsive personality disorder. *Br. J. Addict.* **1986**, *81*, 641-649.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Blum, K.; Chen, A.L.C.; Oscar-Berman, M.; Chen, T.J.H.; Lubar, J.; White, N.; Lubar, J.; Bowirrat, A.; Braverman, E.; Schoolfield, J.; *et al.* Generational Association Studies of Dopaminergic Genes in Reward Deficiency Syndrome (RDS) Subjects: Selecting Appropriate Phenotypes for Reward Dependence Behaviors. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4425-4459; doi:10.3390/ijerph8124425.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Generational Association Studies of Dopaminergic Genes in Reward Deficiency Syndrome (RDS) Subjects: Selecting Appropriate Phenotypes for Reward Dependence Behaviors

Kenneth Blum [1,11,14,16,17,18,19,*], Amanda L. C. Chen [2,*], Marlene Oscar-Berman [3], Thomas J. H. Chen [4], Joel Lubar [5], Nancy White [6], Judith Lubar [7], Abdalla Bowirrat [8], Eric Braverman [9,14], John Schoolfield [10], Roger L. Waite [11], Bernard W. Downs [11], Margaret Madigan [11], David E. Comings [12], Caroline Davis [13], Mallory M. Kerner [14], Jennifer Knopf [14], Tomas Palomo [15], John J. Giordano [16,17], Siobhan A. Morse [16,17], Frank Fornari [18], Debmalya Barh [19], John Femino [20] and John A. Bailey [1]

[1] Department of Psychiatry, School of Medicine and McKnight Brain Institute, University of Florida, W University Ave., Gainesville, FL 32601, USA; E-Mail: baileyjo@ufl.edu
[2] Department of Engineering Management Advanced Technology, Chang Jung Christian University, No. 396, Sec. 1, Changrong Road, Tainan 71101, Taiwan
[3] Department of Anatomy & Neurobiology, Boston University School of Medicine, 72 East Concord Street, Boston, MA 02118, USA; E-Mail: Oscar@bu.edu
[4] Department of Occupational Safety and Health, Chang Jung Christian University, No. 396, Sec. 1, Changrong Road, Tainan 71101, Taiwan; E-Mail: tjhchen@yahoo.com.tw
[5] Emeritus, Department of Physiology, University of Tennessee, 719 Andy Holt Tower, Knoxville, TN 37996, USA; E-Mail: lubar@utkux.utcc.utk.edu
[6] Unique Mindcare, Inc., 1900 Saint James Place, Houston, TX 77056, USA; E-Mail: nancy@Uniquemindcare.com
[7] Department of Neurofeedback, Southeastern Biofeedback and Neurobehavioral Clinic, 101 Westwood Road, Knoxville, TN 37919, USA; E-Mail: lubar@utkux.utcc.utk.edu
[8] Department of Neuroscience & Population Genetics, EMMS Nazareth Hospital, Nazareth, Israel; E-Mail: bowirrat@netvision.net.il
[9] Department of Neurosurgery, Weill Cornell College of Medicine, 1300 York Ave., New York, NY 10065, USA; E-Mail: pathfoundationny@gmail.com
[10] Department of Academic Informatics Services, University of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, TX 78229, USA; E-Mail: schoolfield@uthscsa.edu
[11] Department of Nutrigenomics, LifeGen, Inc., P.O. Box 366, 570 Lederach Stattion Way, Lederach, PA 19450, USA; E-Mails: drw8@san.rr.com (R.L.W.); bernardwdowns@gmail.com (B.W.D.); MARGBETTS@hotmail.com (M.M.)

Int. J. Environ. Res. Public Health **2011**, *8*

[12] Department of Genomic Research, Carlsbad Science Foundation, Department of Medical Genetics, City of Hope National Medical Center, 1500 Duarte Road, Duarte, CA 91010, USA; E-Mail: dcomings@earthlink.net
[13] Department of Kinesiology and Health Sciences, York University, 4700 Keele Street, Toronto, ON M3J 1P3, Canada; E-Mail: cdavis@yorku.ca
[14] Department of Integrative Medicine, PATH Medical Research Foundation, 304 Park Ave. South, New York, NY 10010, USA; E-Mails: Mallory.kerner@gmail.com (M.M.K.); info@pathfoundationny.org (J.K.)
[15] Hospital Universitario 12 de Octubre, Servicio de Psiquiatria, Av. Cordoba SN, Madrid 28041, Spain; E-Mail: tpalomo2004@yahoo.es
[16] Department of Holistic Medicine, G&G Holistic Addiction Treatment, Inc., 1590 Northeast 162nd Street, North Miami Beach, FL 33162, USA; E-Mail: michg8@hotmail.com
[17] Department of Research, National Institute for Holistic Addiction Studies, 1590 Northeast 162nd Street, North Miami Beach, FL 33162, USA; E-Mail: samorse10@aol.com
[18] Dominion Diagnostics, Inc., 211 Circuit Road, North Kingstown, RI 02852, USA; E-Mail: ffornari@me.com
[19] Center for Genomics and Applied Gene Technology, Institute of Integrative Omics and Applied Biotechnology, Nonakuri, Purba Medinipur, West Bengal, India; E-Mail: dr.barh@gmail.com
[20] Meadows Edge Recovery Center, 580 10 Rod Road, North Kingstown, RI 02852, USA; E-Mail: jfemino@meadowsedge.com

* Authors to whom correspondence should be addressed; E-Mails: drd2gene@aol.com (K.B.); tjhchen@yahoo.com.tw (A.L.C.C.).

Received: 26 October 2011; in revised form: 23 November 2011 / Accepted: 23 November 2011 / Published: 29 November 2011

Abstract: Abnormal behaviors involving dopaminergic gene polymorphisms often reflect an insufficiency of usual feelings of satisfaction, or Reward Deficiency Syndrome (RDS). RDS results from a dysfunction in the "brain reward cascade," a complex interaction among neurotransmitters (primarily dopaminergic and opioidergic). Individuals with a family history of alcoholism or other addictions may be born with a deficiency in the ability to produce or use these neurotransmitters. Exposure to prolonged periods of stress and alcohol or other substances also can lead to a corruption of the brain reward cascade function. We evaluated the potential association of four variants of dopaminergic candidate genes in RDS (dopamine D1 receptor gene [DRD1]; dopamine D2 receptor gene [DRD2]; dopamine transporter gene [DAT1]; dopamine beta-hydroxylase gene [DBH]). *Methodology*: We genotyped an experimental group of 55 subjects derived from up to five generations of two independent multiple-affected families compared to rigorously screened control subjects (e.g., N = 30 super controls for DRD2 gene polymorphisms). Data related to RDS behaviors were collected on these subjects plus 13 deceased family members. *Results*: Among the genotyped family members, the DRD2 Taq1 and the DAT1 10/10 alleles were

Int. J. Environ. Res. Public Health **2011**, *8*

significantly (at least $p < 0.015$) more often found in the RDS families *vs*. controls. The TaqA1 allele occurred in 100% of Family A individuals (N = 32) and 47.8% of Family B subjects (11 of 23). No significant differences were found between the experimental and control positive rates for the other variants. *Conclusions*: Although our sample size was limited, and linkage analysis is necessary, the results support the putative role of dopaminergic polymorphisms in RDS behaviors. This study shows the importance of a nonspecific RDS phenotype and informs an understanding of how evaluating single subset behaviors of RDS may lead to spurious results. Utilization of a nonspecific "reward" phenotype may be a paradigm shift in future association and linkage studies involving dopaminergic polymorphisms and other neurotransmitter gene candidates.

Keywords: dopamine; gene polymorphisms; generational association studies; phenotype; "super normal" controls; Reward Deficiency Syndrome (RDS)

1. Introduction

1.1. Background

Reward Deficiency Syndrome (RDS) was first defined by our lab in 1996 as a putative predictor of impulsive and addictive behaviors [1-7]. It refers to an absence of usual feelings of satisfaction and a failure of the system that normally confers gratification, resulting in behaviors such as overeating, heavy cigarette smoking, drug and alcohol abuse, gambling, and hyperactivity (see Table 1 in a later section). The syndrome has been linked to dysfunction of dopamine (DA) receptors, the genes for which show many mutant forms. In an attempt to resolve controversy regarding the causal contributions of mesolimbic DA systems to reward, we have evaluated the three main competing explanatory categories: "liking," "learning," and "wanting" [8], especially as they relate to RDS [9]. That is, DA may mediate: (a) the hedonic impact of reward (liking), (b) learned predictions about rewarding effects (learning), or (c) the pursuit of rewards by attributing incentive salience to reward-related stimuli (wanting). We evaluated these hypotheses, especially as they relate to the Reward Deficiency Syndrome (RDS), and we find that the incentive salience or "wanting" hypothesis of DA function is supported by a majority of the evidence [9].

Neuroimaging studies have shown that drugs of abuse, palatable foods, and anticipated behaviors such as sex and gaming affect brain regions involving reward circuitry, and may not be unidirectional. Gardner [10] has suggested that drugs of abuse that promote DA signals short circuit and sensitize dynamic mesolimbic mechanisms that evolved to attribute incentive salience to rewards. Accordingly, Gardner [10] further suggested that addictive drugs have in common that they are voluntarily self-administered, they enhance (directly or indirectly) dopaminergic synaptic function in the nucleus accumbens (NAc), and they stimulate the functioning of brain reward circuitry (producing the "high" that drug users seek). Blum and Gold [11] pointed out that reward circuitry is very complex, especially as these circuits relate to hedonic tone. Moreover, these circuits now are believed to be functionally more complex, also encoding attention, reward expectancy, disconfirmation of reward expectancy, and

incentive motivation. Elevated stress levels, together with polymorphisms of dopaminergic genes and other neurotransmitter genetic variants, may have a cumulative effect on vulnerability to addiction. We and others believe that the RDS model of etiology holds very well for addictions [10].

The D2 receptor has been associated with pleasure, and the DRD2 has been referred to as a reward gene [9-16]. The DRD2 gene, and especially the Taq1 A1 allele, has been associated with neuropsychiatric disorders in general, including alcoholism, other addictions (e.g., carbohydrate) [17-23], and it also may be involved in co-morbid antisocial personality disorder symptoms [24], especially in children and adults with attention deficit hyperactivity disorder (ADHD) or Tourette Syndrome [5,25] and high novelty seeking [26-29].

DA is involved in a remarkable number of behavioral functions including cognition and motor effects [30], depending upon its localization in the brain. DA has been called the "anti-stress molecule" and/or the "pleasure molecule" [2,31-33] and is released into the synapse in the NAc where it stimulates a number of receptors (D1–D5), which results in increased feelings of well-being and stress reduction. The mesocorticolimbic dopaminergic pathway plays an especially important role in mediating the reinforcement of natural rewards like food and sex, as well as unnatural rewards like drugs of abuse. Natural rewards include satisfaction of physiological drives (e.g., hunger and reproduction) [34,35], and unnatural rewards are learned and involve satisfaction of acquired pleasures such as hedonic sensations [36,37] derived from alcohol and other drugs, as well as from gambling and other risk-taking behaviors [33-36,38,39].

In discussing RDS, we refer specifically to an insensitivity and inefficiency in the reward system [1-4,40,41]. There may be a common neurocircuitry [42] and neurobiology [43] for multiple addictions [44,45] and for a number of psychiatric disorders [43-51]. Due to specific genetic antecedents and environmental influences [52], a deficiency of the D2 receptors may predispose individuals to a high risk for multiple addictive, impulsive, and compulsive behaviors [4,31,53-57]. It is well known that alcohol and other drugs of abuse [58], as well as most positive reinforcers (e.g., sex [59], food [60], gambling [61-63], aggressive thrills [6]) cause activation and neuronal release of brain DA [32,62,63], which in turn can decrease negative feelings and satisfy abnormal cravings for alcohol, cocaine, heroin, and nicotine, and which are linked to low DA function [53,55,56,64-78].

Since there is a commonality in the mechanism by which drugs of abuse, smoking, food, sex, gaming—and their associated cues, e.g., seeing drug paraphernalia—stimulate the release of mesolimbic DA at the NAc, it is difficult to determine which, if any, of these RDS behaviors will specifically manifest in a family member. However, utilizing a Bayesian mathematical model, we have found that at least for carriers of the DRD2 A1 allele, the estimated predictive value is 74% [3]. Simply put, having this high predictive value suggests that an individual carrier could transfer one addiction for another [3]. In terms of gene expression at the mRNA level, there is specificity for many psychoactive drugs [79].

1.2. Brief Description of Risk Alleles in a Number of Dopaminergic Reward Genes

Dopamine D2 receptor gene (DRD2). The DA D2 receptor gene (DRD2) first associated with severe alcoholism is the most widely studied gene in psychiatric genetics [19]. The Taq1 A is a single nucleotide polymorphism (SNP rs: 1800497) originally thought to be located in the 3'-untranslated

region of the DRD2 but has since been shown to be located within exon 8 of an adjacent gene, the ankyrin repeat and kinase domain containing 1 (ANKK1). Importantly, while there may be distinct differences in function, the mis-location of the Taq1 A allele may be attributable to the ANKKI and the DRD2 being on the same haplotype or the ANKKI being involved in reward processing through a signal transduction pathway [80]. The ANKKI and the DRD2 gene polymorphisms may have distinct, different actions with regard to brain function [81]. Presence of the $A1^+$ genotype (A1/A1, A1/A2) compared to the A^- genotype (A2/A2) is associated with reduced receptor density [82-84]. This reduction causes hypodopaminergic functioning in the DA reward pathway. Other DRD2 polymorphisms such as the C (57T, A SNP (rs: 6277) at exon 7 also associates with a number of RDS behaviors including drug use [85]. Compared to the T^- genotype (C/C), the T^+ genotype (T/T, T/C) is associated with reduced translation of DRD2 mRNA and diminished DRD2 mRNA, leading to reduced DRD2 density and a predisposition to alcohol dependence [86]. The Taq1 A allele is a predictive risk allele in families [87].

Dopamine transporter gene (DAT1). The DA transporter protein regulates DA-mediated neurotransmission by rapidly accumulating DA that has been released into the synapse [88]. The DA transporter gene (SLC6A3 or DAT1) is localized to chromosome 5p15.3. Moreover, there is a VNTR polymorphism within the 3' non-coding region of DAT1 [89]. There are two important alleles that may independently increase risk for RDS behaviors. The 9 repeat (9R) VNTR has been shown to influence gene expression and to augment transcription of the DA transporter protein, resulting in an enhanced clearance of synaptic DA, yielding reduced levels of DA to activate postsynaptic neurons. Presence of the 9R VNTR has also been linked to Substance Use Disorder [87]. Moreover, in terms of RDS behaviors, tandem repeats of the DA transporter gene (DAT) [90] have been associated with high risk for ADHD in children and in adults alike [91,92]. The 10-repeat allele is significant for hyperactivity-impulsivity (HI) symptoms [93].

Dopamine D1 receptor gene (DRD1). Abnormalities in the dopaminergic reward pathways have frequently been implicated in substance abuse and addictive behaviors. Recent studies by Self [94] have suggested an important interaction between the DA D1 and D2 receptors in cocaine abuse. To test the hypothesis that the DRD1 gene might play a role in addictive behaviors Comings *et al.*[95], examined the alleles of the Dde I polymorphism in three independent groups of subjects with varying types of compulsive, addictive behaviors Tourette syndrome probands, smokers, and pathological gamblers. Specifically, in all three groups there was a significant representation of the frequency of homozygosity for the DRD1 Dde I 1 or 2 alleles in subjects with addictive behaviors. The DRD1 11 or 22 genotype was present in 41.3% of 63 controls and 57.3% of 227 TS probands ($p = 0.024$). When 23 quantitative traits were examined statistically, those carrying the 11 genotype consistently showed the highest scores. Based on these results, they examined the prevalence of the 11 genotype in controls and in Tourette syndrome probands. There was a highly significant progressive, linear increase in scores for gambling, alcohol use, and compulsive shopping. Problems with three additional behaviors, drug use, compulsive eating, and smoking also were significant. All six variables were related to addictive behaviors. In a totally separate group of controls and individuals attending a smoking cessation clinic, 39.3% of the controls *versus* 66.1% of the smokers carried the 11 or 22 genotype ($p = 0.0002$). In a third independent group of pathological gamblers, 55.8% carried the 11 or 22 genotype ($p = 0.009$ *versus* the combined controls). In the Tourette syndrome group and in smokers there was a significant

additive effect of the DRD1 and DRD2 genes. The results for both the DRD1 and DRD2 genes, which have opposing effects on cyclic AMP, were consistent with negative and positive heterosis, respectively. These results support a role for genetic variants of the DRD1 gene in some addictive behaviors, and an interaction of genetic variants at the DRD1 and DRD2 genes.

Volkow's group [96], using *in vivo* optical microprobe imaging, tested the role of DA D1 receptors relative to DA D2 receptors during acute cocaine administration. Their results suggested that since activation of striatal D1R-expressing neurons (direct-pathway) enhanced cocaine reward, whereas activation of D2R-expressing neurons suppressed it (indirect-pathway), cocaine's rewarding effects entailed both its fast stimulation of D1R (resulting in abrupt activation of direct-pathway neurons) and a slower stimulation of D2R (resulting in longer-lasting deactivation of indirect-pathway neurons). Lobo *et al*. also provided direct *in vivo* evidence of D2R and D1R optogenetic interactions in the striatal responses to acute cocaine administration [97].

Dopamine beta-hydroxylase gene (DBH). DA β-hydroxylase (DBH) is a membrane-bound enzyme that converts DA to norepinephrine, thereby making norepinephrine and epinephrine the only transmitters synthesized inside vesicles (http://en.wikipedia.org/wiki/Dopamine_beta_hydroxylase). It is expressed in noradrenergic nerve terminals of the central and peripheral nervous systems, as well as in chromaffin cells of the adrenal medulla. Even prior to the emergence of the neuropsychogenetic field and candidate gene analysis was begun, Egeland identified a possible link to manic-depressive disorder and the tyrosine hydroxylase enzyme [98]. Subsequently, Comings and his colleagues [99] associated the DBH gene polymorphism with ADHD. Polymorphisms of three different dopaminergic genes, DA D2 receptor (DRD2), DBH, and DA transporter (DAT1), were examined in Tourette syndrome probands, their relatives, and controls. Each gene individually showed a significant correlation with various behavioral variables in these subjects. The additive and subtractive effects of the three genes were examined by genotyping all three genes in the same set of subjects. For nine of 20 Tourette syndrome associated comorbid behaviors, there was a significant linear association between the degree of loading for markers of three genes and the mean behavior scores. The behavior variables showing the significant associations were, in order: ADHD, stuttering oppositional defiant, tics, conduct, obsessive-compulsive, mania, alcohol abuse, and general anxiety. These are behaviors that constitute the most overt clinical aspects of Tourette syndrome. For 16 of the 20 behavior scores, there was a linear progressive decrease in the mean score, with progressively lesser loading for the three gene markers.

In a recent PUBMED search, we coupled the terms DBH and ADHD, and we found 39 citations; the findings were both positive and negative. An interesting noteworthy example involves the work of Hess *et al.* [100]. Their findings did not implicate the DBH C-1021T polymorphism in the pathophysiology of depressive disorders or personality disorders, yet homozygosity at this locus appeared to increase the risk towards personality traits related to impulsiveness, aggression, and related disease states. In 2008, others [101] reported for polymorphisms G444A and C1603T in DBH, which were detected by univariant analysis, haplotype resulted in showing that the risk of ADHD was significantly increased in the presence of allele DBH +444A, as well as in the presence of allele DBH +1603T (O.R. = 15). Specifically, Barkley *et al.* [102] found that the DBH TaqI A2 allele, when homozygous, was associated with increased hyperactivity in childhood, pervasive behavior problems at adolescence, and earning less money on a card-playing task in adulthood. At adolescence, poorer

test scores were also found only in the hyperactive group, which was homozygous for this allele. Similar associations with ADHD related behaviors have been reported by others [62,76,103], as well as in a meta-analysis showing the association of DBH and ADHD etiology [100]. Moreover, McKinney *et al*. [104] found that polymorphisms of DBH and MOA predicted whether a person was a heavy smoker and how many cigarettes they consumed. The findings of McKinney *et al*. [104] support the view that these enzymes help to determine a smoker's requirement for nicotine and may explain why some people are predisposed to tobacco addiction and why some find it very difficult to stop smoking.

We report here the results of the first intra-generational family association study, concerning a sampling of dopaminergic polymorphisms, utilizing a generalized RDS set of behaviors as the "phenotype" (see Table 1 defining thirds phenotype) rather than any single select phenotype, as well as a group of "super normal" control subjects. Super controls have been extensively screened for many associated RDS behaviors as defined herein (see below). Our results provide sufficient evidence to support a new approach to the study of Reward Deficient aberrant behaviors.

Table 1. Examples of behaviors and disorders [1-4,11,12,27,32,33,105-114, 116-140,185,186,196,210,213] associated with Reward Deficiency Syndrome.

ADDICTIVE BEHAVIORS: Alcoholism; Drug Abuse; Smoking; Compulsive Eating and Obesity
IMPULSIVE BEHAVIORS: Attention Deficit Disorder; Attention Deficit Hyperactivity Disorder; Autistic Disorders; Tourette Syndrome
COMPULSIVE DISORDERS: Hypersexuality and Aberrant Sexual Behaviors; Pathological Gambling and Internet Gaming
PERSONALITY DISORDERS: Antisocial Personality Disorder; Conduct Disorder; Pathological Aggression; Generalized Anxiety Disorder

2. Methodology

Although other neurotransmitter systems are involved in these complex behaviors representative of polygenic inheritance, we decided to evaluate the potential association of certain polymorphisms of the DA: D1 receptor (DRD1), DA D2 receptor (DRD2) [rs1800297 as a RFLP in "DRD2"] which it is near in the 3'-untranslated region contains ANKK1 on the opposite strand of DNA and our method herein targets the same site], DA transporter (DAT1), and DBH genes. To that end, we genotyped 55 subjects, from two independent multiple-affected families with documented RDS behaviors. There were four generations (Family A—initial proband was identified with ADHD) and five generations (Family B—initial probands was identified with substance use disorder). We had postmortem data related to RDS behaviors on all the deceased family members. A total of 13 members died, and their respective DNA specimens were not available. Figure 1 illustrates the genotyping and self-reported and family-identified behaviors for each family member.

2.1. Subject Selection

All individuals were evaluated through structured interviews using DMS-IV criteria and a number of neuropsychological and electrophysiological tests (e.g., Meyer-Briggs, Millon, TOVA, qEEG, *etc*.).

Int. J. Environ. Res. Public Health **2011**, *8*

The "super" control group consisted of 30 individuals selected from a total of 189 people attending PATH Medical Clinic, an integrative care center and research foundation in New York City, for both neurological and non-neurological problems (for more details, see [105]). These individuals were carefully screened, including their family history to exclude a number of RDS-related behaviors. The excluded behaviors included but where not limited to: alcoholism, substance use disorder, smoking behavior, carbohydrate binging, obesity, ADHD, posttraumatic stress disorder, conduct disorder, antisocial behavior, pathological gambling, aggressive offenses, pathological aggression, deviant sexual behavior, schizoid/avoidant behavioral cluster, and other Axis 1 and Axis 11 mental disorders. These subjects were genotyped for only the DRD2 gene polymorphisms (A1/A1, A1/A2. and A2/A2). In addition, we also genotyped 91 lesser screened controls (excluding only ADHD, pathological aggression, alcohol, tobacco, and other drug abuse and dependence) for the DAT1 9 and 10 alleles. Among these lesser-screened controls, 61 had DRD1 genotyping for the A1/A1, A2/A2, and A1/A2 alleles, and 51 had DBH genotyping for the B1/B1, B1/B2, and B2/B2 alleles. The study protocol was approved by the PATH Foundation IRB and ethics committee, and participants signed approved informed consent forms.

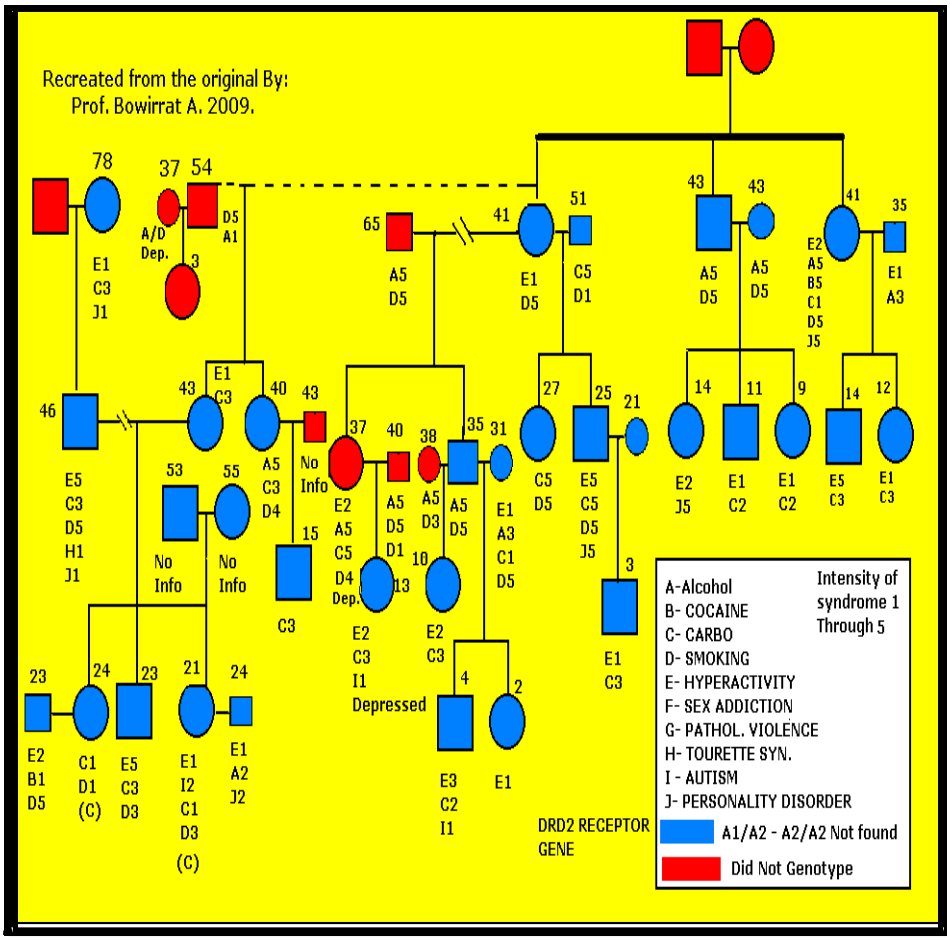
The experimental group consisted of 55 individuals from two independent families. Family A was provided for analysis by the Southeastern Biofeedback and Neurobehavioral Clinic, Knoxville, TN, and Family B was provided for analysis by the Enhancement Institute, Houston, TX (Unique Mind Care. Inc.) RDS diagnosis was available on 42 members from four generations of Family A (Figure 1) and 26 members from five generations of Family B (Table 2 and Figure 2). These included 10 deceased Family A members and three deceased Family B members. A total of 32 Family A and 23 Family B individuals were genotyped. In Family A, there were a total of 14 males and 18 females genotyped at an average age of 31.6 ± 18.9 years, and for Family B there were nine males and 14 females genotyped with average age of 31.5 ± 24.2 years. The members of Family A had been diagnosed with ADHD, and the members of Family B had been diagnosed with substance use disorder. The RDS behaviors observed included alcoholism, cocaine dependence, marijuana abuse, intravenous drug dependence, carbohydrate binging, obesity, smoking, hyperactivity, sex addiction, pathological aggression, Tourette syndrome, autism, criminal activity, gambling, novelty seeking, and personality disorders (see Figures 1 and 2).

2.2. Genotyping

Buccal epithelial cells were collected by cotton swabs for DNA isolation. In some subjects, a blood sample was obtained. In this study we isolated the DNA and analyzed a number of genes. The genes include DRD1, DRD2, DAT1 and DBH. All subjects were genotyped based on a neutral identification number and read without knowledge of the individual being typed. Total genomic DNA was extracted from each coded blood/buccal sample, and aliquots were used for polymerase chain reaction (PCR) analysis. Genotyping was performed by a PCR technique. PCR was performed in 30-μL reaction mixtures containing 1.5 mM $MgCl_2$, 2 mM 2'-deoxynucleotide 5'-triphosphates (dNTPs), 0.5 μM primers, 1 μg of template DNA 1, 5 U of Taq polymerase (Boehringer Mannheim Corp., Indianapolis, IN, USA), and PCR buffer (20 mM Tris-HCL (pH 8.4) and 50 mM KCL. After an initial denaturation

at 94 °C for 4 minutes, the DNA was amplified with 35 cycles of 30 seconds at 94 °C, 30 seconds at 58 °C, and 30 seconds at 72 °C, followed by a final extension step of 5 minutes at 72 °C.

Figure 1. Genotype results of the Dopamine D2 receptor gene (DRD2) polymorphism of family A (n = 32) identified with multiple Reward Deficiency Syndrome (RDS) behaviors.



Dopamine D2 receptor gene. The oligo-nucleotide primers 5'-CCGTCGACCCTTCCTGAGT-GTCATCA-3' and 5'-CCGTCGACGGCTGGCCAAGTTGTCTA-3' were used to amplify a 310-base pair fragment spanning the polymorphic TaqA1site of the DRD2 gene. The D2A1 and D2A2 genotyping was performed by a PCR technique. The PCR product was digested with 5 U of Taq 1 for 22 hours at 65 °C for the Taq1A polymorphism. Digestion products were then resolved on a 3% agarose gel (5 V/cm) containing 0.65 µg/mL ethidium bromide. There were three DRD2 Taq1A genotypes: the predominant homozygote A2/A2, which exhibits three restriction fragments of 180 and 130 bp; the heterozygote A1/A2, which exhibits three restriction fragments of 310, 180, and 130 bp; and the rare homozygote A1/A1, which produces only the uncleaved 310-bp fragment [20].

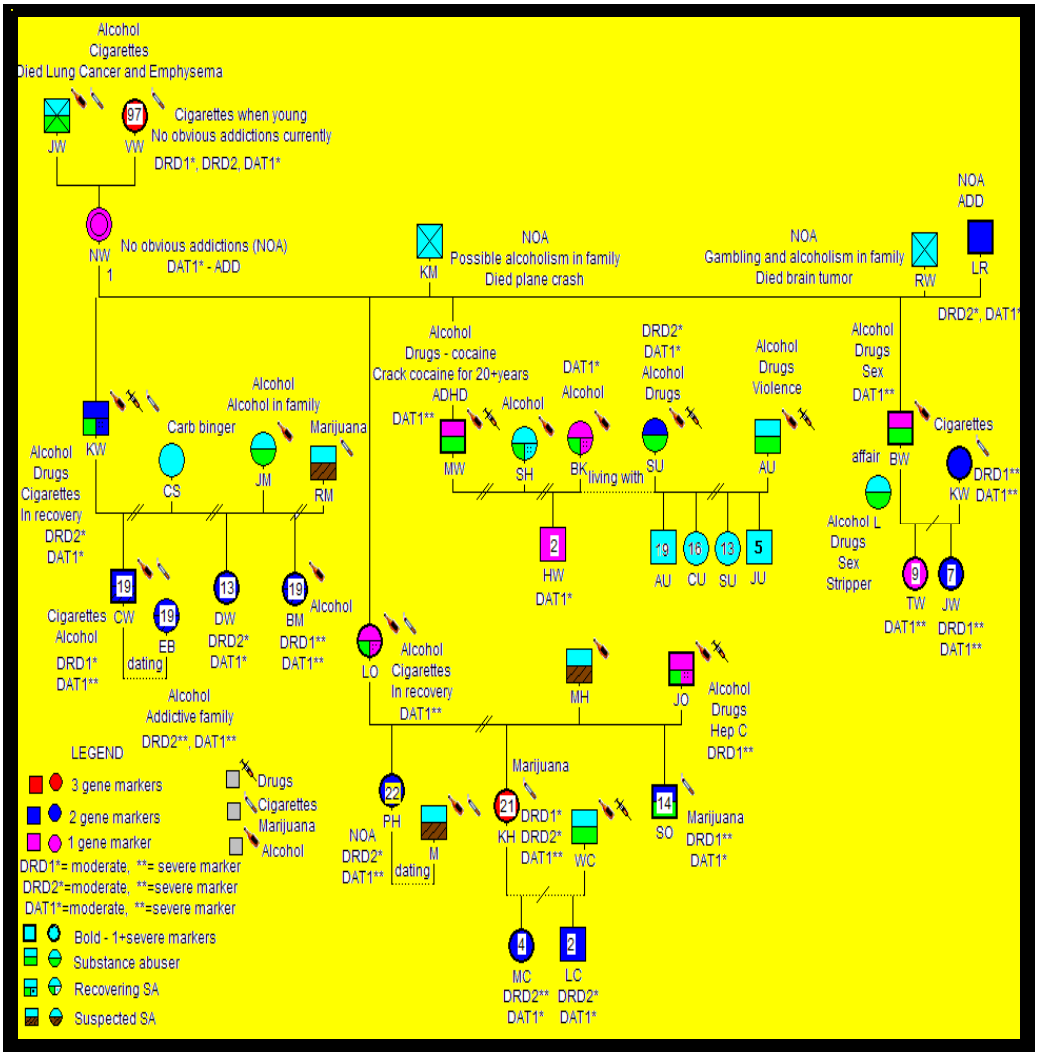
Dopamine transporter gene. DAT1 was genotyped by the technique of Comings *et al.* [5].

Dopamine D1 receptor gene. To examine the DRD1 gene we utilized the Dde I polymorphism consisting of an A to G change in the 5' UTR, tested by the PCR procedure described by Thompson *et al.* [106].

Int. J. Environ. Res. Public Health **2011**, *8*

Dopamine beta-hydroxylase gene. For DBH, the Taq I B polymorphism was genotyped by the technique of d'Amato *et al.* [107].

Figure 2. Association of dopaminergic gene polymorphisms with RDS behaviors in Family B (n = 23).



2.3. Statistical Analyses

Demographic, clinical, laboratory, interview, and questionnaire data were coded and entered into a computer database. DRD1, DRD2, DAT1 and DBH allelic prevalence, obtained by personnel blinded to the aforementioned information, also was coded. All comparisons were based on 2 × 2 contingency tables. Pearson's chi-square statistic was used to identify group differences, with $p < 0.05$ considered statistically significant. In cases where at least one cell of the 2 × 2 contingency table had an expected frequency less than 5, Yates' correction for continuity [108] was applied. To indicate the increase in odds of being gene positive for the experimental group relative to controls, odds ratios with 95% confidence intervals also were calculated. Group sample size of 55 for RDS and 30 for controls

achieve 93% power to detect an odds ratio in the group porportions of 5.00 or more at the 0.05 level. All statistical analyses were performed using SPSS statistical software (SPSS, Inc., Chicago, IL, USA).

3. Results

3.1. Genotyping

Dopamine D2 receptor gene. A large percentage (78.2) of the experimental subjects (43 of 55) carried the DRD2 Taq A1 allele. When compared with "super controls," (1/30 or 3.3% of the controls carried the DRD2A1 allele), the experimental rate was significantly greater ($\chi^2 = 43.6$, $p < 0.001$) with an odds ratio of 103.9 (12.8, 843.2). Similarly, with regard to the DRD2 gene, when the experimental group (n = 55) was compared with unscreened literature controls derived from 15 international studies (LCN) [LCN = 439/3,329 (31.32%)], the experimental rate was significantly greater ($\chi^2 = 187.1$, $p < 0.001$) with an odds ratio of 23.6 (12.3, 45.1) (Table 2). Since the gene positive rate for Family B was lower than that for the combined families, separate experimental *vs*. control comparisons were made using Family B alone. When compared to "super controls", Family B had a significantly greater positive rate ($\chi^2 = 14.7$, $p < 0.001$) with an odds ratio of 26.6 (3.1, 229.3), and a similar result was found when compared to unscreened literature controls ($\chi^2 = 20.7$, $p < 0.001$) with an odds ratio of 6.0 (2.6, 13.8). Based on over 3,000 unscreened subjects, the A1 allele of the DRD2 gene is present in approximately one-third of the American population, whereas the A2 allele is present in two-thirds of the American population [9].

Dopamine transporter gene. We found the less rigorous (no ADHD, alcoholism, substance abuse) 91 screened controls to carry the DAT1 10/10 in 34 subjects (37.4%); the 9/10 in 40 subjects (44.0%) and the 9/9 in 12 subjects (13.2%). With regard to the DAT1 gene, the 10/10 allele was present in 32 of 55 (58.2%) experimental group individuals in the two families. The experimental positive rate for the 10/10 allele was significantly greater ($\chi^2 = 6.0$, $p < 0.015$) with an odds ratio of 2.3 (1.2, 4.6). Moreover, 19 of the 32 probands (59.4%) from Family A (four generations) that were genotyped for the DAT1 gene carried the 10/10 allele, so that Family A had a significantly greater positive rate ($\chi^2 = 4.7$, $p < 0.035$) compared to controls with an odds ratio of 2.5 (1.1, 5.6). In Family B, 57.5% (13 of 23) carried the 10/10 genotype (Table 2 and Figure 1), but owing to the smaller sample size, the Family B positive rate was not significantly different ($\chi^2 = 2.8$, $p = 0.095$) from controls. Based on genotype data for the DAT1 gene on 3,080 subjects, the 480 bp 10/10 allele occurs in approximately 55% of the unscreened American population; the 9/10 occurs in approximately 38% of unscreened Americans; and the rare 9/9 allele occurs in 7% of unscreened Americans [5].

Dopamine D1 receptor gene. Because Comings *et al*. had shown the DA D1 receptor gene to be associated with substance use disorder [95], we analyzed this gene in Family B. When we compared subjects in Family B genotyped for polymorphisms of this gene with 61 controls, we found that in the Family B probands, 65.2 % carried the A1/A2 (15/23), 26.1% the A2/A2 (6/23), and 8.7% the A1/A1 (2/23) genotype (combined homozygosity A1/A1 and A2/A2 = 34.8%). When we compared the experimental homozygosity genotyping result against the 61 controls, for which three (4.9%) were positive for A1/A1, 20 (32.8%) were positive for A2/A2, and the combined homozygosity (A1/A1 and A2/A2) rate was 37.7%, no significant differences were observed ($p > 0.50$). In the American

population, genotyping data revealed that 6% carry the A1/A1 allele (moderately dysfunctional); 35% carry the A2/A2 allele (severe dysfunction); and 60% carry the A1/A2 allele (normal) [5].

Dopamine beta-hydroxylase gene. Comparing the percent prevalence of the DA Beta-hydroxylase gene (DBHB1) in 51 controls [52.9 % (27/51]] with the 32 members genotyped only in Family A [65% (21/32)], DBHB1 was not significantly different ($\chi^2 = 1.3$, $p > 0.25$) (see Table 2). Since we found no significance with this gene in Family A, we decided not to test it in Family B. In the unscreened American population, genotyping data revealed that 21% carry the B1/B1 genotype, 69% carry the B1/B2 genotype and 58% carry the B2/B2 genotype [23,99]. However, the percentage decreases when one screens for no alcohol, drug or tobacco abuse/dependence (B1/B1 = 6%; B1/B2 = 21%; B2/B2 = 24%).

3.2. Findings and Their Implications

Table 2 summarizes the genotype results for dopaminergic genes in Families A and B. The results for each gene polymorphism are presented.

Table 2. Genotype results for dopaminergic genes in Family A and Family B.

Gene and Polymorphism	Percent Prevalence in Non-RDS Group	Percent Prevalence in Super Control Group	Percent Prevalence in RDS Group	Significance level p Value
DRD2-A1 Allele	31.32 (n = 3,143) *	3.3 (n = 30) **	78.2 (n = 55) Family A and B	* $p < 0.001$ ** $p < 0.001$
DAT1-10/10 Allele	37.4 (n = 91) *	Not Applicable	58.18 (n = 55) Family A and B	*$p < 0.015$
DBH-B1 Allele	52.9 (n = 51)	Not Applicable	65.0 (n = 32) Family A	Not significant
DRD1-A1/A1	65.2 (n = 61)	Not Applicable	31.0 (n = 23) Family B	Not significant

Evaluating severity: Genetic Addiction Risk Score (GARS). Interestingly, in Family A, 100% of the subjects had at least one dysfunctional dopaminergic polymorphism. However, in considering the role of dopaminergic gene polymorphisms in RDS behavior, unlike Family A, where 100% of the subjects carried the DRD2 A1 allele, in Family B only 47.8% carried the TaqA1. Therefore, for Family B only, we evaluated severity by utilizing a Genetic Addiction Risk Score (GARS), a multivariate genetic index score methodology (developed by LifeGen, Inc. and Dominion Diagnostics, Inc.; see [109]) to strengthen the predictive value of laboratory testing for genetic predispositions related to disease diagnosis, stratification, prognosis, metabolism, and nutritional response. In this regard, the breakdown of the polymorphic markers was as follows: two subjects carried three polymorphic markers; 13 subjects had two polymorphic markers and eight subjects carried one polymorphic marker.

4. Discussion

4.1. RDS, a Putative Endophenotype: Multiple Behaviors versus a Single Subset

In doing association studies for which an investigator requires a representative control sample for a single RDS psychiatric diagnosis [18,22,24,110-112] or for potential subsets of RDS (see Table 3), the

obvious limitation relates to controls poorly screened for multiple RDS behaviors and other related psychiatric disorders.

Missing behaviors that are part of the RDS subset may be the reason for spurious results [9,87, 113-117] when genotyping for single subsets of RDS behaviors. For example an individual may not drink or use drugs but may have other RDS behaviors like overeating or intensive video gaming. In support of this notion, we found a very strong association of the DA D2 receptor A1 allele (100%) in Family A. In addition, every individual in Family B also has at least one dopaminergic high risk allele (100%) [48% carried the DRD2 A1 allele]. Moreover, in Family B only three adult individuals had no addictive behaviors. When we compared our results in which 55 RDS subjects carried the DRD2 A1 allele at (78.2%) with the results of research by Noble [14] in which 597 severe alcoholic-dependent individuals (49.3%) carried the A1 allele, there was a significant difference between these two groups ($\chi^2 = 16.9$, $p < 0.001$). This demonstrated that the A1allele prevalence increases with multiple RDS behaviors. Here we propose that multifaceted non-specific RDS behaviors should be considered as the true "reward" phenotype (endophenotype) instead of a single subset RDS behavior such as alcoholism. Indeed, this may be a paradigm shift in future association and linkage studies using a different set of statistical methodologies.

Table 3. Correlation of RDS and related neurological and psychiatric disorders with DRD2 SNP (a sampling).

Reward Deficiency Syndrome or Related Disorder	Studies Demonstrating an Association with DRD2 Gene Polymorphism(s)
Pathological Gambling	[4,5,33,61-64,140]
Attention Deficit Hyperactivity Disorder	[2,4,24,99,154,196,207-210]
Post-Traumatic Stress Disorder	[4,176,177,196,212,213]
Eating; Obesity and Related Sequela	[46,133,134,170,178,179,181,182,197,198,200]
Energy Production	[134,180]
Hypertension	[181,182]
Schizophrenia	[12,19,49,112,183]
Early-Onset Sexual Intercourse; Hyper sexuality	[136,203]
Anti-Social Personality	[18,53]
Pathological Aggression	[6,105 ,171,186]
Schizoid-Avoidant Behavior	[143]
Novelty or Sensation Seeking	[26,27,36,83,109,113,144,161]
Substance Abuse	[12,13,20,22,29,46,65,71,90,103,110,114,117, 172-175,193,199,201,202,205]
Heroin Addiction	[13,48,66,111,160]
Nicotine Dependence and Smoking Behavior	[13,17,22,81,91,104,184,185]
Personality Disorders and Crime	[100,186]
Parkinson's Disease	[187,188]
Migraine	[189]
Tourette Syndrome	[5, 99,190]
Huntington's Disease	[191]
Cell Metabolism	[47]
Major Psychoses & Affective Disorder	[192,195,204]
Extraversion and Creativity	[21,23,145]

4.2. "Super" Controls as a Phenotype: Exclusion of Multiple RDS Behaviors

For population-based studies in which the investigator requires a representative control sample, removing confounding cases from the control group may improve chances of finding significant differences between experimental and control groups. This approach, however, may risk a lack of representation in the control group. Even the use of stratified samples (weighting samples) may not be good enough [113-116].

In the case of finding a "pure" phenotype, especially in the psychiatric arena, we really do not know if nature carved out the psychiatric disorders in the same fashion as is seen in the DSM. This is true because behavior is very complex, whereby specific genes for behavioral tendencies (anxiety, impulsivity, compulsivity, harm avoidance, aggressiveness, addiction *etc*.) accounts for only a small risk contribution to the overall phenotype. Therefore, we must shift our emphasis to the "systems biological" approach, which takes into account the inter relationship of dysfunctional behaviors, the polygenic nature of psychiatric disorders, and the environment.

We should consider the established concept of RDS [1-4] to help define this complex array of behaviors associated with molecular dysfunctions. Victims of RDS are at increased vulnerability to addictive activities because they may carry polymorphic genes in dopaminergic pathways [117-122] that result in hypo-dopominergic function [60-62] caused by a reduced number of DA D2 receptors [55,117,118], reduced synthesis of DA (DA beta-hydroxylase) [5], reduced net release of pre-synaptic DA, possibly due to altered synthesis of DA (L-amino-acid decarboxylase) [123], increased synaptic clearance due to a high number of DA transporter sites [92-94,118,120-122] (DA transporter), and low D2 receptor densities [55,56,85,123-127] (DA D2 receptor). The need for a unified set of related symptoms in the affected phenotype is important not only for population-based association studies, but also for linkage analysis. The RDS concept involves, shared genes and behavioral tendencies as summarized in Table 3. While poly-genes are involved [128-145], the common theme in all of these substances and behaviors is that they induce pre-synaptic DA release at the NAc [53,73-77]. Spectrum disorders such as ADHD, Tourette Syndrome, and autism are included due to DA dysregulation [1,5,24,25]. In fact love styles also associate with both serotonergic and dopaminergic gene polymorphisms [146].

A screened control group is essential for uncovering population-based associations where the disease in question may be very common. We know that approximately one-third of the population meet lifetime criteria for common psychiatric disorders according to the results of the Epidemiological Catchment Area survey. Since RDS is a "polygenic disorder" involving multiple genes and many polymorphisms [1-4] and requires a threshold number of polygenes, unaffected individuals in the population also carry some of these genes. AS stated earlier, the DA D2 receptor gene (A1 allele) is present in about one-third of unscreened Americans (29.4% in 3,329 subjects studied up until 2003) [14].

The use of super controls has been criticized by some on the grounds that their relatives will have rates of co-morbid disorders lower than that in the general population and may produce spurious co-aggregation of disorders within families. This argument is valid only if the same psychopathology that is removed from the control group is *not* excluded from among the probands and their relatives.

This provides the rationale to encourage others to begin to carefully select true controls especially when dealing with complex traits such as RDS involving a number of associated gene polymorphisms [88,147-149].

Very few behaviors depend upon a single gene. Complexes of genes (polygenic) drive most of our heredity-based actions [150-154], suggesting that genetic panels or algorithms organized into genetic indexes, such as GARS may be valuable clinically to determine risk. Certainly abnormal functions of these brain systems can be due to specific genetic factors interacting with environmental factors [118-120,122,149,150,152-154]. Understanding the interactions of these components is likely to lead to better treatment.

Because our study began in 1999 when less was known about dopaminergic genes, we did not genotype subjects for the following: D3–5 receptor genes, the MAO-A or MAO-B, or catechol *O*-methyltransferase. However, those will be analyzed in subsequent experiments. When we started these experiments, the specific roles of the dopaminergic genes in brain function remained inconclusive due to the lack of completely selective ligands that could distinguish between the members of the D1-like and D2-like DA receptor families. However, today we are making rapid progress distinguishing among the various DA receptors (see [96]). Our findings, while suggestive, must be interpreted with caution. In terms of inclusion/exclusion criteria, since this was a generational study, all subjects were included in the study without bias.

4.3. Interactive Environmental and Genetic Roles in RDS Behaviors

It is important to consider one of the most important new areas in neurobiology and genetics termed "epigenetics" and its role in RDS. Most recently, our group reviewed the epigenetics of ADHD and detailed the important interaction of environmental elements and gene expression [154]. We are cognizant of the simple mathematical equation P = G + E. whereby, any phenotype (RDS included) is equal to both one's genome and environmental impact. The role of the contribution of genetics to illicit drug abuse was evaluated by van den Bree *et al*. [155] using structural equation, modeling genetic and environmental estimates and DSM-III abuse/dependence for sedatives, opioids, cocaine, stimulants, and cannabis, as well as any other illicit drugs. The authors found genetic influences for most measures, which were strongest for males and for clinical diagnoses of abuse/dependence compared to actual substance use. Most interestingly, common environmental influences played a greater role in use than abuse/dependence, suggesting that the severity of any RDS behavior may have a stronger genetic contribution relative to less severe forms. Moreover, this same group [156] in another study, among Caucasians with alcohol dependence characterized subtypes by differential loading on three dimensions: genetic, general environmental, and dyssocial environmental symptom scales. The mild subtype (60% of men and 66% of women) was distinguished by low mean scores on all three scales; the dyssocial subtype (24% of men and 20% of women) by low mean genetic and general environmental scores but high mean dyssocial environmental scores; and the severe subtype (16% of men and 14% of women) by high scores on the genetic and general environmental scales. Importantly, the severe subtype showed greater comorbid drug dependence and major depression, more treatment seeking, and a higher prevalence of parental alcoholism. Only the severe subtype showed a pattern of scale scores and clinical characteristics suggestive of substantial genetic influence.

It is well known that environmental cues may induce relapse in drug dependent individuals. This phenomenon has been evaluated by Gerasimov *et al*. [157] to better understand the neurochemical mechanisms potentially mediating these cues by measuring NAc DA levels in animals exposed to environmental cues previously paired with cocaine administration. They found that in animals exposed to a cocaine-paired environment, NAc DA increased by 25%. In one study the intake of morphine altered the neurotransmitter turnover of DA differentially as function of passive compared non-passive infusion. Environmental contingent infusion compared to passive infusion of intravenous morphine significantly affected more brain regions up to 5 fold [158] suggesting impact of the environment on neurochemistry. The role of dopaminergic genes as a predictor of risk concerning personality traits has been positively identified in molecular genetic studies. Earlier work in our laboratory identified the relationship between schizoid avoidance [143] as well as impulsive and compulsive behaviors [159]. The work of The *et al*. [160] showing significantly higher frequency for the DRD2 TaqIA polymorphism among addicts (69.9%) compared to control subjects (42.6%; Fisher's exact χ^2, $p < 0.05$) is in agreement with our earlier findings. They also observed that the studied addicts had higher scores for novelty seeking and harm avoidance personality traits but lower scores for reward dependence when compared to control subjects. This has been further supported by the work of Kazantseva *et al*. [161] on personality traits in a sample of 652 healthy individuals (222 men and 430 women) of Caucasian origin (233 Russians and 419 Tatars) from Russia. The subjects' personality traits were assessed with Eysenck Personality Inventory and the Temperament and Character Inventory-125). There were significant effects of ANKK1/DRD2 Taq1A on Neuroticism ($p = 0.016$) and of SLC6A3 rs27072 on Persistence ($p = 0.021$) in both genders. The association between ANKK1/DRD2 Taq1A A2/A2-genotype and higher Novelty Seeking and lower Reward Dependence was shown in men only.

Genetic and environmental influences also are important for the development of alcohol and drug dependence. Exposure to early life stress, has been shown to predict a wide range of psychopathology, including addiction. Enoch [162] has suggested that early life stress can result in permanent neurohormonal and hypothalamic-pituitary-adrenal axis changes, morphological changes in the brain, and gene expression changes in the mesolimbic DA reward pathway, all of which are implicated in the development of addiction. However, he further emphasized that a large proportion of children who have experienced even severe early life stress do not develop psychopathology, indicating that mediating factors such as gene-environment interactions and family and peer relationships are important for resilience. Most interestingly, Israel researchers Raz and Berger [163] published convincing evidence for the role of social interaction as it related to intake of morphine in animals. Specifically, adult Wistar rats housed in short-term isolation (21 days) consumed significantly more morphine solution (0.5 mg/mL) than rats living in pairs, both in one-bottle and in two-bottle tests. They also found that as little as 60-min of daily social-physical interaction with another rat was sufficient to completely abolish the increase in morphine consumption in socially restricted animals. Accordingly, these results indicate that environmental and situational factors influence drug intake in laboratory rats as they do in humans.

Over many years of study, the consensus of the literature has suggested that prevention of drug-seeking relapse could be attenuated by enriched environments. For example, a recent French study by Chauvet *et al*. [164] showed, in animals, the potential "curative" influence of enriched environments in reducing cocaine-induced craving effects, thereby highlighting the importance of positive life conditions in facilitating abstinence and preventing relapse to cocaine addiction.

Int. J. Environ. Res. Public Health **2011**, *8*

There is no doubt that enriched life experiences, as well as reduced early prenatal and post natal stress, have impacts on impulsivity. In addition, reduced release of mesolimbic DA affected both by genes and by the environment, play a significant role in craving behavior and more importantly in relapse [165-169] as well as many RDS behaviors [170-193]

4.4. From Bench to Bedside: Clinical Utility of RDS

Historically, addictive disorders were categorized by the drug the patient was abusing, rather than the underlying neurocontrol circuitry that was being affected [194]. Alcohol and drug dependent individuals were considered to differ so substantially that treatment programs, as well as funding sources and regulatory agencies were distinct and separate. If insurance coverage was available, it was often restricted to the treatment of alcoholism, so that drug addicts often lied about their drug of choice in order to get admitted. Some methadone maintenance programs allowed drug addicts to drink, despite the fact that the leading cause of death at that time prior to viral infections was alcoholic cirrhosis. Self-help groups were organized around the drug of choice, such that if a person with a drug problem went to an Alcoholics Anonymous (AA) meeting, they were told that the meeting was for alcoholic-dependent individuals only and were asked to leave. Many polysubstance abusers who attended AA because they could not relate to Narcotics Anonymous (NA), learned not to volunteer their drug history. Patients on buprenorphine or methadone are still refrained from sponsoring or speaking at NA meetings, secondary to the belief that they are not abstinent (http://www.na.org/?ID= bulletins-bull29).

Over the last 30 years the situation has improved because of our understanding of multiple addictive disorders as a brain disease [195-206]. Many programs have some treatment content on the medical aspects of addiction, although often it remains as an add-on without explicit integration into the other treatment suggestions. Often these presentations are provided by non-physicians in a treatment system that lacks a physician component in their multidisciplinary team, or depends upon medical referral to another agency or provider.

Only a handful of treatment centers have utilized the neurobiology of addiction as an integral part of patient education with explicit integration of RDS into all treatment content especially as it relates ADHD, Posttraumatic stress disorder and drug dependence [207-213]. This section of this paper will share how RDS can assist in patient education and acceptance of treatment recommendations including participating in self help and use of medication assisted therapy. These recommendations are organized around the American Society of Addiction Medicine's (ASAM) six dimensions of care [214] and by treatment chronology.

ASAM's six dimensions help to classify and assess treatment need and placement. They are: intoxication and withdrawal potential; biomedical conditions and complications; emotional behavioral conditions and complications; readiness for change; relapse potential; and recovery environment. Dimension 1 includes the substance use history, the extent of tolerance and physical dependence and the extent of polysubstance use. Clinicians are concerned about the extent of neuroadaptive changes and the severity of the addiction, but patients usually focus upon their drug of choice as the primary focus of intervention. We frequency hear from patients, "I came here to stop using cocaine, not alcohol and marijuana". By reviewing the anatomy of control circuitry, the common pathway of DA release,

and the concept of RDS as an inherited and acquired change in sensitivity and calibration of control circuitry, patients can now move from viewing the treatment providers as prohibitionists to seeing their concerns as medically based. Showing how DA increases in the NAc after natural rewards and by use of alcohol and drugs, patients understanding of the risk of cross addiction improves. Warning patients about continued use of substances that affect reward circuitry is coupled with drug testing to measure patient treatment adherence, especially when the program is providing detoxification and stabilization during early recovery. Documenting by patient history and drug testing that many patients have simultaneous poly substance use and dependence will allow for the program to document the medical necessity for testing and the choice of expanded panels to capture patients attempting to continue drug use that is not detectable by traditional testing. When testing for synthetic cannabinoids became available, we found that over 50% of patients in our adolescent IOP were getting high despite negative THC levels.

Because of the high percentage of patients with co-occurring disorders (ASAM Dimension 2, medical, and Dimension 3, emotional/behavioral conditions and complications), use of RDS as a primary treatment concept allows patients to understand their biological differences and the role of inheritance. Over 80% of patients in our outpatient program have a positive family history of alcoholism and drug addiction, and often as children promised themselves that they would not grow up to be like their parents. Other patients suffer from binge eating, compulsive gambling, "shopaholism", *etc*. A strong emphasis on the genetic aspects of RDS assists the patient in reducing guilt and shame and understanding how they could become something that they never planned on. This strengthens the importance of not over-relying on willpower and learning as primary treatment methods, and provides a medical justification of the counselors and sponsors recommendations. Acknowledging patients' anhedonia during early recovery and incorporating thoughts, feelings and behaviors that increases reward, provides a therapeutic alliance and a potential acceptance by patients of treatment recommendations for proper nutrition, exercise, medications and other enjoyable activities.

By explaining the association with other disorders, RDS allows for the program and the patient to utilize standardized screening techniques for conditions such as ADD, gambling, aberrant sexual behavior as well as for personality profiles of aggressive and antisocial behaviors. Diagnosing patients with an associated condition(s) allows for education and early treatment. We have frequently observed impulsive behavior result in relapses during early recovery, as well as interfering with the quality of life and improved workplace functioning. Treating ADD earlier in recovery can be challenging, especially if the patient is focused upon becoming drug free and views any medications for ADD as part of the addictive process. Explaining how early treatment can help restore neurochemical balances provides a medical explanation that differentiates getting high from experiencing improved reward responsiveness to natural stimuli.

Not only is RDS helpful for the problems associated with ASAM Dimensions 1 through 3, but it also is most important in educating the patient about their disease (ASAM Dimension 4) and directly confronting altering cognition and defense mechanisms such as denial, rationalization, and justification. By explaining the pathophysiology of RDS, patients' understanding of their illness improves significantly, especially in appreciating the subtle details of therapists' recommendations. It is important for clinicians to explicitly connect RDS concepts with AA sayings and slogans so that the patient can understand why and how self-help works and is not simply an "option" in treatment.

Relapse prevention (ASAM Dimension 5) can now be viewed as the processes of altered sensitivity and calibration of neuroadaptive circuitry, which affects hedonic tone, cue reactivity, and executive functioning. Warning patients that entering high risk situations during early recovery (the patient who frequents bars and who decides that it's OK to play pool with his friends at the bar and drink soda), is not simply a matter of choice, but represents unintentional pressure upon altered circuitry. Understanding RDS concepts and the rate of restoration of recovery based neuroadaption allows for the patient to understand that the therapist recommendation is not punitive but protective. Many addicts do not understand that once the alcohol and drugs leave the body that the brain takes a long time to recover and may only reset itself to a genetically determined pre-morbid state. RDS emphasis the dynamics of neuroadaption of recovery and provides hope that improvement is possible, but at a rate of change that is dependent upon the amount of targeted treatment effort and the rate limiting steps from a biological perspective.

ASAM Dimension 6 (recovery environment) emphasizes the environmental impact on treatment effectiveness and encompasses the interactivity of gene expression by environment factors. Patients tend to underestimate the power of the environment in influencing outcome and tend to justify maintenance of their old relationships and living situations as an independent factor. Once patients understand the bidirectional aspect of the recovery environment, their motivation for making difficult changes can be modified by these biologically based factors.

5. Limitations, Caveats and Future Directions

While we agree with the work of Sussman *et al*. [215] and his eloquent PACE model in terms of drug specificity based on many environmental elements, we further suggest that neurochemistry and certain specific genetic polymorphisms may lead to a non-specific hierarchical list of drugs of abuse and behaviors having a common neuro-chemical mechanism such as dopamine release in the NAc. However, this so called RDS phenotype is significantly impacted by availability of drugs,; awareness of drug availability, specific preference for one type over another type (depressant *vs*. stimulant), among other factors.

Additional studies are required prior to any definitive interpretation of these data. We encourage other investigators to extend this work by analyzing many more families for hypodopaminergic genotypes. This should include D1–D5 receptors, MOA-A,B, COMT, as well as other reward genes. Certainly studies involving larger populations even in a few families over many generations (if possible) would strengthen this potentially important concept. Our laboratory is continuing our pursuit to enhance our knowledge base by carrying out linkage analysis to couple hypodopaminergic gene function with RDS behaviors. We encourage other investigators to perform similar experiments.

6. Conclusions

While there have been other reports that help establish the RDS concept [169-171], additional research from multiple laboratories is warranted. The results summarized herein provide preliminary support for the hypothesis that dopaminergic genes, in particular the DRD2 and DAT1 polymorphisms, are significantly associated with the reward-dependent traits [172]. Although based on limited sample sizes, our findings may have direct implications for both the diagnosis and targeted treatment of RDS

behaviors by analyzing the association of these dopaminergic genes and RDS behaviors in the form of proprietary algorithms or GARS. This research underscores the potential involvement of at least D2 receptor dysfunction as an important genetic antecedent to addiction as a disease. In keeping with the notion of common neurogenetic mechanisms, for impulsive, compulsive and addictive disorders, we propose that RDS is a basic phenotype covering many reward behaviors and pertinent psychiatric disorders (including spectrum disorders and Posttraumatic Stress Disorder) that should be included in the future in DSM as a genetic umbrella for many psychiatric diagnoses.

While we are still evaluating these results using linkage analysis (requiring a different set of statistics), our present results suggest a paradigm shift in thinking about selecting appropriate phenotypes (controls and experimental) for reward dependence behaviors (due to reward deficiency). Further confirmation of these results should provide an impetus for appropriate and careful selection and screening of controls especially in reward dependence association studies in order to reduce the possibility of spurious outcomes (e.g., [173,211]).

Conflict of Interest

Kenneth Blum, Roger L. Waite, B. William Downs and Margaret A. Madigan are officers and stock holders of LifeGen, Inc., Lederach, PA, USA. LifeGen Inc. is the worldwide exclusive distributor of products related to patents concerning Reward Deficiency Syndrome. John Giordano and Frank Fornari are LifeGen partners.

Acknowledgements

Support for the writing of this paper came from the US Department of Veterans Affairs Medical Research Service and NIAAA grants R01-AA07112 and K05-AA00219 to MO-B. Additional funding came from grants awarded to PATH Research Foundation from LifeExtension Foundation, LifeGen Inc., Lederach, PA, USA. Funding also came from Electronic Waveform Lab, Huntington Beach, CA, USA, and Dominion Diagnostics, Inc.

References

1. Blum, K.; Cull, J.G.; Braverman, E.R.; Comings, D.E. Reward deficiency syndrome. *Am. Sci.* **1996**, *84*, 132-145.
2. Blum, K.; Braverman, E.R. Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors. *J. Psychoactive Drugs* **2003**, *32(Suppl)*, 1-112.
3. Blum, K.; Sheridan, P.J.; Wood, R.C.; Braverman, E.R.; Chen, T.J.; Cull, J.G.; Comings, D.E. The D2 dopamine receptor gene as a determinant of reward deficiency syndrome. *J. R. Soc. Med.* **1996**, *89*, 396-400.
4. Comings, D.E.; Blum, K. Reward deficiency syndrome: Genetic aspects of behavioral disorders. *Prog. Brain Res.* **2000**, *126*, 325-341.

5. Comings, D.E.; Wu, S.; Chiu, C.; Ring, R.H.; Gade, R.; Ahn, C.; MacMurray, J.P.; Dietz, G.; Muhleman, D. Polygenic inheritance of Tourette syndrome, stuttering, attention deficit hyperactivity, conduct, and oppositional defiant disorder: The additive and subtractive effects of the three dopaminergic genes—DRD2, D beta H, and DAT1. *Am. J. Med. Genet.* **1996**, *67*, 264-288.
6. Chen, T.J.H.; Blum, K.; Matthews, D.; Fisher, L.; Schnautz, N.; Braverman, E.R.; Schoolfield, J.; Downs, B.W.; Blum, S.H.; Mengucci, J.; *et al.* Preliminary association of both the Dopamine D2 Receptor (DRD2) [*Taq1 A1* Allele] and the Dopamine Transporter (DAT1) [480 bp Allele] genes with pathological aggressive behavior, a clinical subtype of Reward Deficiency Syndrome (RDS) in adolescents. *Gene Ther. Mol. Biol.* **2007**, *11*, 93-112.
7. Eisenberg, D.T.; Campbell, B.; Mackillop, J.; Lum, J.K.; Wilson, D.S. Season of birth and dopamine receptor gene associations with impulsivity, sensation seeking and reproductive behaviors. *PLoS One* **2007**, *2*, doi:10.1371/journal.pone.0001216.
8. Berridge, K.C. The debate over dopamine's role in reward: The case for incentive salience. *Psychopharmacology (Berl.)* **2007**, *191*, 391-431.
9. Blum, K.; Liu, Y.; Shriner, R.; Gold, M.S. Reward circuitry dopaminergic activation regulates food and drug craving behavior. *Curr. Pharm. Des*. **2011**, *17*, 1158-1167.
10. Gardner, E.L. Addiction and brain reward and antireward pathways. *Adv. Psychosom. Med*. **2011**, *30*, 22-60.
11. Blum, K.; Gold, M.S. Neuro-chemical activation of brain reward meso-limbic circuitry is associated with relapse prevention and drug hunger: A hypothesis. *Med. Hypotheses* **2011**, *76*, 576-584.
12. Noble, E.P. The D2 dopamine receptor gene: A review of association studies in alcoholism and phenotypes. *Alcohol* **1998**, *16*, 33-45.
13. Noble, E.P. The DRD2 gene in psychiatric and neurological disorder and its phenotypes. *Pharmacogenomics* **2000**, *1*, 309-333.
14. Noble, E.P. D2 Dopamine receptor gene in psychiatric and neurologic disorders and its phenotypes. *Am. J. Med. Genet.* **2003**, *116B*, 103-125.
15. Volkow, N.D.; Fowler, J.S.; Wang, G.J. Role of dopamine in drug reinforcement and addiction in humans: Results from imaging studies. *Behav. Pharmacol.* **2002**, *13*, 355-366.
16. Downs, B.W.; Chen, A.L.C.; Chen, T.J.H.; Waite, R.L.; Braverman, E.R.; Kerner, M.; Braverman, D.; Rhoades, P.; Prihoda, T.J.; Palpmo, T.; *et al.* Nutrigenomic targeting of carbohydrate craving behavior: Can we manage obesity and aberrant craving behavior with neurochemical pathway manipulation by immunological compatible substances (nutrients) using a Genetic Positioning System (GPS) Map? *Med. Hypotheses* **2009**, *73*, 427-434.
17. Vandenbergh, D.J.; O'Connor, R.J.; Grang, M.D.; Jefferson, A.L.; Vogler, G.P.; Strasser, A.A.; Kozlowski, L.T. Dopamine receptor genes (DRD2, DRD3, DRD4) and gene-gene interactions associated with smoking-related behaviors. *Addict. Biol.* **2007**, *12*, 106-116.
18. Wang, T.J.; Huang, S.Y.; Lin, W.W.; Lo, H.Y.; Wu, P.L. Wang, Y.S.; Wu, Y.S.; Ko, H.C.; Shih, J.C.; Lu, R.B. Possible interaction between MAOA and DRD2 genes associated with antisocial alcoholism among Han Chinese men in Taiwan. *Prog. Neuropsychopharmacol. Biol. Psychiatry* **2007**, *31*, 108-114.

19. Arinami, T.; Itokawa, M.; Aoki, J.; Shibuya, H.; Ookubo, Y.; Iwawaki, A.; Ota, K.; Shimizu, H.; Hamaguchi, H.; Toru, M. Further association study on dopamine D2 receptor S311C in schizophrenia and affective disorders. *Am. J. Med. Genet.* **1996**, *67*, 133-138.
20. Blum, K.; Noble, E.P.; Sheridan, P.J.; Montgomery, A.; Ritchie, T.; Jagadeeswaran, P.; Nogami, H.; Briggs, A.H.; Cohn, J.B. Allelic association of human dopamine D2 receptor gene in alcoholism. *JAMA* **1990**, *263*, 2055-2060.
21. Cohen, M.X.; Young, J.; Baek, J.M.; Kessler, C.; Ranganath, C. Individual differences in extraversion and dopamine genetics predict neural reward responses. *Brain Res. Cogn. Brain Res.* **2005**, *25*, 851-861.
22. Preuss, U.W.; Zill, P.; Koller, G.; Bondy, B.; Sokya, M. D2 dopamine receptor gene haplotypes and their influence on alcohol and tobacco consumption magnitude in alcohol-dependent individuals. *Alcohol* **2007**, *42*, 258-266.
23. Reuter, M.; Roth, S.; Holve, K.; Hennig, J. Identification of first candidate genes for creativity: A pilot study. *Brain Res.* **2006**, *1069*, 190-197.
24. Comings, D.E.; Gade-Andavilu, R.; Gonzalez, N.; Wu, S.; Muhlman, D.; Blake, H.; Chiu, F.; Wang, E.; Farwell, K.; Darakjy, S.; *et al.* Multivariate analysis of associations of 42 genes in ADHD, ODD and conduct disorder. *Clin. Genet.* **2000**, *58*, 31-40.
25. Chen, C.K.; Chen, S.L.; Mill, J.; Huang, Y.S.; Lin, S.K.; Curran, S.; Purcell, S.; Sham, P.; Asherson, P. The dopamine transporter gene is associated with attention deficit hyperactivity disorder in a Taiwanese sample. *Mol. Psychiatry* **2003**, *8*, 393-396.
26. Noble, E.P.; Ozkaragoz, T.Z.; Ritchie, T.; Zhang, X.; Bekin, T.R.; Belin, T.R.; Sparkes, R.S. D2 and D4 dopamine receptor polymorphisms and personality. *Am. J. Med. Genet.* **1998**, *81*, 257-267.
27. Han, D.H.; Yoon, S.J.; Sung, Y.H.; Lee, Y.S.; Kee, B.S.; Lyoo, I.K.; Renshaw, P.F.; Cho, S.C. A preliminary study: Novelty seeking, frontal executive function, and dopamine receptor (D2) TaqI A gene polymorphism in patients with methamphetamine dependence. *Compr. Phsyciatry* **2008**, *49*, 387-392.
28. Ratsma, J.E.; van der Stelt, O.; Schoffelmeer, A.N.M.; Westerveld And, A.; Boudewijn Gunning, W. P3 event-related potential, dopamine D2 receptor A1 allele, and sensation-seeking in adult children of alcoholics. *Alcohol Clin. Exp. Res.* **2001**, *25*, 960-967.
29. Hill, S.Y.; Zezza, N.; Wipprecht, G.; Xu, J.; Neiswanger, K. Linkage studies of D2 and D4 receptor genes and alcoholism. *Am. J. Med. Genet.* **1999**, *88*, 676-685.
30. van Holstein, M.; Aarts, E.; van der Schaaf, M.E.; Geurts, D.E.; Verkes, R.J.; Franke, B.; van Schouwenburg, M.R.; Cools, R. Human cognitive flexibility depends on dopamine D2 receptor signaling. *Psychopharmacology (Berl.)* **2011**, *218*, 567-578.
31. Bau, C.H.D.; Almeida, S.; Hutz, M.H. The TaqI A1 allele of the dopamine D2 receptor gene and alcoholism in Brazil: Association and interaction with stress and harm avoidance on severity prediction. *Am. J. Med. Genet.* **2000**, *96*, 302-306.
32. Comings, D.E.; Gade, R.; MacMurray, J.P.; Muhlleman, D.; Peters, W.R. Genetic variants of the human obesity (OB) gene: association with body mass index in young women psychiatric symptoms, and interaction with the dopamine D2 receptor gene. *Mol. Psychiatry* **1996**, *1*, 325-335.

33. Comings, D.E.; Rosenthal, R.J.; Lesieur, H.R.; Rugle, L.J.; Muhleman, D.; Chiu, C.; Dietz, G.; Gade, R. A study of the dopamine D2 receptor gene in pathological gambling. *Pharmacogenetics* **1996**, *6*, 223-234.
34. Koob, G.F. Neurobiology of addiction. Toward the development of new therapies. *Ann. NY Acad. Sci.* **2000**, *909*, 170-185.
35. Epping-Jordan, M.P.; Markou, A.; Koob, G.F. The dopamine D-1 receptor antagonist SCH 23390 injected into the dorsolateral bed nucleus of the stria terminalis decreased cocaine reinforcement in the rat. *Brain Res.* **1998**, *784*, 105-115.
36. Wightman, R.M.; Robinson, D.L. Transient changes in mesolimbic dopamine and their association with "reward". *J. Neurochem.* **2002**, *82*, 721-735.
37. Suhara, T.; Yasuno, F.; Sudo, Y.; Yamamoto, M.; Inoue, M.; Okubo, Y.; Suzuki, K. Dopamine D2 receptors in the insular cortex and the personality trait of novelty seeking. *Neuroimage* **2001**, *13*, 891-895.
38. Hodge, C.W.; Chappelle, A.M.; Samson, H.H. Dopamine receptors in the medial prefrontal cortex influence ethanol and sucrose-reinforced responding. *Alcohol Clin. Exp. Res.* **1996**, *20*, 1631-1638.
39. Hodge, C.W.; Cox, A.A. The discriminative stimulus effects of ethanol are mediated by NMDA and GABA(A) receptors in specific limbic brain regions. *Psychopharmacology (Berl.)* **1998**, *139*, 95-107.
40. Grant, K.A. Emerging neurochemical concepts in the actions of ethanol at ligand-gated ion channels. *Behav. Pharmacol.* **1994**, *5*, 383-404.
41. Althaus, M.; Groen, Y.; Wijers, A.A.; Mulder, L.J.; Minderaa, R.B.; Kema, I.P.; Dijck, J.D.; Hartman, C.A.; Hoekstra, P.J. Differential effects of 5-HTTLPR and DRD2/ANKKI1 polymorphisms on electrocortical measures of error and feedback processing in children. *Clin. Neurophysiol.* **2009**, *120*, 93-107.
42. Rothman, R.B.; Blough, B.E.; Baumann, M.H. Duel dopamine/serotonin releasers as potential medications for stimulant and alcohol addictions. *AAPS J.* **2007**, *9*, E1-E10.
43. Merlo, L.J.; Gold, M.S. Special report—Frontiers in psychiatric research: Addiction research: The state of the art in 2008. *Psychiatr. Times* **2008**, *25*, 52-57.
44. Paczynski, R.P.; Gold, M.S. Cocaine and crack. In *Lowinson and Ruiz's Substance Abuse: A Comprehensive Textbook*, 5th ed.; Ruiz, P., Strain, E., Eds.; Lippincott Williams & Wilkins: Baltimore, MD, USA, 2011.
45. Barnes, J.J.; Dean, A.J.; Nandam, L.S.; O'Connell, R.G.; Bellgrove, M.A. The molecular genetics of executive function: role of monoamine system genes. *Biol. Psychiatry* **2011,** *69*, e127-43.
46. Blum, K.; Braverman, E.R.; Wood, R.C.; Gill, J.; Li, C.; Chen, T.J.; Taub, M.; Montgomery, A.R.; Sheridan, P.J.; Cull, J.G. Increased prevalence of the Taq1 A1 allele of the dopamine receptor gene in obesity with comorbid substance use disorder. *Pharmacogenetics* **1996**, *6*, 297-305.
47. Volkow, N.D.; Chang, L.; Wang, G.J.; Fowler, J.S.; Ding, Y.S.; Sedler, M.; Logan, J.; Franceschi, D.; Gatley, J.; Hitzemann, R.; *et al.* Low level of brain dopamine D2 receptors in methamphetamine abusers: Associations with metabolism in the orbitofrontal cortex. *Am. J. Psychiatry* **2001**, *158*, 2015-2021.

48. Perez de los Cobos, J.; Baiget, M.; Trujols, J.; Sinol, N.; Volpini, V.; Banuls, E.; Calafell, F.; Luquero, E.; del Rio, E.; Alvarez, E. Allelic and genotypic associations of DRD2 TaqI A polymorphism with heroin dependence in Spanish subjects: A case control study. *Behav. Brain Funct.* **2007**, *3*, doi:10.1186/1744-9081-3-25.
49. Schindler, K.M.; Pato, M.T.; Dourado, A.; Macedo, A.; Azevedo, M.H.; Kennedy, J.L.; Pato, C.N. Association and linkage disequilibrium between a functional polymorphism of the dopamine-2 receptor gene and schizophrenia in a genetically homogeneous Portuguese population. *Mol. Psychiatry* **2002**, *7*, 1002-1005.
50. Oscar-Berman, M.; Marinkovic, K. Alcohol: Effects on neurobehavioral functions and the brain. *Neuropsychol. Rev.* **2007**, *17*, 239-257.
51. Dackis, C.A.; Gold, M.S.; Davies, R.K.; Sweeney, D.R. Bromocriptine treatment for cocaine abuse: The dopamine depletion hypothesis. *Int. J. Psychiatry Med.* **1985**, *15*, 125-135.
52. Gold, M.S.; Graham, N.A.; Cocores, J.A.; Nixon, S.J. Editorial: Food addiction? *J. Addict. Med.* **2009**, *3*, 42-45.
53. Rowe, D.C. Genetic and environmental components of antisocial behavior: A study of 265 twin pairs. *Criminology* **1986**, *24*, 513-532.
54. Halbus, M.; Magnusson, T.; Magnusson, O. Influence of 5-HT1B/1D receptors on dopamine in the guinea pig NAc: A microdialysis study. *Neurosci. Lett.* **1997**, *225*, 57-60.
55. Koob, G.F. Alcoholism: Allostasis and beyond. *Alcohol Clin. Exp. Res.* **2003**, *27*, 232-243.
56. Hietata, J.; West, C.; Syvalahti, E.; Nagren, K.; Lehikoinen, P.; Sonninen, P.; Ruotsalainen, U. Striatal D2 dopamine receptor binding characteristics *in vivo* in patients with alcohol dependence. *Psychopharmacology (Berl.)* **1994**, *116*, 285-290.
57. Cools, A.R.; Gingras, M.A.; Nijmegen, J. High and low responders to novelty: A new tool in the search after the neurobiology of drug abuse liability. *Pharmacol. Biochem. Behav.* **1998**, *60*, 151-159.
58. Kuikka, J.T.; Repo, E.; Bergstrom, K.A.; Tupala, E.; Tihonen, J. Specific binding and laterality of human extrastriatal dopamine D2/D3 receptors in the late onset type 1 alcoholic patients. *Neurosci. Lett.* **2000**, *292*, 57-59.
59. Miller, W.B.; Pasta, D.J.; MacMurray, J.; Chiu, C.; Wu, H.; Comings, D.E. Dopamine receptor genes are associated with age at first sexual intercourse. *J. Biosoc. Sci.* **1999**, *31*, 43-54.
60. Davis, C.; Levitan, R.D.; Kaplan, A.S.; Carter, J.; Reid, C.; Curtis, C.; Patte, K.; Hwang, R.; Kennedy, J.L. Reward sensitivity and the D2 dopamine receptor gene: A case-control study of binge eating disorder. *Prog. Neuropsychopharmacol. Biol. Psychiatry.* **2008**, *32*, 620-628.
61. Comings, D.E.; Gade-Andavolu, R.; Gonzalez, N.; Wu, S.; Muhleman, D.; Chen, C.; Koh, P.; Farwell, K.; Blake, H.; Dietz, G.; *et al.* The additive effect of neurotransmitter genes in pathological gambling. *Clin. Genet.* **2001**, *60*, 107-116.
62. Ibanez, A.; Blanco, C.; Donahue, E.; Lesieur, H.R.; Perez de Castro, I.; Fernandez-Piqueras, J.; Saiz-Ruiz, J. Psychiatric comorbidity in pathological gamblers seeking treatment. *Am. J. Psychiatry* **2001**, *158*, 1733-1735.
63. Reuter, J.; Raedler, T.; Rose, M.; Hand, I.; Glasher, J.; Buchel, C. Pathological gambling is linked to reduced activation of the mesolimbic system. *Nat. Neurosci.* **2005**, *8*, 147-148.

64. Koepp, M.J.; Gunn, R.N.; Lawrence, A.D.; Cunningham, V.J.; Dagher, A.; Jones, T.; Brooks, D.J.; Bench, C.J.; Grasby, P.M. Evidence for striatal dopamine release during a video game. *Nature* **1998**, *393*, 266-268.
65. Noble, E.P.; Syndilko, K.; Fitch, R.J.; Ritchie, T.; Bohlman, M.C.; Guth, P.; Sheridan, P.J.; Montgomery, A.; Heinzmann, C.; Sparkes, R.S.; *et al.* D2 dopamine receptor Taq1 A alleles in medically ill alcoholic and nonalcoholic patients. *Alcohol Alcohol.* **1994**, *129*, 729-744.
66. Li, Y.; Shao, C.; Zhang, D.; Zhao, M.; Lin, L.; Yan, P.; Xie, Y.; Jiang, K.; Jin, L. The effect of dopamine D2, D5 receptor and transporter (SLC6A3) polymorphisms on the cue-elicited heroin craving in Chinese. *Am. J. Med. Genet. B Neuropsychiatr. Genet.* **2006**, *141B*, 269-273.
67. Little, K.Y.; Zang, L. Striatal dopaminergic abnormalities in human cocaine users. *Am. J. Psychiatry* **1999**, *156*, 238-245.
68. Hutchinson, K.E.; McGeary, J.; Smolen, A.; Bryan, A.; Swift, R.M. The DRD4 VNTR polymorphism moderates craving after alcohol consumption. *Health Psychol*. **2002**, *21*, 139-146.
69. Adler, C.M.; Elman, I.; Weisenfield, N.; Kestler, L.; Pickar, D.; Breier, A. Effects of acute metabolic stress on striatal dopamine release in healthy volunteers. *Neuropsychopharmacology* **2000**, *22*, 545-550.
70. Blum, K.; Payne, J. *Alcohol and the Addictive Brain*; The Free Press (Simon and Schuster): New York, NY, USA, 1991.
71. Blum, K.; Noble, E.P.; Sheridan, P.J.; Finley, O.; Montgomery, A.; Ritchie, T.; Ozkaragoz, T.; Fitch, R.J.; Sadlack, F.; Sheffield, D.; *et al.* Association of the A1 allele of the D2 dopamine receptor gene with severe alcoholism. *Alcohol* **1991**, *8*, 409-416.
72. Carboni, E.; Silvagni, A.; Rolando, M.T.P.; Di Chiara, G. Stimulation of *in vivo* dopamine transmission in the bed nucleus of stria terminalis by reinforcing drugs. *J. Neurosci.* **2000**, *20*, 1-5.
73. Di Chiara, G. Drug addiction as dopamine-dependent associative learning disorder. *Eur. J. Pharmacol.* **1999**, *375*, 13-30.
74. Di Chiara, G. NAc shell and core dopamine: Differential role in behavior and addiction. *Behav. Res.* **2002**, *137*, 75-114.
75. Di Chiara, G.; Tanda, G.; Bassare, V.; Pontieri, F.; Acquas, E.; Fenu, S.; Cadoni, C.; Carboni, E. Drug addiction as a disorder of associative learning. Role of nucleus accumbens shell/extended amygdale dopamine. *Ann. NY Acad. Sci.* **1999**, *877*, 461-485.
76. Di Chiara, G.; Imperato, A. Drugs abused by humans preferentially increase synaptic dopamine concentrations in the mesolimbic systems of freely moving rats. *Proc. Natl. Acad. Sci. USA* **1988**, *85*, 5274-5278.
77. Gessa, G.; Mutoni, F.; Coller, M.; Vargin, L.; Mercer, G. Low doses of ethanol activate dopaminergic neurons in the ventral tegmental area. *Brain Res.* **1986**, *48*, 201-203.
78. Eshleman, A.J.; Henningsen, R.A.; Neve, K.A.; Janowsky, A. Release of dopamine via the human transporter. *Mol. Pharmacol.* **1994**, *45*, 312-316.
79. Piechota, M.; Korostynski, M.; Solecki, W.; Gieryk, A.; Slezak, M.; Bilecki, W.; Ziolkowska, B.; Kostrzewa, E.; Cymerman, I.; Swiech, L.; *et al.* The dissection of transcriptional modules regulated by various drugs of abuse in the mouse striatum. *Genome Biol.* **2010**, *11*, doi:10.1186/gb-2010-11-5-r48.

80. Neville, M.J.; Johnstone, E.C.; Walton, R.T. Identification and characterization of ANKK1: A novel kinase gene closely linked to DRD2 on chromosome band 11q23.1. *Hum. Mutat.* **2004**, *23*, 540-545.
81. Huang, W.; Payne, T.J.; Ma, J.Z.; Beuten, J.; Dupont, R.T.; Inohara, N.; Li, M.D. Significant association of ANKK1 and detection of a functional polymorphism with nicotine dependence in an African-American sample. *Neuropsychopharmacology* **2009**, *34*, 319-330.
82. Noble, E.P.; Blum, K.; Ritchie, T.; Montgomery, A.; Sheridan, P. Allelic associations of the D2 dopamine receptor gene with receptor-binding characteristics. *Arch. Gen. Psychiatry.* **1991**, *48*, 648-654.
83. Montag, C.; Markett, S.; Basten, U.; Stelzel, C.; Fiebach, C.; Canli, T.; Reuter, M. Epistasis of the DRD2/ANKK1 Taq Ia and the BDNF Val66Met polymorphism impacts novelty seeking and harm avoidance. *Neuropsychopharmacology* **2010**, *35*, 1860-1867.
84. Jönsson, E.G.; Nöthen, M.M.; Grünhage, F.; Farde, L.; Nakashima, Y.; Propping, P.; Sedvall, G.C. Polymorphisms in the dopamine D2 receptor gene and their relationships to striatal dopamine receptor density of healthy volunteers. *Mol. Psychiatry* **1999**, *4*, 290-296.
85. Duan, J.; Wainwright, M.S.; Comeron, J.M.; Saitou, N.; Sanders, A.R.; Gelernter, J.; Gejman, P.V. Synonymous mutations in the human dopamine receptor D2 (DRD2) affect mRNA stability and synthesis of the receptor. *Hum. Mol. Genet*. **2003**, *12*, 205-216.
86. Hirvonen, M.; Laakso, A.; Någren, K.; Rinne, J.O.; Pohjalainen, T.; Hietala, J. C957T polymorphism of the dopamine D2 receptor (DRD2) gene affects striatal DRD2 availability *in vivo*. *Mol. Psychiatry* **2004**, *9*, 1060-1061.
87. Hill, S.Y.; Hoffman, E.K.; Zezza, N.; Thalamuthu, A.; Weeks, D.E.; Matthews, A.G.; Mukhopadhyay, I. Dopaminergic mutations: Within-family association and linkage in multiplex alcohol dependence families. *Am. J. Med. Genet. B Neuropsychiatr. Genet*. **2008**, *147B*, 517-526.
88. Vandenbergh, D.J. Molecular cloning of neurotransmitter transporter genes: Beyond coding region of cDNA. *Meth. Enzymol*. **1998**, *296*, 498-514.
89. Michelhaugh, S.K.; Fiskerstrand, C.; Lovejoy, E.; Bannon, M.J.; Quinn, J.P. The dopamine transporter gene (SLC6A3) variable number of tandem repeats domain enhances transcription in dopamine neurons. *J. Neurochem*. **2001**, *79*, 1033-1038.
90. Guindalini, C.; Howard, M.; Haddley, K.; Laranjeira, R.; Collier, D.; Ammar, N.; Craig, I.; O'Gara, C.; Bubb, V.J.; Greenwood, T.; *et al.* A dopamine transporter gene functional variant associated with cocaine abuse in a Brazilian sample. *Proc. Natl. Acad. Sci. USA* **2006**, *103*, 4552-4557.
91. Vandenbergh, D.J.; Bennett, C.J.; Grant, M.D.; Strasser, A.A.; O'Connor, R.; Stauffer, R.L.; Vogler, G.P.; Kozlowski, L.T. Smoking status and the human dopamine transporter variable number of tandem repeats (VNTR) polymorphism: Failure to replicate and finding that never-smokers may be different. *Nicotine Tob. Res*. **2002**, *4*, 333-340.
92. Cook, E.H., Jr.; Stein, M.A.; Krasowski, M.D.; Cox, N.J.; Olkon, D.M.; Kieffer, J.E.; Leventhal, B.L. Association of attention-deficit disorder and the dopamine transporter gene. *Am. J. Hum. Genet.* **1995**, *56*, 993-998.

93. Lee, S.S.; Lahey, B.B.; Waldman, I.; Van Hulle, C.A.; Rathouz, P.; Pelham, W.E.; Loney, J.; Cook, E.H. Association of dopamine transporter genotype with disruptive behavior disorders in an eight-year longitudinal study of children and adolescents. *Am. J. Med. Genet. Neuropsychiatr. Genet.* **2007**, *144B*, 310-317.
94. Self, D.W. Regulation of drug-taking and -seeking behaviors by neuroadaptations in the mesolimbic dopamine system. *Neuropharmacology* **2004**, *47(Suppl 1)*, 242-255.
95. Comings, D.E.; Gade, R.; Wu, S.; Chiu, C.; Dietz, G.; Muhleman, D.; Saucier, G.; Ferry, L.; Rosenthal, R.J.; Lesieur, H.R.; *et al.* Studies of the potential role of the dopamine D1 receptor gene in addictive behaviors. *Mol. Psychiatry* **1997**, *2*, 44-56.
96. Luo, Z.; Volkow, N.D.; Heintz, N.; Pan, Y.; Du, C. Acute cocaine induces fast activation of D1 receptor and progressive deactivation of D2 receptor striatal neurons: *In vivo* optical microprobe [Ca^{2+}]i imaging. *J. Neurosci.* **2011**, *31*, 13180-13190.
97. Lobo, M.K.; Covington, H.E., 3rd.; Chaudhury, D.; Friedman, A.K.; Sun, H.; Damez-Werno, D.; Dietz, D.M.; Zaman, S.; Koo, J.W.; Kennedy, P.J.; *et al.* Cell type-specific loss of BDNF signaling mimics optogenetic control of cocaine reward. *Science* **2010**, *330*, 385-390.
98. Egeland, J.A. A genetic study of manic-depressive disorder among the old order Amish of Pennsylvania. *Pharmacopsychiatry* **1988**, *21*, 74-75.
99. Comings, D.E. Clinical and molecular genetics of **ADHD** and Tourette syndrome. Two related polygenic disorders. *Ann. N. Y. Acad. Sci.* **2001**, *931*, 50-83.
100. Hess, C.; Reif, A.; Strobel, A.; Boreatti-Hümmer, A.; Heine, M.; Lesch, K.P.; Jacob, C.P. A functional dopamine-beta-hydroxylase gene promoter polymorphism is associated with impulsive personality styles, but not with affective disorders. *J. Neural. Transm.* **2009**, *116*, 121-130.
101. McClernon, F.J.; Fuemmeler, B.F.; Kollins, S.H.; Kail, M.E.; Ashley-Koch, A.E. Interactions between genotype and retrospective ADHD symptoms predict lifetime smoking risk in a sample of young adults. *Nicotine Tob. Res.* **2008**, *10*, 117-127.
102. Barkley, R.A.; Smith, K.M.; Fischer, M.; Navia, B. An examination of the behavioral and neuropsychological correlates of three ADHD candidate gene polymorphisms (DRD4 7+, DBH TaqI A2, and DAT1 40 bp VNTR) in hyperactive and normal children followed to adulthood. *Am. J. Med. Genet. B Neuropsychiatr. Genet.* **2006**, *141B*, 487-498.
103. Kirsch, P.; Reuter, M.; Mier, D.; Lonsdorf, T.; Stark, R.; Gallhofer, B.; Vaitl, D.; Hennig, J. Imaging gene-substance interactions: The effect of the DRD2 TaqIA polymorphism and the dopamine agonist bromocriptine on the brain activation during the anticipation of reward. *Neurosci. Lett.* **2006**, *405*, 196-201.
104. McKinney, E.F.; Walton, R.T.; Yudkin, P.; Fuller, A.; Haldar, N.A.; Mant, D.; Murphy, M.; Welsh, K.I.; Marshall, S.E. Association between polymorphisms in dopamine metabolic enzymes and tobacco consumption in smokers. *Pharmacogenetics* **2000**, *10*, 483-491.

105. Chen, T.J.; Blum, K.; Mathews, D.; Fisher, L.; Schnautz, N.; Braverman, E.R.; Schoolfield, J.; Downs, B.W.; Comings, D.E. Are dopamonergic genes involved in a predisposition to pathological aggression? Hypothesizing the importance of "super normal controls" in psychiatric genetic research of complex behavioral disorders. *Med. Hypotheses* **2005**, *65*, 703-707.
106. Thompson, M.; Comings, D.E.; Feder, L.; George, S.R.; O'Dowd, B.F. Mutation screening of the dopamine D1 receptor region gene in Tourette's syndrome and alcohol dependent patients. *Am. J. Med. Genet.* **1998**, *81*, 241-244.
107. D'Amato, T.; Leboyer, M.; Malafosse, A.; Samolyk, D.; Lamouroux, A.; Junien, C.; Mallet, J. Two TaqI dimorphic sites at the human beta-hydroxylase locus. *Nucleic Acids Res.* **1989**, *17*, 5871.
108. Siegel, S. *Nonparametric Statistics for the Behavioral Sciences*; McGraw Hill Book Co: New York, NY, USA, 1957; pp. 104-111.
109. Blum, K.; Giordano, J.; Morse, S.; Liu, Y.; Tan, J.; Bowirrat, A.; Smolen, A.; Waite, R.; Downs, W.; Madigan, M.; *et al.* Genetic Addiction Risk Score (GARS) analysis: Exploratory development of polymorphic risk alleles in poly-drug addicted males. *IIOAB J.* **2010**, *1*, 1-14.
110. Smith, L.; Watson, M.; Gates, S.; Ball, D.; Foxcroft, D. Meta-analysis of the association of the Taq1A polymorphism with the risk of alcohol dependency: A HuGE gene-disease association review. *Am. J. Epidemiol*. **2008**, *167*, 125-138.
111. Lawford, B.R.; Young, R.M.; Noble, E.P.; Sargent, J.; Rowell, J.; Shadforth, S.; Zhang, X.; Ritchie, T. The D(2) dopamine receptor A(1) allele and opioid dependence: Association with heroin use and response to methadone treatment. *Am. J. Med. Genet*. **2000**, *6*, 592-598.
112. Monakhov, M.; Golimbet, V.; Abramova, L.; Kaleda, V.; Karpov, V. Association study of three polymorphisms in the dopamine D2 receptor gene and schizophrenia in the Russian population. *Schizophr. Res.* **2008**, *100*, 302-307.
113. López, J.; López, V.; Rojas, D.; Carrasco, X.; Rothhammer, P.; García, R.; Rothhammer, F.; Aboitiz, F. Effect of psychostimulants on distinct attentional parameters in attentional deficit/hyperactivity disorder. *Biol. Res.* **2004**, *37*, 461-468.
114. Gorwood, P. Contribution of genetics to the concept of risk status for alcohol dependence. *J. Soc. Biol.* **2000**, *194*, 43-49.
115. Shao, H.; Burrage, L.C.; Sinasac, D.S.; Hill, A.E.; Ernest, S.R.; O'Brien, W.; Courtland, H.W.; Jepsen, K.J.; Kirby, A.; Kulbokas, E.J.; Daly, M.J.; Broman, K.W.; Lander, E.S.; Nadeau, J.H. Genetic architecture of complex traits: Large phenotypic effects and pervasive epistasis. *Proc. Natl. Acad. Sci. USA* **2008**, *105*, 19910-19914.
116. Hill, S.Y.; Neiswanger, K. The value of narrow psychiatric phenotypes and "Super" normal controls. In *Handbook of Psychiatric Genetics*; Blum, K., Noble, E.P., Eds.; CRC Press: Boca Raton, FL, USA, 1997.
117. Uhl, G.; Blum, K.; Noble, E.; Smith, S. Substance abuse vulnerability and D2 receptor genes. *Trends Neurosci.* **1993**, *16*, 83-88.
118. Mash, D.C.; Staley, J.K.; Doepel, F.M.; Young, S.N.; Ervin, F.R.; Palmour, R.M. Altered dopamine transporter densities in alcohol-preferring vervet monkeys. *Neuroreport* **1996**, *7*, 457-462.
119. Stice, E.; Spoor, S.; Bohon, C.; Small, D.M. Relation between obesity and blunted striatal response to food is moderated by Taq1A A1 allele. *Science* **2008**, *322*, 448-452.

120. Tupala, E.; Hall, H.; Bergstrom, K.; Mantere, T.; Rasanen, P.; Sarkioja, T.; Tiihonen, J. Dopamine D2 receptors and transporter in type 1 and 2 alcoholics measured with human whole hemisphere autoradiography. *Hum. Brain Mapp.* **2003**, *20*, 91-102.
121. Tupala, E.; Hall, H.; Bergstrom, K.; Sarkioja, T.; Rasanen, P.; Mantere, P.; Callaway, J.; Hiltunen, J.; Tiihonen, J. Dopamine D(2)/D(3)-receptor and transporter densities in NAc and amygdale of type 1 and type 2 alcoholics. *Mol. Psychiatry* **2001**, *6*, 261-267.
122. Tupala, E.; Kuikka, J.T.; Hall, H.; Bergstrom, K.; Sarkioja, T.; Rasanen, P.; Mantere, T.; Hiltunen, J.; Vepsalainen, J.; Tiihonen, J. Measurement of the striatal dopamine transporter density and heterogeneity in type 1 alcoholics using human whole hemisphere autoradiography. *Neuroimage* **2001**, *1*, 87-94.
123. Laakso, A.; Pohjalainen, T.; Bergman, J.; Kajander, J.; Haaparanta, M.; Solin, O.; Syvalahti, E.; Hietala, J. The A1 allele of the human D2 dopamine receptor gene is associated with increased activity of striatal L-amino acid decarboxylase in healthy subjects. *Pharmacogenet. Genomics* **2005**, *15*, 387-391.
124. Sambataro, F.; Fazio, L.; Taurisano, P.; Gelao, B.; Porcelli, A.; Mancini, M.; Sinibaldi, L.; Ursini, G.; Masellis, R.; Caforio, G.; *et al.* DRD2 genotype-based variation of default mode network activity and of its relationship with striatal DAT binding. *Schizophr Bull.* **2011**, doi:10.1093/schbul/sbr128.
125. Miller, N.S.; Gold, M.S. A hypothesis for a common neurochemical basis for alcohol and drug disorders. *Psychiatr. Clin. North Am.***1993**, *15*, 105-117.
126. Parsian, A.; Todd, R.D.; Devor, E.J.; O'Malley, K.L.; Suarez, B.K.; Reich, T.; Cloninger, C.R. Alcoholism and alleles of the human D2 dopamine receptor locus: Studies of Association and linkage. *Arch. Gen. Psychiatry* **1991**, *48*, 655-663.
127. Repo, E.; Kuikka, J.T.; Bergstrom, K.A.; Karbu, J.; Hiltunen, J.; Tiihonen, J. Dopamine transporter and D2-receptor density in late-onset alcoholism. *Psychopharmacology (Berl.)* **1999**, *147*, 314-318.
128. Oscar-Berman, M.; McNamara, P.; Freedman, M. Delayed-Response Tasks: Parallels between experimental ablation studies and finding in patients with frontal lesions. In *Frontal Lobe Function and Injury*; Levin, H.S., Eisenberg, H.M., Benton, A.L., Eds.; Oxford University Press: New York, NY, USA, 1991; pp. 231-255.
129. Walsh, S.L.; Cunningham, K.A. Serotonergic mechanisms involved in the discriminative stimulus, reinforcing and subjective effects of cocaine. *Psychopharmacology (Berl.*) **1997**, *130*, 41-58.
130. Pilla, M.; Perachon, S.; Sautel, F.; Garrido, F.; Mann, A.; Wermuth, C.G.; Schwartz, J.C.; Everitt, B.J.; Sokoloff, P. Selective inhibition of cocaine-seeking behavior by a partial dopamine D3 receptor agonist. *Nature* **1990**, *400*, 371-375.
131. Myers, R.D.; Robinson, D.E. Mmu and D2 receptor antisense oligonucleotides injected in nucleus accumbens suppress high alcohol intake in genetic drinking HEP rats. *Alcohol* **1999**, *18*, 225-233.

132. Xu, K.; Lichterman, D.; Kipsky, R.H.; Franke, P.; Liu, X.; Hu, Y.; Cao, L.; Schwab, S.G.; Wildenauer, D.B.; Bau, C.H.; *et al.* Association of specific haplotypes of D2 dopamine receptor gene with vulnerability to heroin dependence in distinct populations. *Arch. Gen. Psychiatry* **2004**, *61*, 567-606.
133. Noble, E.P.; Noble, R.E.; Ritchie, T.; Grandy, D.K.; Sparkes, R.S. D1 receptor gene and obesity. *Int. J. Eat. Disord.* **1994**, *15*, 205-217.
134. Epstein, L.H.; Temple, J.L.; Neaderhiser, B.J.; Salis, R.J.; Erbe, R.W.; Leddy, J.J. Food reinforcement, the dopamine D2 receptor genotype, and energy intake in obese and nonobese humans. *Behav. Neurosci.* **2007**, *121*, 877-886.
135. Meredith, J.M.; Moffatt, A.C.; Auger, A.P.; Snyder, G.L.; Greengard, P.; Blaustein, J.D. Mating-related stimulation induced phosphorylation of dopamine-and cyclic AMP-regulated phosphoprotein-32 in progestin receptor-containing areas in the female rat brain. *J. Neurosci.* **1998**, *18*, 10189-10195.
136. Miller, W.B.; Pasta, D.J.; MacMurray, J.; Chiu, C.; Wu, H.; Comings, D.E. Dopamine receptor genes are associated with age at first intercourse. *J. Biosoc. Sci*. **1999**, *31*, 43-54.
137. Pani, L.; Porcella, A.; Gessa, G.L. The role of stress in the pathophysiology of the dopaminergic system. *Mol. Psychiatry* **2000**, *5*, 14-21.
138. Binczycka-Anholcer, M.N. Aggressive behavior and the public health condition. *Wiad Lek.* **2002**, *55*, 627-632.
139. Pontius, A.A. Forensic significance of the limbic psychotic trigger reaction. *Bull. Am. Acad. Psychiatry Law* **1996**, *24*, 125-134.
140. Kamarajan, C.; Rangaswamy, M.; Tang, Y.; Chorlian, D.B.; Pandey, A.K.; Roopesh, B.N.; Manz, N.; Saunders, R.; Stimus, A.T.; Porjesz, B. Dysfunctional reward processing in male alcoholics: An ERP study during a gambling task. *J. Psychiatr. Res.* **2010**, *44*, 576-590.
141. Gebhardt, C.; Leisch, F.; Schussler, P.; Fuchs, K.; Stompe, T.; Sieghart, W.; Hornik, K.; Kasper, S.; Aschauer, H.N. Non-association of dopamine D4 and D2 receptor genes with personality in healthy individuals. *Psychiatr. Genet.* **2000**, *10*, 131-137.
142. Sugiura, M.; Kawashima, R.; Nakagawa, M.; Okada, K.; Sato, T.; Goto, R.; Sato, K.; Ono, S.; Schormann, T.; Zilles, K.; *et al.* Correlation between human personality and neuronal activity in cerebral cortex. *Neuroimage* **2000**, *11*, 541-546.
143. Blum, K.; Braverman, E.R.; Wu, S.; Cull, J.G.; Chen, T.J.; Gill, J.; Wood, R.; Eisenberg, A.; Sherman, M.; Davis, K.R.; *et al.* Association of polymorphisms of dopamine D2 receptor (DRD2) and dopamine transporter (DAT1) genes with schizoid/avoidant behavior (SAB). *Mol. Psychiatry* **1997**, *2*, 239-246.
144. Keltikangas-Jarvinene, L.; Pulkki-Raback, L.; Eiovainio, M.; Raltakari, O.T.; Vilkari, J.; Lehtimaki, T. DRD2 C32806T modifies the effect of child-rearing environment on adulthood novelty seeking. *Am. J. Med. Genet. B Neuropsychiatr. Genet.* **2009**, *150B*, 389-394.
145. Golimbet, V.E.; Alfimova, M.V.; Gritsenko, I.K.; Ebstein, R.P. Relationship between dopamine system genes and extraversion and novelty seeking. *Neurosci. Behav. Physiol*. **2007**, *37*, 601-606.
146. Emanuele, E.; Brondino, N.; Pesenti, S.; Re, S.; Geroldi, D. Genetic loading on human loving styles. *Neuro Endocrionl. Lett.* **2007**, *28*, 815-821.

147. Parsons, L.H.; Weiss, F.; Koob, G.F. Serotonin1b receptor stimulation enhances dopamine-mediated reinforcement. *Psychopharmacology (Berl.)* **1996**, *128*, 150-160.
148. Wise, R.A. Brain reward circuitry: Insight from unsensed incentives. *Neuron* **2002**, *36*, 229-240.
149. Yadid, G.; Pacak, K.; Kopin, I.J.; Goldstein, D.S. Endogenous serotonin stimulates striatal dopamine release in conscious rats. *J. Pharmacol. Exp. Ther*. **1994**, *270*, 1158-1165.
150. Hill, S.Y. Alternative strategies for uncovering genes contributing to alcoholism risk: Unpredictable findings in a genetic wonderland. *Alcohol* **1998**, *16*, 53-59.
151. Bouchard, T.J. Genes, environment, and personality. *Science* **1994**, *9*, 415-421.
152. Thut, G.; Schultz, W.; Roelcke, U.; Nienhusmeier, M.; Missimer, J.; Maguire, R.P.; Leenders, K.L. Activation of the human brain by monetary reward. *Neuroreport* **1997**, *8*, 1225-1228.
153. Clark, W.R.; Grunstein, M. Are we hardwired? The role of genes. In *Human Behavior*; Oxford University Press: New York, NY, USA, 2000.
154. Archer, T.; Oscar-Berman, M.; Blum, K. Epigenetics in developmental disorder: ADHD and endophenotypes. *J. Genet. Syndr. Gene Ther.* **2011**, *2*, doi:10.4172/2157-7412.1000104.
155. van den Bree, M.B.; Johnson, E.O.; Neale, M.C.; Pickens, R.W. Genetic and environmental influences on drug use and abuse/dependence in male and female twins. *Drug Alcohol Depend.* **1998**, *52*, 231-241.
156. Johnson, E.O.; van den Bree, M.B.; Gupman, A.E.; Pickens, R.W. Extension of a typology of alcohol dependence based on relative genetic and environmental loading. *Alcohol Clin. Exp. Res*. **1998**, *22*, 1421-1429.
157. Gerasimov, M.R.; Schiffer, W.K.; Gardner, E.L.; Marsteller, D.A.; Lennon, I.C.; Taylor, S.J.; Brodie, J.D.; Ashby, C.R., Jr.; Dewey, S.L. GABAergic blockade of cocaine-associated cue-induced increases in nucleus accumbens dopamine. *Eur. J. Pharmacol.* **2001**, *414*, 205-209.
158. Smith, J.E.; Co, C.; Freeman, M.E.; Lane, J.D. Brain neurotransmitter turnover correlated with morphine-seeking behavior of rats. *Pharmacol. Biochem. Behav.* **1982**, *16*, 509-519.
159. Blum, K.; Sheridan, P.J.; Wood, R.C.; Braverman, E.R.; Chen, T.J.; Comings, D.E. Dopamine D2 receptor gene variants: Association and linkage studies in impulsive-addictive-compulsive behaviour. *Pharmacogenetics* **1995**, *5*, 121-141.
160. The, L.K.; Izzuddin, A.F.; Fazleen Haslinda, M.H.; Zakaria, Z.A.; Salleh, M.Z. Tridimensional personalities and polymorphism of dopamine D2 receptor among heroin addicts. *Biol. Res. Nurs.* **2011**, doi:10.1177/1099800411405030.
161. Kazantseva, A.; Gaysina, D.; Malykh, S.; Khusnutdinova, E. The role of dopamine transporter (SLC6A3) and dopamine D2 receptor/ankyrin repeat and kinase domain containing 1 (DRD2/ANKK1) gene polymorphisms in personality traits. *Prog. Neuropsychopharmacol. Biol. Psychiatry* **2011**, *35*, 1033-1040.
162. Enoch, M.A. The role of early life stress as a predictor for alcohol and drug dependence. *Psychopharmacology (Berl.)* **2011**, *214*, 17-31.
163. Raz, S.; Berger, B.D. Social isolation increases morphine intake: Behavioral and psychopharmacological aspects. *Behav. Pharmacol*. **2010**, *21*, 39-46.

164. Chauvet, C.; Lardeux, V.; Goldberg, S.R.; Jaber, M.; Solinas, M. Environmental enrichment reduces cocaine seeking and reinstatement induced by cues and stress but not by cocaine. *Neuropsychopharmacology* **2009**, *34*, 2767-2778.
165. Rhodes, T. Risk environments and drug harms: A social science for harm reduction approach. *Int. J. Drug Policy* **2009**, *20*, 193-201.
166. Perry, J.L.; Carroll, M.E. The role of impulsive behavior in drug abuse. *Psychopharmacology (Berl.)* **2008**, *200*, 1-26.
167. Kippin, T.E.; Szumlinski, K.K.; Kapasova, Z.; Rezner, B.; See, R.E. Prenatal stress enhances responsiveness to cocaine. *Neuropsychopharmacology* **2008**, *33*, 769-782.
168. Abreu-Villaça, Y.; Queiroz-Gomes Fdo, E.; Dal Monte, A.P.; Filgueiras, C.C.; Manhães, A.C. Individual differences in novelty-seeking behavior but not in anxiety response to a new environment can predict nicotine consumption in adolescent C57BL/6 mice. *Behav. Brain Res*. **2006**, *167*, 175-182.
169. Lu, L.; Xu, N.J.; Ge, X.; Yue, W.; Su, W.J.; Pei, G.; Ma, L. Reactivation of morphine conditioned place preference by drug priming: Role of environmental cues and sensitization. *Psychopharmacology (Berl.)* **2002**, *59*, 125-132.
170. Blum, K.; Chen, A.L.C.; Chen, T.J.H.; Rhoades, P.; Prihoda, T.J.; Downs, B.W.; Waite, R.L.; Williams, L.; Braverman, E.R.; Braverman, D.; *et al.* LG839: Anti-obesity effects and polymorphic gene correlates of reward deficiency syndrome. *Adv. Ther.* **2008**, *25*, 894-913.
171. Suzuki, H.; Han, S.D.; Lucas, L.R. Chronic passive exposure to aggression decreases D(2) and 5-HT(1B) receptor densities. *Physiol. Behav.* **2010**, *99*, 562-570.
172. Noble, E.P. Addiction and its reward process through polymorphisms of the D2 dopamine receptor gene: A review. *Eur. Psychiatry* **2000**, *15*, 79-89.
173. Lohoff, F.W.; Bloch, P.J.; Hodge, R.; Nall, A.H.; Ferraro, T.N.; Kampman, K.M.; Dackis, G.A.; O'Brien, C.P.; Pettinati, H.M.; Oslin, D.W. Association analysis between polymorphisms in dopamine D2 receptor (DRD2) and dopamine transporter (DAT1) genes with cocaine dependence. *Neurosci. Lett.* **2010**, *473*, 87-91.
174. Yang, B.Z.; Kranzler, H.R.; Zhao, H.; Gruen, J.R.; Luo, X.; Gelernter, J. Haplotypic variants in DRD2, ANKK1, TTc12, and NCAM1 are associated with comorbid alcohol and drug dependence. *Alcohol Clin. Exp. Res.* **2008**, *32*, 2117-2127.
175. Volkow, N.D.; Wang, G.J.; Begleiter, H.G.; Porjesz, B.; Fowler, J.S.; Telang, F.; Wong, C.; Ma, Y.; Logan, J.; Goldstein, R.; *et al.* High levels of dopamine D2 receptors in unaffected members of alcoholic families. *Arch. Gen. Psychiatry* **2006**, *63*, 999-1008.
176. Young, R.M.; Lawford, B.R.; Noble, E.P.; Kann, B.; Wilkie, A.; Ritchie, T.; Arnold, L.; Shadforth, S. Harmful drinking in military veterans with post-traumatic stress disorder: Association with the D2 dopamine receptor A1 allele. *Alcohol Alcohol.* **2002**, *37*, 451-456.
177. Lawford, B.R.; McD Young, R.; Noble, E.P.; Kann, B.; Arnold, L.; Rowell, J.; Ritchie, T.L. D2 dopamine receptor gene polymorphism: Paroxetine and social functioning in posttraumatic stress disorder. *Eur. Neuropsychopharmacol.* **2003**, *13*, 313-320.
178. Comings, D.E.; Flanagan, S.D.; Dietz, G.; Muhleman, D.; Knell, E.; Grysin, R. The dopamine D2 receptor (DRD2) as a major gene in obesity and height. *Biochem. Med. Metab. Biol.***1993**, *50*, 176-185.

179. Jenkinson, C.P.; Hanson, R.; Cray, K.; Wiedrich, C.; Knowler, W.C.; Bogardus, C.; Baier, L. Associations of dopamine D2 receptor polymorphisms Ser311Cys and TaqIA with obesity or type 2 diabetes mellitus in Pima Indians. *Int. J. Obes. Relat. Metab. Disord.* **2000**, *24*, 1233-1238.
180. Tataranni, P.A.; Baier, L.; Jenkinson, C.; Harper, I.; Del parigi, A.; Bogardus, C. A Ser311Cys mutation in the human dopamine receptor D2 gene is associated with reduced energy expenditure. *Diabetes* **2001**, *50*, 901-904.
181. Thomas, G.N.; Tomlinson, B.; Critchley, J.A. Modulation of blood pressure and obesity with the dopamine D2 receptor gene TaqI polymorphism. *Hypertension* **2000**, *36*, 177-182.
182. Thomas, G.N.; Critchley, J.A.; Tomlinson, B.; Cockram, C.S.; Chan, J.C. Relationships between the TaqI polymorphism of the dopamine D2 receptor and blood pressure in hyperglycaemic and normoglycaemic Chinese subjects. *Clin. Endocrinol. (Oxf.)* **2001**, *55*, 605-611.
183. de Haan, L.; van Bruggen, M.; Lavalaye, J.; Booij, J.; Dingemans, P.M.; Linszen, D. Subjective experience and D2 receptor occupancy in patients with recent-onset schizophrenia treated with low-dose olanzapine or haloperidol: A randomized, double-blind study. *Am. J. Psychiatry* **2003**, *160*, 303-309.
184. Spitz, M.R.; Shi, H.; Yang, F.; Hudmon, K.S.; Jiang, H.; Chamberlain, R.M.; Amos, C.I.; Wan, Y.; Cinciripini, P.; Hong, W.K.; *et al.* Case-control study of the D2 dopamine receptor gene and smoking status in lung cancer patients. *J. Natl. Cancer Inst.* **1998**, *90*, 358-363.
185. Vandenbergh, D.J.; O'Connor, R.J.; Grant, M.D.; Jefferson, A.L.; Vogler, G.P.; Strasser, A.A.; Kozlowski, L.T. Dopamine receptor genes (DRD2, DRD3, and DRD4) and gene-gene interactions associated with smoking-related behaviors. *Addict. Biol.* **2007**, *12*, 106-116.
186. Guo, G.; Roettger, M.E.; Shih, J.C. Contributions of the DAT1 and DRD2 genes to serious and violent delinquency among adolescents and young adults. *Hum. Genet.* **2006**, *121*, 125-136.
187. Nakajima, T.; Nimura, T.; Yamaguchi, K.; Ando, T.; Itoh, M.; Yoshimoto, T.; Shirane, R. The impact of stereotactic pallidal surgery on the dopamine D2 receptor in Parkinson disease: A positron emission tomography study. *J. Neurosurg.* **2003**, *98*, 57-63.
188. Stoessl, A.J.; de la Fuente-Fernandez, R. Dopamine receptors in Parkinson's disease: Imaging studies. *Adv. Neurol.* **2003**, *91*, 65-71.
189. Peroutka, S.J.; Price, S.C.; Wilhoit, T.L.; Jones, K.W. Comorbid migraine with aura, anxiety, and depression is associated with dopamine D2 receptor (DRD 2) Ncol alleles. *Mol. Med.* **1998**, *4*, 14-21.
190. Lee, C.C.; Chou, I.C.; Tsai, C.H.; Wang, T.R.; Li, T.C.; Tsai, F.J. Dopamine receptor D2 gene polymorphisms are associated in Taiwanese children with Tourette syndrome. *Pediatr. Neurol.* **2005**, *33*, 272-276.
191. Vetter, J.M.; Jehle, T.; Heinemeyer, J.; Franz, P.; Behrens, P.F.; Jackisch, R.; Landwehrmeyer, G.B.; Feuerstein, T.J. Mice transgenic for exon 1 of Huntington's disease: Properties of cholinergic and dopaminergic pre-synaptic function in the striatum. *J. Neurochem.* **2003**, *85*, 1054-1063.
192. Serretti, A.; Lattuada, E.; Lorenzi, C.; Lilli, R.; Smeraldi, E. Dopamine receptor D2 Ser/Cys 311 variant is associated with delusion and disorganization symptomatology in major psychoses. *Mol. Psychiatry* **2000**, *5*, 270-274.

193. Lowinson, J.; Ruiz, P.; Millman, R.; Langrod, J. *Substance Abuse: A Comprehensive Textbook*, 3rd ed.; William & Wilkins: Philadelphia, PA, USA, 1997.
194. Panagis, G.; Nomikos, G.G.; Miliaressis, E.; Chergui, K.; Kastellakis, A.; Svensson, T.H.; Spyraki, C. Ventral pallidum self-stimulation induces stimulus dependent increase in c-fos expression in reward-related brain regions. *Neuroscience* **1997**, *77*, 175-186.
195. Smith, M.; Wasmuth, J.; McPherson, J.D. Cosegregation of an 11q22.3–9p22 translocation with affective disorder: Proximity of the dopamine D2 receptor gene relative to the translocation breakpoint. *Am. J. Hum. Genet*. **1989**, *45*, A220.
196. Comings, D.E.; Comings, B.G.; Muhleman, D.; Dietz, G.; Shahbahrami, B.; Tast, D.; Knell, E.; Kocsis, P.; Baumgarten, R.; Kovacs, B.W.; *et al.* The dopamine D2 receptor locus as a modifying gene in neuropsychiatric disorders. *JAMA* **1991**, *266*, 1793-1800.
197. Joranby, L.; Frost Peneda, K.; Gold, M.S. Addiction to food and brain reward systems. *Sex. Addict. Compul.* **2005**, *12*, 201-217.
198. Hoebel, B.G.; Avena, N.M.; Bocarsly, M.E.; Rada, P. Natural addiction: A behavioral and circuit model based on sugar addiction in rats. *J. Addict. Med.* **2009**, *3*, 33-41.
199. Hammond, J.C.; Gold, M.S. Caffeine dependence, withdrawal, overdose and treatment: A review. *Direct. Psychiatry* **2008**, *28*, 177-189.
200. Volkow, N.D.; Wise, R.A. How can drug addiction help us understand obesity? *Nat. Neurosci.* **2005**, *8*, 555-560.
201. Kleiner, K.D.; Gold, M.S.; Frost-Pineda, K.; Lenz-Brunsman, B.; Perri, M.G.; Jabocs, W.S. Body mass index and alcohol use. *J. Addict. Dis.* **2004**, *23*, 105-118.
202. Warren, M.; Forst-Pineda, K.; Gold, M. Body mass index and marijuana use. *J. Addict. Dis.* **2005**, *24*, 95-100.
203. Merlo, L.L.; Carnes, S.; Carnes, P.J.; Gold, M.S. Hypersexuality disorders: Addiction, compulsion, or impulse behaviors? *Biol. Psychiatry* **2008**, *63*, S1-S301.
204. Bruijnzeel, A.W.; Repetto, M.; Gold, M.S. Neurobiological mechanisms in addictive and psychiatric disorders. *Psychiatr. Clin. North Am.* **2004**, *27*, 661-674.
205. Bruijnzeel, T.; Gold, M.S. The role of corticotrophin-releasing factor-like peptides in cannabis, nicotine, and alcohol dependence. *Brain Res. Rev.* **2005**, *49*, 505-528.
206. Gold, M.S. *Dual Diagnosis: Nosology, Diagnosis, and Treatment Confusion—Chicken or Egg?* Haworth Press: New York, NY, USA, 2007.
207. Volkow, N.D.; Wang, G.J.; Kollins, S.H.; Wigal, T.L.; Newcorn, J.H.; Telang, F.; Fowler, J.S.; Zhu, W.; Logan, J.; Ma, Y.; *et al.* Evaluating dopamine reward pathway in ADHD: Clinical implications. *JAMA* **2009**, *302*, 1084-1091.
208. Nyman, E.S.; Ogdie, M.N.; Loukola, A.; Varilo, T.; Taanila, A.; Hurtig, T.; Moilanen, I.K.; Loo, S.K.; McGough, J.J.; Järvelin, M.R.; *et al.* ADHD candidate gene study in a population-based birth cohort: Association with DBH and DRD2. *J. Am. Acad. Child. Adolesc. Psychiatry* **2007**, *46*, 1614-1621.
209. Faraone, S.V.; Perlis, R.H.; Doyle, A.E.; Smoller, J.W.; Goralnick, J.J.; Holmgren, M.A.; Sklar, P. Molecular genetics of attention-deficit/hyperactivity disorder. *Biol. Psychiatry* **2005**, *57*, 1313-1323.

210. Gizer, I.R.; Ficks, C.; Waldman, I.D. Candidate gene studies of ADHD: A meta-analytic review. *Hum. Genet*. **2009**, *126*, 51-90.
211. Lu, R.B.; Ko, H.C.; Chang, F.M.; Yin, S.J.; Pakstis, A.J.; Kidd, J.R.; Kidd, K.K. No association between alcoholism and multiple polymorphism at the dopamine D2 receptor gene (DRD2) in three distinct Taiwanese populations. *Biol. Psychiatry* **1996**, *39*, 419-429.
212. Comings, D.E.; Muhleman, D.; Gysin, R. Dopamine D2 receptor (DRD2) gene and susceptibility to posttraumatic stress disorder: A study and replication. *Biol. Psychiatry* **1996**, *40*, 368-372.
213. Bowirrat, A.; Chen, T.J.; Blum, K.; Madigan, M.; Bailey, J.A.; Chuan Chen, A.L.; Downs, B.W.; Braverman, E.R.; Radi, S.; Waite, R.L.; *et al.* Neuro-psychopharmacogenetics and neurological antecedents of posttraumatic stress disorder: Unlocking the mysteries of resilience and vulnerability. *Curr. Neuropharmacol.* **2010**, *8*, 335-358.
214. Lee, D.M. *ASAM Patient Placement Criteria for the Treatment of Substance Related Disorders*, 2nd ed. (revised); American Society of Addiction Medicine: Chevy Chase, MD, USA, 2001.
215. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health*. **2011**, *8*, 3399-3415.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Griffin, K.W.; Scheier, L.M.; Acevedo, B.; Grenard, J.L.; Botvin, G.J. Long-Term Effects of Self-Control on Alcohol Use and Sexual Behavior among Urban Minority Young Women. *Int. J. Environ. Res. Public Health* **2012**, *9*, 1-23; doi:10.3390/ijerph9010001.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Long-Term Effects of Self-Control on Alcohol Use and Sexual Behavior among Urban Minority Young Women

Kenneth W. Griffin [1,*], Lawrence M. Scheier [2], Bianca Acevedo [1], Jerry L. Grenard [3] and Gilbert J. Botvin [1]

[1] Department of Public Health, Weill Cornell Medical College, New York, NY 10065, USA; E-Mails: bia2006@med.cornell.edu (B.A.); gjbotvin@med.cornell.edu (G.J.B.)
[2] LARS Research Institute, Las Vegas, NV 89135, USA; E-Mail: scheier@cox.net
[3] School of Community & Global Health, Claremont Graduate University, Claremont, CA 91773, USA; E-Mail: jerry.grenard@cgu.edu

* Author to whom correspondence should be addressed; E-Mail: kgriffin@med.cornell.edu; Tel.: +1-646-962-8056; Fax: +1-646-962-0284.

Received: 28 September 2011; in revised form: 11 December 2011 / Accepted: 19 December 2011 / Published: 23 December 2011

Abstract: High risk alcohol use and sexual behaviors peak in young adulthood and often occur in the same individuals. Alcohol use has been found to impair decision-making and contribute to high risk sexual activity. However, the association between alcohol use and risky sexual behavior may also reflect enduring individual differences in risk taking, sociability, self-control, and related variables. Both behaviors can serve similar functions related to recreation, interpersonal connection, and the pursuit of excitement or pleasure. The present study examined the extent to which high risk drinking and sexual behavior clustered together in a sample of urban minority young adult women, a demographic group at elevated risk for negative outcomes related to sexual health. We tested whether psychosocial functioning measured at the beginning of high school predicted classes of risk behaviors when girls were tracked longitudinally into young adulthood. Latent class analysis indicated three distinct profiles based on high risk drinking and sexual behavior (*i.e.*, multiple sex partners) in young adulthood. The largest class (73% of the sample) reported low levels of risky drinking and sexual behavior. The next largest class (19%) reported high risk drinking and low risk sexual behavior, and the smallest class (8%) reported high levels of both behaviors. Compared to women from other racial/ethnic groups, black women were more likely to be categorized in the high risk drinking/low risk

sex class. Multinomial logistic regression indicated that self-control in adolescence had a broad and enduring protective effect on risk behaviors eight years later and was associated with a greater probability of being in the low risk drinking/low risk sex class. Findings are discussed in terms of understanding the phenotypic expressions of risk behavior as they relate to early psychosocial development and the long-term protective function of self-control in reducing high risk drinking and sexual behaviors.

Keywords: alcohol; sexual behavior; young adulthood; self-control; minority; latent class analysis

1. Introduction

Prevalence of high risk drinking and high risk sexual behaviors peak during late adolescence and young adulthood. In 2010, epidemiological evidence from the National Survey on Drug Use and Health [1] revealed that alcohol use was highest among young adults from 21 to 25 years of age, with 45.5% of these individuals reporting binge drinking (five or more drinks on the same occasion) and 18% reporting heavy drinking (binge drinking on 5 or more days) in the past month. Alcohol abuse among young adults contributes a variety of negative outcomes such as unintentional injuries and deaths, traffic fatalities, sexual assault, academic failure, interpersonal aggression, and psychiatric problems [2-5]. High risk sexual behaviors and the resulting health effects also disproportionately affect young people. Almost half of all new sexually transmitted infections (STIs) in the US occur among 15 to 24 year olds [6], and half of all new HIV infections in the US occur in young people aged 24 years or under [7].

1.1. Co-Morbidity of High Risk Drinking and Sexual Behavior

Research has shown that patterns of high risk alcohol use and sexual behavior often co-occur in the same individuals. Several studies have shown that alcohol abuse is associated with an increased risk for unwanted pregnancy and sexually transmitted infections [8,9]. However, research also shows that the relationship between the two behaviors is complex. Studies have examined patterns of alcohol use and sexual behavior either at the level of *global overlap* or *situational overlap* [10]. Global overlap is the extent to which an individual engaging in one behavior is more likely to engage in another behavior [11]. Studies assessing global overlap generally report positive associations between indices of drinking, sexual frequency, and high risk sex [12-14]. *Situational overlap* is a more refined level of analysis that examines whether an individual engaging in one behavior on a specific occasion is more likely to engage in another behavior on the same occasion. Findings from situation or event-level studies show a more nuanced relationship between alcohol use and risky sex. In a comprehensive review on drinking and risky sexual behavior among college students, Cooper [8] distinguished between *indiscriminate sex behaviors* (e.g., casual, non-steady, multiple, or unknown partners, failure to discuss risk topics prior to intercourse) or *failure to take protective actions* (e.g., lack of condom use or birth control). Event-level studies have shown that alcohol can affect indiscriminate high risk sexual

behaviors, particularly sex with non-steady partners [14], but its ability to predict failure to take protective action is more complex and conditional.

Conceptual models linking alcohol use and sexual behaviors emphasize the role of *perceived norms*, *expectancies* regarding drinking and sex, *cognitive impairment* due to alcohol consumption, and *motives* surrounding drinking and sex [15-19]. Models of *perceived norms* propose that the extent to which an individual engages in high risk drinking and/or sex is a function of their perceptions of the prevalence of the behavior among people in general or people like them in particular. Individuals who believe that alcohol use prior to sex is common or normative will be more likely to engage in these behaviors compared to individuals who don't share these perceptions. An *expectancies* approach emphasizes the importance of what an individual expects to happen as a result of drinking (e.g., perceived benefits/costs). In a value-expectancy framework, when perceived positive outcomes outweigh negative ones, this provides an impetus for greater behavioral involvement (more drinking before sex). A *cognitive impairment* model suggests that alcohol-induced deficits in cognitive functioning limit one's ability to evaluate risks, such that risks associated with sex are less likely to be perceived, encoded, and fully processed when intoxicated. A *drinking motives* model focuses on the psychological functions of alcohol consumption for an individual in a particular context. For example, an individual may observe that their own alcohol consumption facilitates social interaction that may lead to sex, and thus drink as a way to disinhibit their behavior and reduce feelings of awkwardness in social situations.

This brief review demonstrates that both empirical research and psychosocial theory have focused extensively on the links between high risk drinking and sexual behaviors. However, the association between these behaviors may be spurious to the extent that both are manifestations of long-standing individual differences in risk-taking propensity, social confidence, self-control, and related variables [14,20,21]. Alcohol use and sexual activity both typically involve social activity and may serve similar functions related to recreation, relaxation, interpersonal connectedness, and the pursuit of excitement or pleasure. They are both appetitive behaviors associated with an urge to consume in order to satisfy a physical or psychological need. Because of these similar influences and functions, one might expect high risk drinking and sexual behaviors to occur in the same set of individuals or result from similar etiologic processes.

1.2. The Development and Expression of Self-Control

A central component in understanding appetitive behavior is the concept of self-control, which has been defined as the deliberate, conscious, effortful control over behavior, attention, thoughts, emotions, performance, and impulses [22-24]. When people exercise self-control they are more deliberative and thoughtful in their decision-making process and thus self-control may contribute independently to decisions to avoid alcohol consumption and high risk sexual behaviors. In fact, research has demonstrated that self-control is a robust protective factor for a number of social and health risk behaviors, particularly during adolescence [25,26]. Conversely, low self-control is significantly associated with a variety of negative outcomes including illicit drug use, obesity, and depressive symptoms [27,28]. Self-control develops out of a complex interaction of biological, psychological, and social factors. Innate factors related to self-control include underlying neural, physiological, and

genetic variables as well as temperament [21,29,30]. Social and environmental factors such as caregiving support, sibling and peer relationships, and social norms play an important role in the development of self-control [31,32]. A number of studies have shown that maladaptive family functioning in childhood (e.g., parent mental illness or substance abuse; family violence; physical, emotional, or sexual abuse) predisposes individuals to poor self-control in adulthood [33]. Conversely, factors such as social support, parental monitoring, and appropriate discipline provide a structured social environment that promotes self-control among children and adolescents [34]. Furthermore, social and environmental factors may interact with genetic influences to produce low self-control [35].

Wills and colleagues have described how temperament factors that develop in early childhood predict later self-control and substance use in adolescence [21]. According to their conceptual model, temperament factors such as the propensity for activity, emotionality, attention, sociability, and inhibition provide a substrate for the later development of self-control during later childhood and adolescence. Because temperament reflects the *style* of typical behavior for an individual, rather than its *content*, the effect of temperament on the development of self-control and engagement in risk behavior is shaped by a combination of socializing agents in one's environment (e.g., parents, teachers and peers) and individual-level psychosocial risk and protective factors. As individuals mature in cognitive and social skills during later childhood and adolescence, temperamental characteristics result in phenotypic expressions of complex self-control skills such as goal-setting, impulse control, and delay of gratification. These regulatory functions, in combination with environmental and social factors, specify how self-control is exhibited and whether it is protective regarding risky behavior [36,37]. For example, low self-control may be linked to the onset or escalation of substance use by increasing vulnerability to negative life events, deviant peers, or poor academic competence [26,38-40].

1.3. A Focus on Urban Minority Women

National surveillance data show that patterns of risk behavior differ across demographic subgroups of adolescents and young adults. Much of the research focus on alcohol use among young adults has been on college students because the frequency and quantity of alcohol consumption among those who attend college typically surpass rates among nonstudents during the college years [41,42]. College students have also been the focus of a substantial amount of research on the patterns and health consequences of high risk sexual behavior [43]. Studies of college student populations are limited in that many often focus on fairly homogenous samples of middle class, white youth. There is a need for more research on the epidemiology, etiology, and consequences of high risk drinking and sex among other demographic groups. Indeed, research has documented consistently higher rates of teen pregnancy, HIV and other STIs among racial/ethnic minority groups in the US compared to whites [44,45]. A recent CDC report [46] revealed that 46% of people diagnosed with HIV in the US in 2009 were black/African American and that high-risk heterosexual contact among women accounted for more than half of HIV/AIDS cases. Among women diagnosed with AIDS in the US in 2009, 78% were black/African American. Thus, it is important to examine individual-level psychosocial factors among urban minority women in order to increase our understanding of the causes of high risk behaviors and to inform the development of more effective preventive interventions for this group.

Int. J. Environ. Res. Public Health **2012**, *9*

1.4. Study Goals

The goals of the present study were to examine whether there are distinct classes of high risk drinking and high risk sex among urban minority young adult women, a demographic group at high risk for negative outcomes related to sexual health. We sought to determine the extent to which high risk drinking and high risk sexual behavior cluster together or occur independently of one another in this population. Further, we examined whether several domains of psychosocial functioning potentially relevant for both behaviors (*i.e.*, self-control, risk taking, social confidence, and self-esteem) were efficient predictors of class membership over a period of time from the beginning of high school to young adulthood. If adolescent risk and protective factors contribute in a similar fashion to high risk drinking and sexual behavior, it may help explain why these two behaviors cluster together among some individuals.

2. Methods

2.1. Sample

Participants in the present study were part of a larger school-based drug abuse and violence prevention trial conducted with a sample of predominantly minority urban youth. For the present analysis, we included girls from schools that were randomized to the control group condition in order to examine developmental hypotheses in the absence of intervention. Participants were included in the analysis if they were present in the 9th grade and also present at the last follow-up assessment conducted in young adulthood (N = 692). The 9th grade sample included girls (N = 1,233), with a mean age of 14.6 (SD = 0.47), from 21 New York City public and parochial middle schools. Approximately 46% of participants received free lunch at school, 31% lived in mother-only households, and 48% lived with both parents. The racial/ethnic composition was 54% black, 21% Hispanic, 5% white, 5% Asian, 4% American Indian, and 11% reported other or mixed racial self-identification.

At the time of the young adult follow-up interview, 38% of participants reported being single, 39% had a girlfriend/boyfriend, 15% were in some type of committed marital or significant partner relationship, and 8% were engaged. The mean age of participants at the young adult assessment was 22.8 years (range 21–26). Almost three-quarters of the sample (70%) reported not having any children, 21% had one child, and 9% had two or more children. Less than 11% of the sample had not received a high school diploma, 53% had received a high school diploma or equivalent, 12% had an associate degree, and 24% had a bachelor's degree or higher. In terms of occupational status, 37% said they were employed part-time, 35% said they had one full-time job, and 28% said they were not employed. About one third of participants (34%) indicated that they earned less than $5,000 a year, 21% earned between $5,000 and %10,000, 18% between $10,000 and $20,000, 14% between $20,000 and $30,000, and 9% reported income of over $30,000 (3.7% did not answer this question).

Int. J. Environ. Res. Public Health **2012**, *9*

2.2. Procedure

In the 9th grade, students completed a self-report questionnaire that assessed a wide range of substance use behaviors (*i.e.*, alcohol, cigarettes, marijuana, and inhalants) and psychosocial variables. Unique identification numbers were pre-coded onto each survey rather than student names in order to ensure confidentiality, and each pre-coded survey was distributed to the appropriate student by trained data collectors. These unique ID codes were used to match student surveys over the course of the study, using a master list connecting the IDs to the student's name that was kept under lock and key by the investigator. Students were informed that their responses would not be made available to school personnel, teachers, or parents. Questionnaires were administered during regular classroom periods over a two-day period by a team of several data collectors, and all students completed the same version of the questionnaire.

Participants were followed-up once as young adults in 2009 to 2010 and were mailed a packet of information requesting their participation in a telephone survey. The packet included a brief description of the continuing study, a 90-day calendar to be used during a scheduled telephone interview, along with $2 and an offer of additional compensation ($40 to $60) upon completion of a phone interview. Participants were provided with a toll-free telephone number in order to complete the interview. During the telephone interview, participants were provided with a brief description of the study and were asked for oral consent to participate in a telephone interview which included questions about their alcohol and drug use, history of sexual partners, participation in high risk sexual behavior, a variety of psychosocial measures, and personal descriptive information (*i.e.*, income, employment, occupation, marital history, and other demographic items). A portion of the interview assessed risk behaviors over the past 90 days using timeline follow-back (TLFB) interview procedures. The TLFB interview method is a widely used assessment procedure that uses calendar-based guided recall to accurately measure alcohol and illicit drug use [47] or HIV risk behavior [48]. TLFB procedures have been shown to be psychometrically valid when completed either in person or over the telephone [49]. As a first step in this procedure, a trained interviewer reviewed a 90-day calendar with the participant, marking on the calendar holidays and significant events (birthdays, anniversaries, illnesses of oneself or close friends and family, or any event that was deemed personally meaningful to the participant) to create "anchor points" for facilitating accurate recall. The interviewer then guided the participant to elicit their day-to-day risk behaviors working backwards over a 90-day period. The length of the telephone interview depended on the degree of engagement in risky behavior and ranged from 20 minutes for respondents engaging in little risk behavior to over 60 minutes for individuals engaging in frequent risk behavior. The research protocol and consent procedures were reviewed and approved by the Institutional Review Board at Cornell Medical College.

2.3. Measures

Young Adult Latent Class Indicators of Risk Behavior. All of the indicators for the Latent Class Analysis (LCA) models were categorized *a priori* to indicate low and high risk. The alcohol use items included frequency of drinking beer, wine, or hard liquor (a few times a month *vs.* once a week), frequency of drinking until you get drunk (not getting drunk *vs.* a few times per year), quantity of

drinks per drinking occasion (three or less *vs.* four or more), and number of days having five or more drinks (no days *vs.* one or more days). The same procedure was used for the high risk sexual behavior items, with four items including number of sexual partners in the past three months (one or less *vs.* two or more, respectively), a quantified index of partners (one or less sets of initials used by respondent to indicate sexual activity *vs.* more two or more sets of initials), whether or not the participant had a new partner within the past three months, and number of sex event with individuals not designated as main partner (one *vs.* two or more sexual events).

Adolescent Psychosocial Markers. The following measures were included at the 9th grade assessment and were used to predict membership in the latent classes at the young adult follow-up. The variables reflect individual-level characteristics that may instigate high risk drinking and sex (propensity for risk taking) and social and interpersonal factors that either facilitate (social confidence and self-esteem) or inhibit engagement in these behaviors (self-control).

Risk-Taking. Four items ($\alpha = 0.80$) were taken from the Eysenck Personality Inventory [50] to assess impulsive and daring behavior. Sample items include "I would do almost anything on a dare" and "I enjoy taking risks." Students indicated responses on a 5-point scale ranging from (1) *really not true for me* to (5) *really true for me*.

Social-Confidence. A 14-item scale assessed students' confidence about their ability to use specific social skills ($\alpha = 0.82$). Participants were provided a stem "How confident are you that you could do well in the following situations..." with sample items including "ending a conversation with friends without offending them," "starting a conversation with someone you've just met," and "asking questions to avoid a misunderstanding." Response categories ranged from (1) *not at all confident* to (5) *very confident*.

Self-Esteem. Five items from the [51] Self-Esteem Scale were used to assess the positive evaluative component of self-esteem ($\alpha = 0.85$). Sample items include "I feel that I have a number of good qualities," and "I have a positive attitude about myself." Response categories ranged from (1) *strongly disagree* to (5) *strongly agree*.

Self-Control. Fourteen items ($\alpha = 0.72$) from the Kendall and Wilcox Self-Control Rating Scale [52] were used to measure self-control skills. The SCRS assesses cognitive-behavioral elements of self-regulation including the ability to manage impulsive, distracting, or disruptive behavior, particularly those occurring in school settings. Sample items include "I stick to what I am doing until I'm finished with it," "I have to be reminded several times to do something," and "I am easily distracted from my work." Response categories ranged from (1) *strongly disagree* to (5) *strongly agree*.

2.4. Data Analysis Plan

Latent class analysis (LCA) represents a broad class of random coefficient analytic methods that can be used to summarize meaningful subgroups of individuals based on their patterns of response profiles. In recent years, investigators using LCA have been successful in defining unique classes of individuals based on nicotine use [53], patterns of delinquency [54], problem behaviors [55], and different types of drug use [56-58]. This approach has become an important alternative to traditional variable-centered methods that can produce summaries of how one variable relates to another (*i.e.*, traditional factor analysis methods) but provides little information as to how individuals cluster with

regard to their response profiles. In the simplest case with two questions each having yes/no answers, a 2 × 2 cross-tabulation produces four possible response profiles (including yes-yes, yes-no, no-no, and no-yes). When the possible contingency table is this straightforward the naked eye can scan the possible response profiles and an investigator can apply the chi-square statistic to assess the degree of dependence among measures. However, when the sheer number of possible profiles expands considerably (*i.e.*, with 8 dichotomous items there are 2^8 or 256 possible response patterns), more refined statistical methods that can consider probabilistically the joint probabilities of a multiway table are needed to identify meaningful response patterns [59].

LCA is traditionally employed with categorical indicators (*i.e.*, yes/no) that are used to define membership in discrete groups. These indicators are considered imperfect, but informative with regard to a latent (unobserved) categorical variable that contains discrete and mutually exclusive subpopulations or classes. The members of a particular class differ from each other only with respect to random measurement error but are uniquely and systematically different from members in other classes based on their response profiles [60]. Important model parameters include the latent class prevalence or proportion of individuals assigned to a particular class (*i.e.*, estimated posterior probabilities) and the item response probabilities, which are measurement parameters interpreted as the likelihood of endorsing a particular item if you are a member of a particular class. As we explain below, statistical fit can be evaluated on the basis of how well a particular model positing a latent categorical variable with *k* classes can reproduce the observed response patterns (observed *vs.* expected cell frequencies) and correctly classify individuals (*i.e.*, the model produces consistent and efficient estimators), the parsimony achieved through a reduced number of classes, whether an obtained latent class model is theoretically interesting, and the overall stability of the obtained classes.

3. Results

3.1. Attrition Analyses

There was 56% sample retention from the 9th grade to the young adult assessment. Mean comparisons showed that participants retained through young adulthood did not differ significantly from dropouts on their alcohol consumption (measured by a mean composite of drinking frequency, intensity, and drunkenness). A logistic regression model indicated that several demographic measures predicted attrition in the study, including being older in age (b = 0.381 [SE = 0.179], being black (b = 0.433 [SE = 0.168], and coming from a single parent home (b = 0.589 [SE = 0.168], all $p < 0.05$. This suggests that those who dropped out of the study were at higher risk than those who remained in the study.

3.2. Prevalence of Young Adult High-Risk Behaviors

Prevalence estimates for the latent variable indicators showed that 13% of the young adult sample reported drinking at least once a week, 31% reported drinking until they got drunk, 17% said they were having at least four or more drinks each time they drank, and 26% reported heavy drinking (one or more days where they drank five or more drinks per occasion). Regarding the sexual risk items, 8% of the panel sample respondents said they had two or more sexual partners in the past three months, an

additional 8% provided two or more sets of initials for sexual partners spanning the same period, 18% said the individual they had sexual relations within the past three months was a "new" partner, and of those who had multiple partners 28% reported multiple sex events with someone other than their main partner. We used the MCMC procedure for arbitrary missing data under the assumption of missing at random. This procedure relied on single imputation for these analyses (there was no gain in efficiency for estimating the standard errors with more than a single imputation).

3.3. LCA Results

Table 1 shows the fit statistics corresponding to the sequence of models tested. Models were tested from the most parsimonious one class model (all participants responded in the same way) to a solution with ten classes. Evaluative model fit statistics include the Bayesian Information Criteria [61] and the Akaike's Information Criteria [62,63], both of which penalize the log-likelihood for increased number of parameters as models increase in complexity (*i.e.*, increasing the number of classes). The log likelihood ratio test statistic (L^2) shows the amount of variation left in the model among the variables after extracting the classes (in all cases smaller numbers indicate a better fit). The likelihood ratio chi-square test statistic (G^2), which assesses "departure" of the population or expected model from the observed sample data, should be evaluated with respect to the degrees of freedom in the model (an approximate value of 1.0 indicates better fit). A well fitting model will have G^2 distributed as a chi-square statistic with the degrees of freedom equivalent to the number of possible response patterns (in the current model this is 2^8) less the number of estimates (indicator variables) minus one [64]. The column labeled L^2/df provides an approximate F-statistic [65].

Table 1. Fit statistics from the latent class analyses.

Number of classes	Log-likelihood (L^2)	BIC-L^2	AIC-L^2	Npar/DF	G^2	L^2/df	*p*-value [a]	%ER[b]
1-class	2548.2	5148.7	5112.4	8/238	880.3	10.71	0.0000	0.000
2-class	2216.3	4543.7	4466.6	17/238	472.1	9.31	0.0000	0.087
3-class	2060.1	4290.3	4172.3	26/229	159.8	8.99	0.9829	0.081
4-class	2038.6	4306.0	4147.1	35/220	116.7	9.26	1.0	0.800
5-class	2022.1	4332.0	4132.2	44/211	83.8	9.58	1.0	0.794
6-class	2014.8	4376.1	4135.5	53/202	69.1	9.97	1.0	0.790
7-class	2009.2	4423.9	4142.4	62/193	57.9	10.41	1.0	0.790
8-class	2005.1	4474.4	4152.1	71/184	49.6	10.89	1.0	0.787
9-class	2001.3	4525.8	4162.7	80/175	42.2	11.44	1.0	0.785
10-class	1997.4	4576.9	4172.8	89/166	34.4	12.03	1.0	0.784

Note: [a] Significance values can be computed using the Lo-Mendell-Rubin likelihood-ratio test (Lo *et al.*, 2001) allowing for direct tests between models with '*k*' and '*k-1*' classes. Low p-values indicate the model with one less class should be rejected in favor of the estimated model. [b] %ER = percent error reduction in L^2 when model is pitted against the null model of complete independence (one-class model).

Int. J. Environ. Res. Public Health **2012**, *9*

An inspection of Table 1 shows that the AIC and BIC fit indices become progressively smaller through the three and four class models and the error terms in these models was substantially less than models containing more classes. Notably, extraction of the fourth class (low risk drinking, high risk sex) required migration of participants from other classes and the resulting class was quite sparse (N = 33, or 4% of the total sample was located within this class). The low cell counts associated with the four class solution might increase the likelihood of misclassification and strain the robustness of the subsequent estimation procedures. This is consistent with the notion that, in the process of identifying a satisfactory latent class model, one can reach a saturation point that produces weak identifiability [66] beyond which there are too many classes, too few reliable class indicators, and too few people allocated to the classes [67]. With these considerations in mind, the three-class model provided the best fit based on shrinkage in the AIC and BIC, the G^2, the percent error reduction (an alternative fit to entropy-based measures) and the overall ratio of L^2/df.

Table 2. Comparison of parameters across the unconstrained and constrained models.

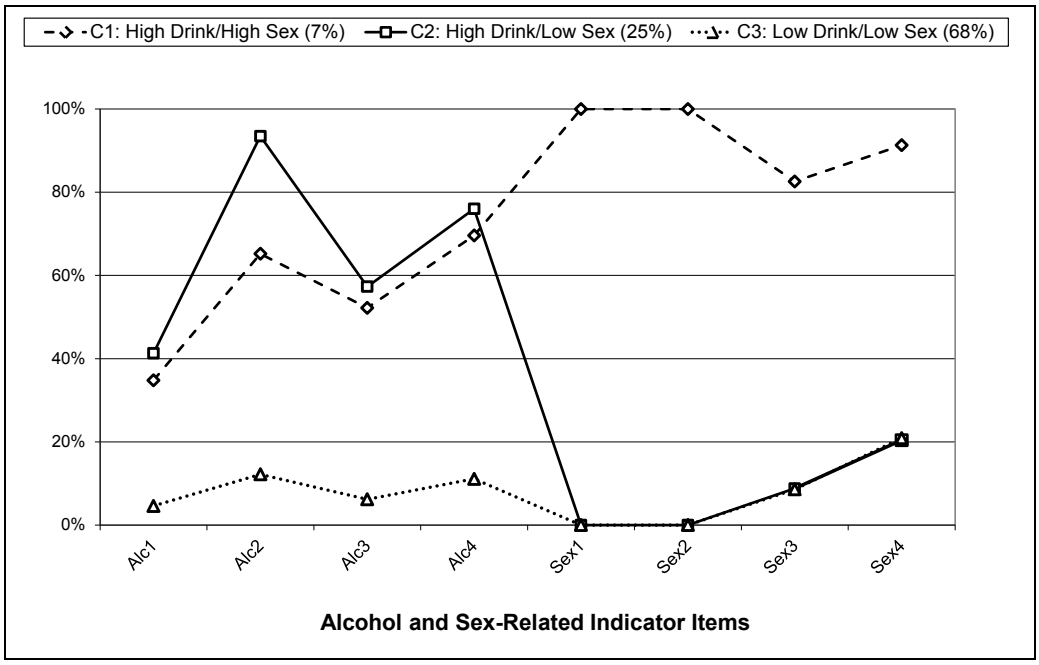
	Model 1: Unconstrained			**Model 2: Constrained**		
	Class 1	**Class 2**	**Class 3**	**Class 1**	**Class 2**	**Class 3**
Black Participants						
Proportion	0.033	0.113	0.304	0.033	0.118	0.299
Count (N = 312)	23	78	211	23	82	207
	7%	25%	68%	7%	26%	66%
Item Probabilities (for higher risk responding)						
Drinking Frequency (Alc1)	0.348	0.413	0.046	0.263	0.409	0.046
Drunkenness Frequency (Alc2)	0.652	0.935	0.122	0.526	0.899	0.134
Drinking Quantity (per occasion) (Alc3)	0.522	0.573	0.062	0.316	0.571	0.051
Binge Days in Past 3 Months (Alc4)	0.696	0.760	0.111	0.544	0.766	0.102
# Sexual Partners (Sex1)	1.000	0.000	0.000	1.000	0.000	0.000
Recent Sexual Partners (Sex2)	1.000	0.000	0.000	1.000	0.000	0.000
New Sexual Partners (Sex3)	0.826	0.088	0.086	0.772	0.141	0.120
Sex Events w/ Non-Main Partner (Sex4)	0.913	0.204	0.209	0.912	0.271	0.215
	Class 1	**Class 2**	**Class 3**	**Class 1**	**Class 2**	**Class 3**
Other Participants						
Proportion	0.049	0.092	0.407	0.049	0.075	0.425
Count (N = 380)	34	64	282	34	52	294
	9%	17%	74%	9%	14%	77%
Item Probabilities (for higher risk responding)						
Drinking Frequency (Alc1)	0.206	0.371	0.035	0.263	0.409	0.046
Drunkenness Frequency (Alc2)	0.441	0.734	0.133	0.526	0.899	0.134
Drinking Quantity (per occasion) (Alc3)	0.176	0.475	0.038	0.316	0.571	0.051
Binge Days in Past 3 Months (Alc4)	0.441	0.719	0.072	0.544	0.766	0.102
# Sexual Partners (Sex1)	1.000	0.000	0.000	1.000	0.000	0.000
Recent Sexual Partners (Sex2)	1.000	0.000	0.000	1.000	0.000	0.000
New Sexual Partners (Sex3)	0.735	0.281	0.125	0.772	0.141	0.120
Sex Events w/ Non-Main Partner (Sex4)	0.912	0.438	0.195	0.912	0.271	0.215

Notes: Proportions represent classification of individuals based on their most likely latent class pattern; Count represents latent class count based upon most likely class.

Int. J. Environ. Res. Public Health **2012**, *9*

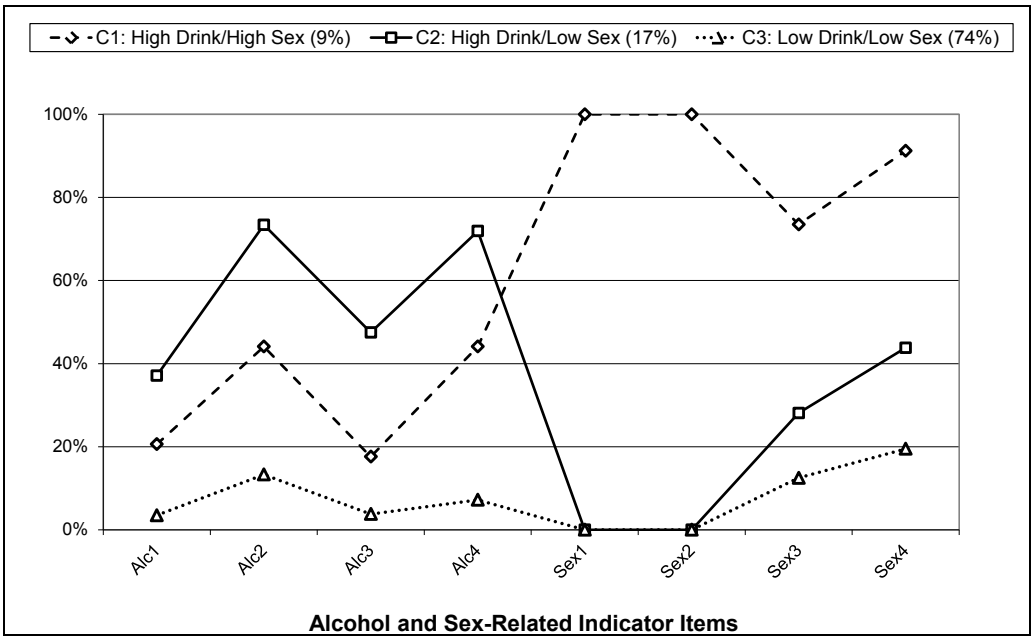
Item response (or endorsement) probabilities show that Class 1 (8% of participants) contains women who reported high risk drinking and high risk sexual behaviors (referred to as "high-high"). The second class (19%) contains women who reported high risk drinking but low risk sex (referred to as "high-low"). The third and largest class (73%) contains women who reported low risk drinking and low risk sex (referred to as "low-low"). After deriving the three-class model we estimated a mixture model with race/ethnicity as a grouping variable (black *vs.* other). This model addresses whether race/ethnicity contributes to any of the heterogeneity underlying the classes as well as defining class membership. Table 2 shows the class membership probabilities and item response probabilities for the three class model tabulated by race/ethnicity. Model parameters are presented for both constrained and unconstrained models; the former constrained the item response probabilities across the two race/ethnicity groups. The conditional likelihood ratio difference test and the non-significant p-value indicated the model with these constraints was tenable, $\Delta G^2(24) = 34.32$, $p > 0.10$ (*i.e.*, there was no degradation in fit by implying the item thresholds and class probabilities are equal across groups). Class assignment probabilities from the unconstrained model indicated that 7% of black women were assigned to the high-high class, 25% were assigned to the high-low class, and 68% were assigned to the low-low class (based on N = 312). The same numbers for the non-black women (based on N = 380) indicated that 9% were assigned to the high-high class 1, 17% were assigned to the high-low class, and 74% were assigned to the low-low class.

Figure 1. (**a**) Item response probabilities by class membership among black women; (**b**) Item response probabilities by class membership among other women (reference category).



(**a**)

Figure 1. *Cont.*



(**b**)

Figures 1(a,b) illustrate the item response probabilities for each alcohol and sex-related indicator for black women and for women of other race/ethnicities. Members of the low-low class did not exceed the critical 0.5 threshold for any single item (this pattern held for black and non-black women). Compared to women from other race/ethnic groups, black women in the low-low class were more likely to report drinking excessively for more than five days in a row and less likely to report new sexual partners in the same time frame. Black women in the high-low class were characterized by high likelihood of drinking to get drunk, episodes of drunkenness, and number of days having five or more drinks but low levels of all high risk sexual behaviors. Black women in the high-high class were characterized by high scores for all four sexual behavior items and drinking until drunk and binge drinking. The same pattern of endorsement was not evident for the non-black women in the high-high class. These individuals were much less likely to report high risk drinking and generally equally likely to report high risk sexual behaviors.

3.4. Results of the MNR Model

After extraction of the classes grouped by race/ethnicity, the next step included examining several possible determinants of class membership using multinomial logistic regression. The reference class for these comparisons was Class 3 (low risk drinking/low risk sex). The MNR model held constant some of the important 9th grade socio-demographic factors that can contribute to class differences including nuclear family status (intact *vs.* other), free lunch (receipt *vs.* no subsidy), and a continuous measures of self-reported grades (a proxy for conventional behavior and school bonding). Other measures of psychosocial functioning modeled included risk-taking, social confidence, self-esteem, and self-control. Table 3 presents the findings from the MNR including regression coefficients and relative odds ratios for class membership based on these external markers. Findings indicated that

self-control played an instrumental role for members of Class 1 (high-high). Compared to members of Class 3 (low-low), the logs odds of their being assigned to the high-high class decreased by −0.571 for a unit increase in self-control (*i.e.*, indicating a strong protective role for self-control). Nuclear family status also helped to distinguish members of Class 1 (high-high) from the reference Class 3 (low-low). Residing with both biological parents was also protective as the log odds of being assigned to Class 1 (high-high) decreased −0.611 for a unit increase in family status (*i.e.*, living in an intact family).

Table 3. Relative odds of latent class membership.

	Beta		***p*-value**		**Odds Relative to Class 3**	
	Class 1	**Class 2**	**Class 1**	**Class 2**	**Class 1**	**Class 2**
Black (Reference = Other)	0.171	0.824	0.540	0.002	1.186	2.280
Psychosocial Markers (9th Grade)						
Risk-Taking	−0.058	0.180	0.712	0.154	0.944	1.197
Social Confidence	0.247	0.108	0.091	0.407	1.280	1.114
Self-Esteem	0.390	−0.197	0.108	0.210	1.477	0.821
Self-Control	−0.571	−0.275	0.002	0.101	0.565	0.760
Covariates (9th Grade)						
Grades In School	0.215	−0.034	0.128	0.818	1.240	0.967
Free School Lunch	0.102	−0.426	0.721	0.094	1.107	0.653
Nuclear Family	−0.611	0.081	0.038	0.731	0.543	1.084

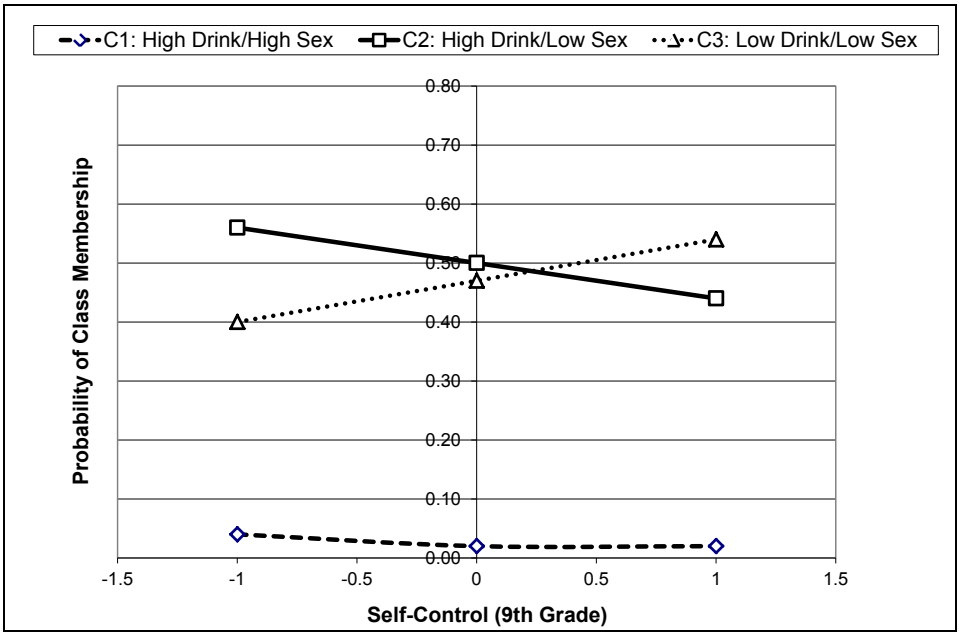
Notes: Class 1 (High Risk Drinking, High Risk Sex); Class 2 (High Risk Drinking, Low Risk Sex); Class 3 (Low Risk Drinking, Low Risk Sex).

Figures 2(a,b) graphically portray the association between 9th grade self-control and class assignment for the different race groups. Figure 2(a) shows that for black women self-control plays a minimal role in the risk of assignment to Class 1 (high-high); the line is relatively flat and unchanging for unit increases in self-control. However, the probability of assignment to Class 3 (low-low) increases substantially with corresponding increases in self-control (*i.e.*, protective effect). The gross differences in these two slopes is what contributes to the significant effect shown in Table 3 with the corresponding negative beta weight (b = −0.571) for Class 1 versus Class 3. Although the difference between Class 1 and Class 2 was not significant, the probability of assignment to Class 2, designated the high-low risk group (characterized by excessive drinking practices), decreases with corresponding increases in self-control (these individuals can potentially migrate to the other two classes).

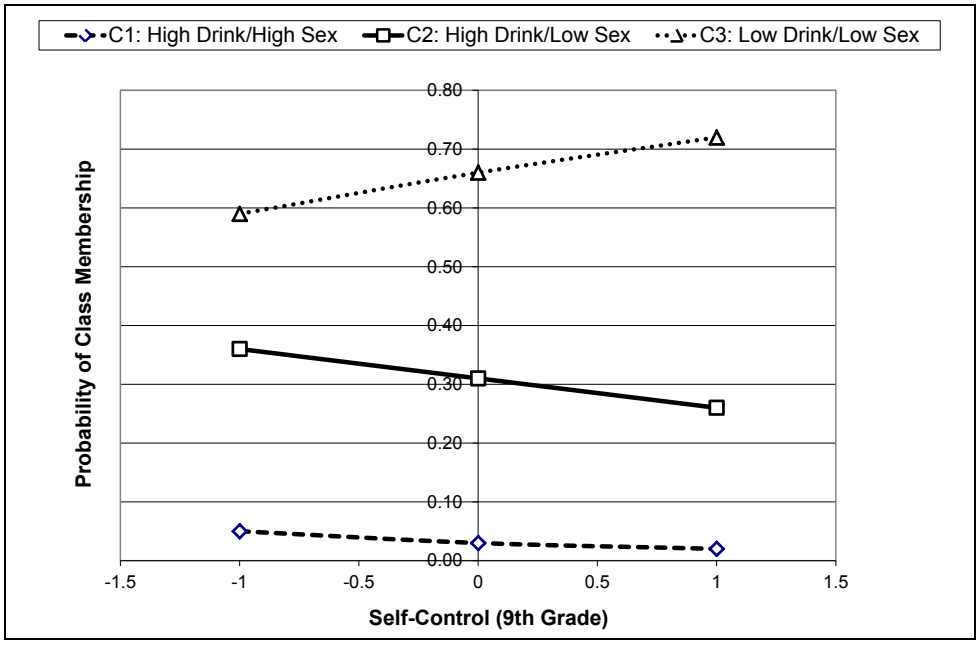
Figure 2(b) shows the same plots for the non-black women in the sample. Here, the effects also shows that self-control is protective for members of Class 3 (low-low) in ways that do not appear to apply for members of Class 2 (high-low) in that self-control decreases; and Class 3 (low-low) with a relatively flat line. There are also some notable differences between the plots. Although the effect of self-control is fairly consistent for women of both black and non-black race groups assigned to the high-high risk group (relatively flat), the slope of the line for the low-low risk group is much steeper for the non-black race group showing that self-control is more protective (increasing levels of self-control increases the odds of being assigned to this group). Also notable is that probabilities for

class assignment for Class 2 (high-low) are somewhat higher in the black compared to non-black women, although the line is negatively sloped in both groups.

Figure 2. (**a**) Probability of class membership in young adulthood as a function of 9th grade self-control among black women; (**b**) Probability of class membership in young adulthood as a function of 9th grade self-control among other women (reference category).



(**a**)



(**b**)

Int. J. Environ. Res. Public Health **2012**, *9*

4. Discussion

The present study examined the extent to which high risk drinking and sexual behavior clustered together in a sample of urban minority young adult women, a population that has been shown to be at elevated risk for HIV, STIs, and other negative outcomes related to sexual health. We also examined whether several relevant aspects of psychosocial functioning measured at the beginning of high school predicted classes of risk behavior years later when participants were assessed as young women. Young adulthood provides a unique window to examine high risk drinking and sexual behavior. These behaviors peak during the early to mid-twenties, as young people live more independently and autonomously from their family of origin, enjoy new freedoms such as legal drinking and the ability to enter bars and nightclubs, and have increased opportunities for sexual and romantic relationships. Indeed, *emerging adulthood* provides young adults with the opportunity to pursue novel, intense, and risky experiences with greater freedom compared to any other developmental period [68].

Because alcohol use and sexual behavior share a number of similar social and personal functions, one might expect widespread comorbidity for engagement in high risk levels of these behaviors in young adulthood. Drinking and sexual activity are both appetitive behaviors that young people may pursue for the purposes of recreation, relaxation, social connection, excitement, and/or pleasure. Furthermore, both behaviors may be influenced similarly by individual-level variables that promote engagement in behavior (e.g., propensity for risk taking), social and interpersonal factors that facilitate behavior (e.g., social confidence and self-esteem), and characteristics that inhibit engagement (e.g., self-control) [20,21]. However, findings from the present study showed that overlap between high risk drinking and sexual behavior in the same individuals was relatively infrequent. At the young adult assessment point, there were three latent classes or homogenous groups of women based on their patterns of high risk alcohol use and sexual behaviors. The largest class (73%) represented women characterized by low risk alcohol use and low risk sexual behaviors ("low-low"). The next largest class of women (19%) was characterized by high risk alcohol use and low risk sexual behaviors ("high-low"). The smallest class (8%) was characterized by high risk alcohol and high risk sexual behaviors ("high-high"). Thus, in the final three class model, we found a low proportion of individuals in the "high-high" class, and over twice as many women in the "high-low" class (high risk drinking/low risk sex). In choosing the three over the four class solution, we noted the relatively small proportion of individuals (4%) assigned to a low risk drinking and high risk sexual behavior class; that migration from other classes likely created this small class of individuals; and that using a four class solution would likely create estimation problems given the sparse cells.

Although there was less overlap in risk behaviors than expected, we found that both risky drinking and risky sexual behaviors were predicted in a similar manner by early adolescent psychosocial functioning. In particular, self-control measured at 14 years of age—but not risk-taking, social confidence, or self-esteem—reduced the likelihood of being in the high-high class eight years later when the girls were young adults. This demonstrates a broad and enduring protective effect of self-control on multiple high risk behaviors over a lengthy period of developmental change. These findings provide evidence that the benefits of self-control persist into young adulthood and are not limited to children and adolescents, who have been the focus of most research in this area [25,36,69,70].

4.1. Implications for Self-Control and Addictive Behaviors

Although the focus of this paper was on high risk drinking and sex, our findings may have implications for compulsive or addictive patterns of alcohol abuse and sexual behavior. In particular, they may be informative regarding addiction specificity, or the extent which an individual engages in one category of addictive behaviors but not another. Sussman *et al.* [71] propose a model of addiction specificity that outlines how a number of biological, environmental, situational, and learning factors may explain why some addictive behaviors occur in an individual at the exclusion of other seemingly similar behaviors. Our findings of relatively low levels of overlap in patterns of risky drinking and risky sex are consistent with the idea of addiction specificity: the two behaviors were found to co-occur in a relatively small proportion of the sample even though individuals may engage in both of these behaviors for similar purposes.

Self-control is central to the concept of addiction. Someone engaging in an addictive behavior does so compulsively and experiences a subjective inability to control consumption or stop behavior when it has clear negative consequences. Indeed, there are a number of similarities between conceptual models of self-control [21] and the model of addiction specificity [71]. Both describe multifactorial etiologic mechanisms resulting from a complex interplay of biopsychosocial influences that may include factors such as genetic and neurobiological systems, as well as exposure to unique environments, experiences, and learning opportunities, that can begin to influence behavior or precursors of behavior in childhood and adolescence. Also for both self-control and addiction specificity, their ultimate expression is shaped by a variety of socialization factors and environmental influences that provide opportunities for engagement and social reinforcement. Thus, the development of high risk and compulsive behaviors, as well as the ability to control these behaviors, is a function of a variety of contingencies that lead to different trajectories of behavior. These may channel specific individuals towards either high risk drinking or risky sexual behavior. The low levels of overlap between these behaviors in our study may reflect the large number of variables that can contribute to individual trajectories, resulting in differential expression of high risk drinking and sexual behaviors.

Theory and research suggest that self-control can have direct and indirect effects on risk behavior. For example, Wills and Dishion [72] describe how self-control stems from early temperamental factors including attentional focusing, a characteristic that contribute to adaptive problem-solving and enhance self-control abilities in ways that provide direct protective effects. Alternatively, self-control can also be protective through indirect or buffering effects. It may, for example, buffer the impact of negative peer influences such that teens with good self-control would be less influenced by substance using peers. Empirical evidence also shows that self-control and similar variables related to self-regulation can have direct and indirect protective effects on risky drinking and risky sex. Quinn and Fromme [20] found that self-regulation served as a protective factor against risky drinking and sexual behavior in a sample of college students over the age of 21; they found that self-regulation was particularly important for buffering the impact of heavy episodic drinking on unprotected sex among participants high in risk-taking. Although we did not test for buffering effects of self-control in our analyses, we did find a durable and direct protective of effect of self-control on risky drinking and sex over a lengthy time period of developmental change. Further, the protective effect of self-control remained

significant when other key variables (risk taking, social confidence, and self-esteem) that may be correlated with self-control and/or the outcomes were controlled for in the model.

To the extent that self-control has enduring protective effects across multiple risk behaviors during the transition to young adulthood, implementing effective interventions to increase self-control would serve to promote healthy behavior and enhance public health. Dual process conceptual models suggest that impulsivity and self-control work together to predict engagement in high risk behavior. People balance their impulses and urges for immediate gratification with behavioral self-restraint in order to achieve longer term goals or to act in accordance with personal standards, attitudes, or expectancies [73]. Experimental studies suggest that interventions may focus on factors associated with impulsive tendencies, such as efforts to modify automatic maladaptive associations linking an unhealthy behavior to positive affect, or training individuals to direct attention away from unwanted temptation [74]. However, more research has focused on interventions to increase self-control abilities. In this context, self-control is often conceptualized as a limited resource that can be depleted with use—much like a muscle that becomes tired after exertion. To further the muscle analogy, it appears that self-control can be strengthened by regular "exercise." Several studies have shown that people can enhance their self-control abilities such that their "strength" is less quickly depleted when responding to demands [75].

4.2. Strengths and Limitations

Strengths of the study included the longitudinal panel data collected over a period from adolescence to young adulthood in a sample of ethnic minority young women from economically disadvantaged urban communities. The study utilized a unique person-centered data analysis approach relying on latent class analysis to examine subgroup heterogeneity that might otherwise have escaped detection using conventional data analytic methods. This method provides an alternative way to summarize behavior by sifting through categorical response profiles and capturing similarities in the way participants answer questions about their behavior. Other data analysis approaches may have missed the subtle nature of the underlying classes and as a result also missed the protective effects self-control exerts on high risk behaviors.

Several limitations should also be noted. The study utilized a restricted range of sexual behavior measures which focused largely on the number of partners and frequency of sexual activity. These are only a subset of the spectrum of variables that relate to high risk sexual behavior and have relevance for sexual addiction. This may explain in part the lack of a low drink/high sex group from the LCA analysis. There was substantial participant attrition between the 9th grade and the young adult follow-up, thus findings may have limited generalizability to the most at-risk members of this population. The study took a cross-sectional "snapshot" of the lives of these individuals at one point in time as they entered young adulthood. Of course, many events transpired between adolescence and the follow-up interview conducted in young adulthood that may have influenced engagement in high risk drinking and sex. Nevertheless, the fact that self-control was associated with both behaviors almost eight years later highlights its durable role in predicting long-term behavioral outcomes. It is also likely that a variety of unmeasured variables (e.g., externalizing behaviors, substance abuse diagnosis, social modeling effects, *etc.*) explain whether or not sex and alcohol co-occur among young women, lead to

compulsive or addictive behavior, and whether or not one behavior may serve as a substitute addiction for the other for some women but not others. There are also some limitations of LCA as an analytic approach. LCA is a statistical technique that relies on mathematical formulations to achieve class assignment. Characterization of the derived classes is "model dependent" and assignment of individuals to their respective classes hinges on the measures used, the sample size, covariates, and interpretational decisions made by the investigator [76]. Thus the derived classes are not literal entities but rather statistical abstractions that contain an element of subjectivity that is imposed on the data [77]. However, LCA methods do help to reduce complexity in behaviors and in the present study have produced important information about the interplay of high-risk drinking and sexual behaviors.

4.3. Directions for Future Research

Future studies may expand on the present findings by examining how specific events in the years during the transition to young adulthood influence engagement in risky drinking and sex, as well as the events, experiences, and opportunities that shape the development of self-control and addictive behaviors. These efforts will be important for understanding the nature of these phenomena and for the purposes of developing effect prevention and treatment efforts. This is particularly important because the transition to young adulthood has changed considerably compared to previous generations. The years between adolescence and adulthood are now extended in time with young adults transitioning in and out of school, relationships, jobs, and living arrangements. There are more opportunities for young people to take on different roles and explore various career and lifestyle options and opportunities for self-expression. Adult responsibilities such as marriage, parenting, and career—which have been associated with decreases in risk behavior—occur later now. The extended transition into young adulthood, which now often lasts through the late twenties or beyond, lengthens the period of development when substance use, sexual exploration, and a variety of potentially high risk behaviors have historically peaked in prevalence. It also may provide the time necessary for individuals to develop an extended history with a potentially compulsive behavior, increasing the likelihood of addictive patterns of behavior that interfere with successful development. For these reasons, increasing knowledge about the etiology of risk behaviors during this time of life is increasingly important for effective prevention efforts.

Acknowledgements

This paper was supported by grants from the National Institute on Drug Abuse (R01 DA023890) and National Institute on Alcoholism and Alcohol Abuse (R21 AA016531) to the first author.

References

1. Substance Abuse and Mental Health Services Administration (SAMHSA). *Results from the 2010 National Survey on Drug Use and Health: Summary of National Findings*; NSDUH Series H-41; US Department of Health and Human Services: Washington, DC, USA, 2011; HHS Publication No. (SMA) 11-4658.

2. Hingson, R.; Heeren, T.; Winter, M.; Wechsler, H. Magnitude of alcohol-related mortality and morbidity among U.S. college students ages 18–24: Changes from 1998 to 2001. *Annu. Rev. Publ. Health* **2005**, *26*, 259-279.
3. Perkins, H.W. Surveying the damage: A review of research on consequences of alcohol misuse in college populations. *J. Stud. Alcohol* **2002**, *S14*, 91-100.
4. O'Neill, S.E.; Parra, G.R.; Sher, K.J. Clinical relevance of heavy drinking during the college years: Cross-sectional and prospective perspectives. *Psychol. Addict. Behav.* **2001**, *15*, 350-359.
5. Yi, H.; Williams, G.D.; Smothers, B.A. *Trends in Alcohol-Related Fatal Traffic Crashes: United States, 1977–2002*; Surveillance Report No. 69; National Institute on Alcohol Abuse and Alcoholism: Bethesda, MD, USA, 2004.
6. Weinstock, H.; Berman, S.; Cates, W. Sexually transmitted diseases among American youth: Incidence and prevalence estimates, 2000. *Perspect. Sex. Repro. H* **2000**, *36*, 6-10.
7. Futterman, D.C. HIV in adolescents and young adults: Half of all new infections in the United States. *Top. HIV Med.* **2005**, *13*, 101-105.
8. Naimi, T.S.; Lipscomb, L.E.; Brewer, R.D.; Gilbert, B.C. Binge drinking in the preconception period and the risk of unintended pregnancy: Implications for women and their children. *Pediatrics* **2003**, *111*, 1136-1141.
9. Cook, R.L.; Clark, D.B. Is there an association between alcohol consumption and sexually transmitted diseases? A systematic review. *Sex Transm Dis*. **2005**, *32*, 156-164.
10. Cooper, M.L. Alcohol use and risky sexual behavior among college students and youth: Evaluating the evidence. *J. Stud. Alcohol* **2002**, *S14*, 101-117.
11. Leigh, B.C.; Stall, R. Substance use and risky sexual behavior for exposure to HIV: Issues in methodology. *Am. Psychol.* **1993**, *48*, 1035-1045.
12. Hingson, R.W.; Strunin, L.; Berlin, B.M.; Heeren, T. Beliefs about AIDS, use of alcohol and drugs, and unprotected sex among Massachusetts adolescents. *Am. J. Public Health* **1990**, *80*, 295-299.
13. Leigh, B.C. The relationship of sex-related alcohol expectancies to alcohol consumption and sexual behavior. *Br. J. Addict.* **1990**, *85*, 919-928.
14. Brown, J.L.; Vanable, P.A. Alcohol use, partner type, and risky sexual behavior among college students: Findings from an event-level study. *Addict. Behav.* **2007**, *12*, 2940-2952.
15. Cooper, M.L.; Peirce, R.S.; Huselid, R.F. Substance use and sexual risk taking among Black and White adolescents. *Health Psychol.* **1994**, *13*, 251-262.
16. Dermen, K.H.; Cooper, M.L. Sex-related alcohol expectancies among adolescents. *Psychol. Addict. Behav.* **1994**, *8*, 161-168.
17. Martens, M.P.; Page, J.C.; Mowry, E.S.; Damann, K.M.; Taylor, K.K.; Cimini, M.D. Differences between actual and perceived student norms: An examination of alcohol use, drug use, and sexual behavior. *J. Am. Coll. Health* **2006**, *54*, 295-300.
18. Patrick, M.E.; Maggs, J.L. Profiles of motivations for alcohol use and sexual behavior among first-year university students. *J. Adolesc.* **2010**, *33*, 755-765.
19. Steele, C.M.; Josephs, R.A. Alcohol myopia: Its prized and dangerous effects. *Am. Psychol.* **1990**, *45*, 921-933.

20. Quinn, P.D.; Fromme, K. Self-regulation as a protective factor against risky drinking and sexual behavior. *Psychol. Addict. Behav.* **2010**, *24*, 376-385.
21. Wills, T.A.; Ainette, M.G. Temperament, self-control, and adolescent substance use: a two-factor model of etiological processes. In *Handbook of Drug Use Etiology: Theory, Methods, and Empirical Findings*; Scheier, L.M., Ed.; American Psychological Association: Washington, DC, USA, 2010; pp. 127-146.
22. Baumeister, R.F.; Vohs, K.D.; Tice, D.M. The strength model of self-control. *Curr. Dir. Psychol. Sci.* **2007**, *16*, 396-403.
23. Gross, J. Emotion regulation: Past, present, future. *Cogn. Emot.* **1999**, *13*, 551-573.
24. Posner, M.I.; Rothbart, M.K. Developing mechanisms of self-regulation. *Dev. Psychopathol.* **2000**, *12*, 427-441.
25. Sussman, S.; McCuller, W.J.; Dent, C.W. The associations of social self-control, personality disorders, and demographics with drug use among high-risk youth. *Addict. Behav.* **2003**, *28*, 1159-1166.
26. Tangney, J.P.; Baumeister, R.F.; Boone, A.L. High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *J. Persons.* **2004**, *72*, 271-324.
27. Otten, R.; Barker, E.D.; Maughan, B.; Arseneault, L.; Engels, R.C. Self-control and its relation to joint developmental trajectories of cannabis use and depressive mood symptoms. *Drug Alcohol Depend*. **2010**, *112*, 201–208.
28. Baumeister, R.F.; Heatherton, T.F.; Tice, D.M. *Losing Control: How and Why People Fail at Self-Regulation*; Academic Press: San Diego, CA, USA, 1994.
29. Calkins, S.D. Origins and outcomes of individual differences in emotional regulation. In *Emotion Regulation: Behavioral and Biological Considerations, Monographs of the Society for Research in Child Development*; Fox, N.A., Ed.; University of Chicago Press: Chicago, IL, USA, 1994; Volume 59, Issue 2–3, Series 240.
30. Fox, N.A.; Calkins, S.D.; Bell, M.A. Neural plasticity and development in the first two years of life: Evidence from cognitive and socioemotional domains of research. *Dev. Psychopathol.* **1994**, *6*, 677-696.
31. Vazsonyi, A.T.; Huang, L. Where self-control comes from: On the development of self-control and its relationship to deviance over time. *Dev. Psychol.* **2010**, *46*, 245-257.
32. Wills, T.A.; Ainette, M.G.; Stoolmiller, M.; Gibbons, F.X.; Shinar, O. Good self-control as a buffering agent for adolescent substance use: An investigation in early adolescence with time-varying covariates. *Psychol. Addict. Behav.* **2008**, *22*, 459-471.
33. Engels, R.C.; Vermulst, A.A.; Dubas, J.S.; Bot, S.M.; Gerris, J. Long-term effects of family functioning and child characteristics on problem drinking in young adulthood. *Eur. Addict. Res.* **2005**, *11*, 32-37.
34. Mestre, V.; Samper, P.; Nacher, M.J.; Tur, A.; Cortes, M.T. Psychological processes and family variables as prosocial behavior predictors in a sample of Spanish adolescents. *Psychol. Rep.* **2006**, *98*, 30-36.
35. Beaver, K.M.; Ratchford, M.; Ferguson, C.J. Evidence of genetic and environmental effects on the development of low self-control. *Crim. Justice Behav*. **2009**, *36*, 1158-1172.

36. Brannigan, A.; Gemmell, W.; Pevalin, D.J.; Wade, T.J. Self-control and social control in childhood misconduct and aggression: The role of family structure, hyperactivity, and hostile parenting. *Can. J. Criminol.* **2002**, *44*, 119-142.
37. Wills, T.A.; Cleary, S.D.; Filer, M.; Shinar, O.; Mariani, J.; Spera, K. Temperament related to early-onset substance use: Test of a developmental model. *Prev. Sci.* **2001**, *2*, 145-163.
38. Longshore, D.; Chang, E.; Messina, N. Self-control and social bonds: A combined control perspective on juvenile offending. *J. Quant. Criminol.* **2005**, *21*, 419-437.
39. Wills, T.A.; Vaccaro, D.; McNamara, G. Life events, family support, and competence in adolescent substance use. *Am. J. Commun. Psychol.* **1992**, *20*, 349-374.
40. Windle, M. The difficult temperament in adolescence: Associations with substance use, family support, and problem behaviors. *J. Clin. Psychol.* **1991**, *47*, 310-315.
41. O'Malley, P.; Johnston, L.D. Epidemiology of alcohol and other drug use among American college students. *J. Stud. Alcohol* **2002**, *14*, 23-39.
42. Slutske, W.S.; Hunt-Carter, E.E; Nabors-Oberg, R.E.; Sher, K.J.; Bucholz, K.K.; Madden, P.A; Anokhin, A.; Heath, A.C. Do college students drink more than their non-college-attending peers? Evidence from a population-based longitudinal female twin study. *J. Abnorm. Psychol.* **2004**, *113*, 530-540.
43. Fromme, K.; Corbin, W.R.; Kruse, M.I. Behavioral risks during the transition from high school to college. *Dev. Psychol.* **2008**, *44*, 1497-1504.
44. Kost, K.; Henshaw, S.; Carlin, L. *US Teenage Pregnancies, Births and Abortions: National and State Trends and Trends by Race and Ethnicity*; Guttmacher Institute: New York, NY, USA, 2010. Available online: http://www.guttmacher.org/pubs/USTPtrends.pdf (accessed on 10 September 2011).
45. Miller, W.C.; Ford, C.A.; Morris, M.; Handcock, M.S.; Schmitz, J.L.; Hobbs, M.M.; Cohen, M.S.; Harris, K.M.; Udry, J.R. Prevalence of chlamydial and gonococcal infections among young adults in the United States. *J. Am. Med. Assoc.* **2004**, *291*, 2229-2236.
46. *Diagnoses of HIV infection and AIDS in the United States and Dependent Areas, 2009*; HIV Surveillance Report; Centers for Diseases and Control and Prevention: Atlanta, GA, USA, 2011; Volume 21. Available online: http://www.cdc.gov/hiv/surveillance/resources/reports/2009report/index.htm (accessed on 10 September 2011).
47. Sobell, L.C.; Sobell, M.B. Timeline follow-back: A technique for assessing self-reported alcohol consumption. In *Measuring Alcohol Consumption*; Litten, R., Allen, J., Eds.; The Humana Press Inc.: Rockville, MD, USA, 1992; pp. 207-224.
48. Weinhardt, L.S.; Carey, M.P.; Maisto, S.A.; Carey, K.B.; Cohen, M.M.; Wickramasinghe, S.M. Reliability of the timeline followback sexual behavior interview. *Ann. Behav. Med.* **1998**, *20*, 25-30.
49. Sobell, L.C.; Brown, J.; Leo, G.I.; Sobell, M.B. The reliability of the Alcohol Timeline Followback when administered by telephone and by computer. *Drug Alcohol Depend.* **1996**, *42*, 49-54.
50. Eysenck, S.B.J.; Eysenck, H.J. The place of impulsiveness in a dimensional system of personality description. *Br. J. Social. Clin. Psychol.* **1977**, *16*, 57-68.

51. Rosenberg, M. *Society and the Adolescent Self-Image*; Princeton University Press: Princeton, NJ, USA, 1965.
52. Kendall, P.C; Wilcox, L.E. Self-control in children: Development of a rating scale. *J. Consult. Clin. Psychol.* **1979**, *47*, 1020-1029.
53. Storr, C.L.; Zhou, H.; Liang, K.-Y.; Anthony, J.C. Empirically derived latent classes of tobacco dependence syndromes observed in recent-onset tobacco smokers: Epidemiological evidence from a national probability sample survey. *Nicotine Tob. Res.* **2004**, *6*, 533-545.
54. Hasking, P.A.; Scheier, L.M.; Abdallah, A.B. The three latent classes of adolescent delinquency and the risk factors for membership in each class. *Aggress. Behav.* **2010**, *37*, 19-35.
55. Fergusson, D.M.; Horwood, L.J.; Lynskey, M.T. The comorbidities of adolescent problem behaviors: A latent class model. *J. Abnorm. Child Psych.* **1994**, *22*, 339-354.
56. Collins, L.M.; Graham, J.W.; Long, J.D.; Hansen, W.B. Crossvalidation of latent class models of early substance use onset. *Multivar. Behav. Res.* **1994**, *29*, 165-183.
57. Coffman, D.L.; Patrick, M.E.; Palen, L.A.; Rhoades, B.L.; Ventura, A.K. Why do high school seniors drink? Implications for a targeted approach to intervention. *Prev. Sci.* **2007**, *8*, 241-248.
58. Flaherty, B.P. Assessing reliability of categorical substance use measures with latent class analysis. *Drug Alcohol Depend.* **2002**, *68*, S7-S20.
59. Clogg, C.C.; Goodman, L.A. Latent structure analysis of a set of multidimensional contingency tables. *J. Am. Stat. Assoc.* **1984**, *79*, 762-771.
60. Hagenaars, J.A.; McCutcheon, A.L. *Applied Latent Class Analysis*; Cambridge University Press: Cambridge, UK, 2002.
61. Schwarz, G. Estimating the dimension of a model. *Ann. Stat.* **1978**, *6*, 461-464.
62. Akaike, H. Factor analysis and the AIC. *Psychometrika*, **1987**, *52*, 317-332.
63. Singer, J.D.; Willett, J.B. *Applied Longitudinal Data Analysis: Modeling Change and Even Occurrence*; Oxford University Press: New York, NY, USA, 2003.
64. Agresti, A. *Categorical Data Analysis*; John Wiley & Sons: New York, NY, USA, 1990.
65. Haberman, S.J. *Analysis of Quantitative Data. Vol. 2. New Developments*; Academic Press: New York, NY, USA, 1979.
66. Kotz, S.; Johnson, N.L. *Encyclopedia of Statistical Sciences*; John Wiley & Sons: New York, NY, USA, 2006.
67. Garrett, E.S.; Zeger, S.L. Latent class model diagnosis. *Biometrics* **2000**, *56*, 1055-1067.
68. Arnett, J.J. Emerging adulthood: A theory of development from the late teens through the twenties. *Am. Psychol.* **2000**, *55*, 469-480.
69. Raffaelli, M.; Crockett, L.J. Sexual risk taking in adolescence: The role of self-regulation and attraction to risk. *Dev. Psychol.* **2003**, *39*, 1036-1046.
70. Wills, T.A.; Stoolmiller, M. The role of self-control in early escalation of substance use: A time-varying analysis. *J. Consult. Clin. Psychol.* **2002**, *70*, 986-997.
71. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
72. Wills, T.A.; Dishion, T.J. Temperament and adolescent substance use: A transactional analysis of emerging self-control. *J. Clin. Child Adolesc.* **2004**, *33*, 69-81.

73. Hofmann, W.; Friese, M.; Strack, F. Impulse and self-control from a dual systems perspective. *Perspect Psychol. Sci.* **2009**, *4*, 162-176.
74. Friese, M.; Hofmann, W.; Wiers, R.W. On taming horses and strengthening riders: Recent developments in research on interventions to improve self-control in health behaviors. *Self Identity* **2011**, *10*, 336-351.
75. Baumeister, R.F.; Gailliot, M.; DeWall, C.N.; Oaten, M. Self-regulation and personality: How interventions increase regulatory success, and how depletion moderates the effects of traits on behavior. *J. Personal.* **2006**, *74*, 1773-1801.
76. Lubke, G.H.; Muthén, B.O. Investigating population heterogeneity with factor mixture models. *Psychol. Methods* **2005**, *10*, 21-39.
77. Nagin, D.S.; Tremblay, R.E. Developmental trajectory groups: Fact or a useful statistical fiction? *Criminology* **2005**, *43*, 873-904.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Freimuth, M.; Moniz, S.; Kim, S.R. Clarifying Exercise Addiction: Differential Diagnosis, Co-occurring Disorders, and Phases of Addiction. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4069-4081; doi:10.3390/ijerph8104069.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Clarifying Exercise Addiction: Differential Diagnosis, Co-occurring Disorders, and Phases of Addiction

Marilyn Freimuth *, Sandy Moniz and Shari R. Kim

School of Psychology, Fielding Graduate University, Santa Barbara, CA 93105, USA;
E-Mails: smoniz180@gmail.com (S.M.); skim@email.fielding.edu (S.R.K.)

* Author to whom correspondence should be addressed; E-Mail: mfreimuth@fielding.edu; Tel.: +1-212-496-7183; Fax: +1-920-983-9120.

Received: 6 September 2011; in revised form: 8 October 2011 / Accepted: 17 October 2011 / Published: 21 October 2011

Abstract: This paper sets out to clarify the unique features of exercise addiction. It begins by examining how this addiction can be distinguished from compulsions and impulse control disorders both of which, like an addiction, involve excessive behavior that creates adverse effects. Assessment of exercise addiction also requires that clinicians be attuned to other forms of excessive behavior, especially eating disorders that can co-occur with exercise. Finally in an effort to clarify exercise addiction, this paper uses the four phases of addiction to examine the attributes of exercise that define it as a healthy habit distinct from an addiction. The paper ends with a discussion of the implications of these topics for effective assessment and treatment.

Keywords: exercise addiction; phases of addiction; behavioral addictions; co-addictions

1. Introduction: Exercise as an Addiction

The upcoming Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [1] will include behavioral addictions. Although gambling will be the only designated behavioral addiction, this new diagnostic nomenclature will no doubt lead to increased research into all forms of excessive behavior, such as exercise, that have been considered to be addictive. This research will require a clear description of exercise addiction as distinct from a healthy habit [2]. As with other behavioral addictions, it will also be necessary to distinguish exercise addiction from compulsions and impulse

control disorders. An understanding of common co-occurring disorders will also be important to the extent that they mask exercise addiction and/or complicate treatment. The following presentation is based on accessing the CORK bibliography on exercise addiction and an extensive PsycINFO search of the topic.

Defining Exercise Addiction

What distinguishes the everyday gym enthusiast from someone addicted to exercise? Would we consider an elite athlete training for the Olympics as having an exercise addiction? What about the devoted runner who adds an extra three miles to his or her running schedule after lunch at a fast food restaurant? Hausenblas and Downs [3,4] identify exercise addiction based on the following criteria that are modifications of the DSM-IV TR [5] criteria for substance dependence:

- *Tolerance*: increasing the amount of exercise in order to feel the desired effect, be it a" buzz" or sense of accomplishment;
- *Withdrawal*: in the absence of exercise the person experiences negative effects such as anxiety, irritability, restlessness, and sleep problems [6];
- *Lack of control*: unsuccessful at attempts to reduce exercise level or cease exercising for a certain period of time;
- *Intention effects*: unable to stick to one's intended routine as evidenced by exceeding the amount of time devoted to exercise or consistently going beyond the intended amount;
- *Time:* a great deal of time is spent preparing for, engaging in, and recovering from exercise;
- *Reduction in other activities*: as a direct result of exercise social, occupational, and/or recreational activities occur less often or are stopped;
- *Continuance:* continuing to exercise despite knowing that this activity is creating or exacerbating physical, psychological, and/or interpersonal problems.

Although others have defined exercise addiction using different models [7-11], the above definition is most closely aligned with the DSM-5 criteria for behavioral addiction which will be modeled after those for substance dependence [1]. Based on a review of a wide range of studies on exercise addiction, Sussman, Lisha, and Griffiths [12] estimate the prevalence in the general population to be close to 3%. Among certain groups such as ultra-marathon runners [8] and sport science students [10] the figure is even higher. According to Lejoyeux, Avril, Richoux, Embouazza, and Navoli [13], 42% of the members at a Parisian fitness club met criteria for exercise addiction.

2. Results and Discussion

2.1. Distinguishing Exercise Addiction from Other Disorders

If research on exercise addiction is to move ahead, it will be important to know when this behavior actually represents an addiction and not some other disorder. Like other behavioral addictions, exercise addiction is often referred to as being compulsive or impulsive. This paper will address the overlaps among exercise addiction, compulsions, and impulse control disorders. Exercise addiction also needs to be distinguished from exercise that occurs at a high frequency. The Olympic athlete may devote a

great deal of time to the activity, experience a significant reduction in other activities and go through withdrawal when the behavior is stopped or cut back. Despite meeting three exercise addiction criteria [3], an elite athlete is not necessarily addicted to his or her sport. Failure to distinguish exercise addiction from exercise done with high frequency and intensity has been a source of confusion in the literature [8,14]. Phases of addiction [2] will be used to distinguish exercise addiction from other forms of intense and frequent exercise behavior. Finally, exercise addiction co-occurs with other addictions that, if left unrecognized, can complicate the treatment process. The frequent link between exercise addiction and eating disorders will be emphasized.

2.2. Exercise Addiction, Exercise Compulsion, or Impulse Control Disorder?

Addictive behavior often is described as impulsive [15]. Impulsivity consists of rapid, unplanned responses to external or internal stimuli. Impulsive behavior is without sufficient contemplation for possible negative consequences and is primarily driven by a desired positive reward [16]. Exercise is a pleasurable activity that, in its addicted form, can occur without full consideration of negative consequences. For example, the addicted runner enjoys this activity and goes for a run despite knowledge of an impending rainstorm that increases the chance of injury. However, unlike an impulse-control disorder there is often considerable thought that precedes the action of engaging in an addiction. Like others who are addicted, the person addicted to exercise often considers the negative consequences but ultimately ignores them [17]. Furthermore addictive behaviors, unlike impulse-control disorders, develop tolerance and withdrawal.

Like other addictive behaviors, excessive exercise often is described as compulsive by theorists [18] and those highly involved with exercise [19]. But is this really a compulsive disorder or simply a term to describe the compelling nature of an addiction? A compulsive disorder consists of ritualized and stereotyped behaviors of which the most common examples are frequent checking and hand-washing [19]. Intrusive thoughts (*i.e.*, obsessions) that accompany compulsive behaviors are very much like the ruminations an addicted person experiences when having an urge or craving to do the behavior. However, Yates' research [20] shows that these obsessive qualities of addictions are distinct from those in obsessive-compulsive disorders. Where obsessions are focused on unrealistic outcomes (e.g., a house fire will ensue if lights are left on), an addicted person ruminates about realistic negative outcomes of his or her behavior.

Compulsive disorders, as a type of anxiety disorder, are maintained primarily by negative reinforcement through anxiety reduction. Research shows that exercise addiction, like a compulsion, is maintained by its mood-altering effects. However, these effects extend beyond anxiety reduction to include lessening other negative affects including anger [21], depression, and boredom [11]. Unlike compulsive behavior, addictions are also maintained by enhancement of positive affect. In the case of exercise, there are the mood-improving effects of aerobic exercise [22] and increased self-esteem as the result of maintaining a disciplined regimen or improving appearance.

According to Goodman [23], addictions are distinguished from impulsive and compulsive behavior by their dual capacity to reduce negative affective states while also creating positive affects be it a rush or improved mood. In those cases in which exercise occurs only for its capacity to reduce anxiety, the

problem may be better thought of as a compulsion. Christenson and colleagues [24] have made a similar argument for excess urge-driven buying that serves only to reduce anxiety.

2.3. Addictions that Co-Occur with Exercise Addiction

While the research on disorders co-occurring with exercise addiction is scant, estimates suggest that 15–20% of exercise addicted individuals are addicted to nicotine, alcohol, or illicit drugs [6]. For example, athletes who use stimulants such as amphetamines, cocaine or caffeine to improve athletic performance can become substance addicted [25,26]. However, when it comes to alcohol and nicotine use, some research has not supported a positive relationship [7]. Sussman, Lisha, and Griffiths [12] suggest that up to 25% of people with one addiction have another addiction. Buying addiction has been identified as common among the exercise addicted [13] while exercise addiction is common among individuals addicted to sex [27]. An analysis of the Shorter Promis Questionnaire (SPQ) that examines 16 potentially addictive behaviors finds that exercise tends to cluster with food disorders, caffeine use, and shopping [28]. A more recent study using the SPQ replicated these findings and added work addiction as another co-occurring disorder [29].

Eating disorders are the most common disorder to co-occur with exercise addiction. Approximately 39–48% of people suffering from eating disorders also suffer from exercise addiction [3,30,31].

The relationship between exercise addiction and eating disorders has significance for diagnosis and treatment. De Coverley Veale [9] distinguished primary and secondary exercise addiction; primary exercise addiction occurs in the absence of an eating disorder. Any weight loss is secondary to calories burned or if there is dieting, it occurs solely for the purpose of improving performance. However, for some people, the primary motivation for exercise is weight loss that occurs in the extreme. This kind of primary exercise addiction has been given a special name: *anorexia athletica* [31,32]. With secondary exercise addiction, exercise is paired with a co-occurring eating disorder. Exercise along with vomiting, laxatives, *etc*. serve to avoid the consequences of calorie consumption. Clinicians may think the latter is limited to women but this problem has been reported in college men [33].

Bamber, Cockerill, Rodgers, and Carroll [30] have argued that exercise addiction is always secondary to an eating disorder. In a questionnaire distributed to 194 women, they were unable to differentiate exercise addiction from eating disorders; in the absence of an eating disorder, participants' exercise did not create the level of distress normally associated with an addiction. However, they focused solely on excessive exercise, which fails to take into account the criteria unique to an addiction, such as withdrawal and tolerance.

When exercise addiction and eating disorders co-occur, the danger is that only one problem will be treated. Often the eating disorder, as the better-known disorder, is the focus of treatment and the secondary exercise addiction remains hidden. Despite the improved relationship to food, the patient still does not gain weight, which is managed through an increase in the exercise regimen [2].

2.4. When is Frequent Exercise Not an Addiction?

One of the thornier issues in defining exercise addiction concerns how to distinguish healthy exercise from exercise addiction. In order to reap the health benefits of exercise, the behavior needs to be engaged in relatively frequently and for extended duration. In fact healthy exercise can share attributes

of an addiction. There can be tolerance in which a person runs farther or lifts more weight before feeling gratified that the workout was worthwhile. Normal exercise does not preclude creating negative consequences in the form of physical injury or time taken away from other important activities.

Freimuth's [2] clinical heuristic for distinguishing phases of addiction can be used to explore the distinctions between recreational exercise and exercise addiction. These phases help a clinician decide when a normal behavior is becoming addictive and when an addictive behavior is returning to normal. Each phase is broken into three components: motivation (referring to the person's motivation for exercising in that stage), consequences, and frequency/control. The following example of Sally, based on a composite of several cases of exercise addiction, will be used to distinguish the phases:

> Sally makes a New Year's Resolution to "get in shape." She begins going to the gym every morning before work. She enjoys how exercise has improved her strength and appearance but enjoys running the most because it helps her forget her worries and leaves her feeling relaxed. She begins running longer distances on the treadmill. As her endurance increases, she decides to train for a five-mile race with a group of other runners. She follows this program to the letter. After successfully completing the race, she feels wonderful and decides to continue the training regimen on her own, gradually increasing her distance. One day, while running on the treadmill, Sally twists her ankle. She has a severe sprain and her doctor recommends she stop running for the next few weeks. On the first day of refraining from exercise, Sally feels a little irritable. Over the next few days, she just doesn't seem herself; she misses running and wonders if she is depressed. She begins to think the doctor over-reacted and decides to go to the gym just to lift some weights. She does this for two days but on the third day she cannot resist the urge to get on the treadmill; she runs until her ankle gives out.

2.4.1. Phase One: Recreational Exercise

Recreational exercise primarily occurs because it is a pleasurable and rewarding activity [2]. This pleasure is represented by Sally's enjoyment of the changes in strength and appearance from exercising. Another example would be a person who enjoys regular hikes because the experience of being in nature is pleasurable. Recreational exercise adds to the quality of life whereas addiction takes away from it [34].

Research shows that other sources of motivation in this phase are achieving health and fitness [35]. The behavior is under control; the person sticks to his or her schedule and is able to stop when planned. With recreational exercise, negative consequences are rare, unexpected, and usually a direct outcome of the exercise itself (e.g., a sore muscle, a sprained ankle).

2.4.2. Phase Two: At-Risk Exercise

The recreational level provides the opportunity to discover whether a behavior is intrinsically rewarding, and herein lays the risk. Recreational exercise exposes a person to the potentially mood-altering effects of this behavior. Sally discovers that running has a special effect; it helps her escape her worries.

There is a great deal of evidence to show that exercise has mood-altering effects. Exercise serves to increase positive affect such as increasing self-esteem and decreasing the negative affect associated with depression and anxiety [22].

In some cases, these mood changes have been attributed to altered chemical functioning of the brain. Griffiths [36], after reporting a detailed case study of exercise addiction, proposed three possible biological mechanisms to connect improved mood and exercise:

- The Thermogenic Hypothesis: exercise increases body temperature, thereby reducing somatic anxiety. This decrease in anxiety is related to an increased temperature in certain brain regions [37];
- The Catecholamine Hypothesis: exercise releases catecholamines, which are strongly implicated in control of mood, attention, and movement as well as endocrine and cardiovascular responses linked to stress [38];
- The Endorphin Hypothesis: exercise releases endorphins, which are opiates that occur naturally in the body. This pleasurable experience of exercise may have unplanned consequences. With regular intense aerobic exercise, the increased endorphin production results in the brain down-regulating endorphin production. If this happens, the person will need to continue the exercise in order to maintain the natural balance in the brain [39].

Research with rats suggests another reason why exercise behavior, once it occurs at high frequency, may need to be maintained at high levels. Rats exposed to extreme exercise showed changes in the neurotransmitter dopamine that has been shown to play a significant role in other addictions. This kind of intense exercise in rats reduced the rewarding effect of other substances that also induce dopaminergic responses. Assuming this effect translates to humans, Adams [39] argued that it is possible that with reduced hedonic pleasure from other activities, a person may have to maintain the intense exercise in order to optimally activate reward circuitry of the mesolimbic dopamine system.

The mood-altering effects of exercise are available to all people, but not all people who exercise with increasing frequency and intensity ultimately become addicted to exercise. Those who do not develop problems with their exercise can be considered "highly engaged" in this behavior. Research on highly engaging behavior shows that it shares three common features with addiction: frequent thoughts about the behavior, positive feelings in response to the behavior, and tolerance (*i.e.*, doing more of the behavior to get a good feeling) [2,40]. At one point, Sally is highly engaged in her running as she trains daily with a group, begins to increase her distance, and "feels wonderful" after completing her race.

There are a number of risk factors that help predict if a highly engaging behavior becomes fully addictive. These factors are biological (*i.e.*, genetics and neurological) and psychological (*i.e.*, negative peers, parental drug use, low self-esteem, juvenile delinquency, and low levels of social conformity) [41]. While these factors are known to influence addiction in general, there is evidence demonstrating that they play a role in exercise addiction.

Gapin, Etnier, and Tucker [42] linked frontal lobe asymmetry to increased risk of exercise addiction. Knowing that exercise serves to reduce negative affect, they demonstrated that women's scores on the Exercise Addiction Inventory correlated with increased frontal lobe asymmetry that is a measure of negative affectivity.

On the psychological level, what distinguishes recreational exercise from exercise at-risk of increasing in frequency is the motivation. As LaRose, Lin, and Eastin [43] have shown with Internet

use, an addiction is more likely when the primary motivation is *not* enjoyment from the activity but rather relief from stress or other types of dysphoria or to improve self-esteem. Thornton and Scott [35] have shown this effect for exercise; the likelihood of an addiction increases for those who exercise with the goal of escaping unpleasant feelings or transforming their appearance to improve self-esteem as compared to those who exercise with the goal of improving performance and fitness. The capacity of a behavior, like exercise, to serve a larger function, such as coping with unpleasant experiences, makes it more likely that the behavior will continue to increase in frequency and become problematic. Addiction is most likely to occur when the behavior is the primary or sole means of coping with internal distress [2].

In terms of observable signs, this important transitional phase from at-risk to early addiction is marked by periodic loss of control of the behavior that occurs for longer periods of time or is more intense than intended. Negative consequences increase in frequency. At the at-risk phase, these negative consequences are primarily a direct result of exercise (as distinct from later adverse effects which are more interpersonal in nature). Sally's sprained ankle is a direct negative effect of her running.

2.4.3. Phase Three: Problematic Exercise

Where recreational exercisers integrate their daily physical activity into their lives, those whose exercise is becoming problematic begin to organize their day around their excise regimen, which is becoming more and more rigid [44]. Another distinguishing feature of exercise at the problematic level is the nature of negative consequences. Where previously adverse effects arose directly from the behavior, at the problematic level secondary negative consequences are predominant. Secondary negative consequences include one's own and/or another's response to the adverse effects of exercise [2]. An example of a secondary negative consequence for Sally would be if her boyfriend began complaining about feeling she preferred running to being with him or if Sally became angry with herself for reinjuring her ankle.

Once in the problematic phase, the behavior continues despite having met the stated goal—much like the problematic drinker who continues to drink even after the desired stress relief from alcohol has occurred. In Sally's case, after achieving the gratification of meeting her goal of running a five-mile race, she continues her diligent training regimen. Also common to the problematic level is that a behavior, once done socially, now occurs alone. Sally stops training with a group to train on her own.

Maintaining control over the behavior becomes more difficult in the problematic phase because, when the behavior ceases, withdrawal symptoms set in. The idea that behavioral addictions are associated with the classic signs of dependence-tolerance and withdrawal has been debated. At least with exercise, there is good evidence that aerobic exercise and intense use of large muscles is associated with changes in endorphins. If the body down-regulates endorphin production in response to exercise, the absence of exercise can be associated with withdrawal. Withdrawal from exercise has been demonstrated [45] and is illustrated by Sally's irritability and not feeling like herself after her ankle injury. At this level, the behavior no longer occurs just for its mood-altering effects but also to remove withdrawal symptoms.

At the problematic level there are also signs that the behavior is becoming indiscriminant. Kohut [46] addressed this concept with regard to alcohol although it applies equally to other

addictions: "for the alcoholic, alcohol is the important thing. It does not matter if it is good or bad bourbon, good or bad wine" (p. 118). While one form of behavior is preferred, at this stage, the exercise addict will try other forms of exercise when their preferred form is not available [36]. Sally begins doing more weight training while trying to wait for her ankle to heal.

2.4.4. Phase Four: Exercise Addiction

The frequency and intensity of exercise continues until this behavior becomes life's main organizing principle. The addicted athlete feels the physical rush and sense of gratification but continues to run further distances, lift more weights, or attend more gym classes. Consistent with the paradoxical nature of addiction, a behavior that began as a way to make life more bearable by facilitating coping ultimately makes life unmanageable. As the life of the addicted person revolves around exercise, the pleasure of the behavior recedes as the primary motivation becomes avoiding withdrawal symptoms.

Direct and secondary negative consequences continue to mount leading to tertiary negative consequences in the form of impairments in daily functioning and inability to meet role obligations. In the case of Sally, the loss of relationships due to her running and continued self-blame (secondary negative consequence that follows re-injuring her ankle) could result in a tertiary negative consequence of becoming clinically depressed.

3. Implications and Conclusions

3.1. Assessment and Treatment Implications

The Exercise Dependence Scale (EDS-R) was developed based on modification of the criteria for substance dependence [1,4]. The 21 items are responded to on a 6 point Likert Scale. The Exercise Addiction Inventory or EAI [10] represents another measurement tool based on Griffiths' modification of Brown's model of addiction [47]. Both the EDS-R and EAI have good validity and reliability. The EAI, with just six questions, is designed for use in medical and health care settings where assessment time is limited. Both scales yield three possible categories: asymptomatic, symptomatic, and exercise addicted.

When it comes to treating this addiction, abstinence from exercise may not be the required goal. Because exercise in moderation is considered a healthy habit, a typical treatment goal will be to return to moderate exercise. In some cases, a new form of exercise may be recommended; the runner becomes a swimmer. In other cases the person may continue to do the same form of exercise in a more controlled or moderate manner [34]. Whether moderating the original exercise behavior or replacing one activity with another, clinicians can use the attributes of the four phases of addiction as a way to help patients distinguish problematic or addictive exercise from moderate or recreational exercise.

There is scant literature on actual treatment of exercise addiction. Like most behavioral addictions, usually some form of cognitive-behavioral therapy is recommended [39]. One of the first issues will be to motivate clients for treatment given that qualitative studies of exercise addiction suggest that clients are insufficiently attuned to the adverse effects created by their behavior [44]. Without this recognition, exercise behavior is unlikely to be considered a problem meriting treatment. Once motivated, attention can turn to identifying and correcting automatic thoughts such as those related to

the need to control the body [36] and the idea that exercise is always good even if it is done in a driven/obsessive manner [44]. Behavioral strategies, such as contingency management, that reward abstinence from a type of exercise or maintaining lower levels of a once addictive behavior, have also been recommended [39].

Whether assessing or treating exercise addiction it is always important to be attuned to the common co-occurring disorders, especially if it is an eating disorder or food related problem. If only the exercise addiction is treated, as exercise is reduced, a person will resort to increased bulimia or anorexic behavior in order to maintain low weight levels. Similarly the eating disorder specialist must attend to exercise behaviors in order that increased caloric intake is not compensated for by more intense exercise.

Clinicians who specialize in substance-related disorders and behavioral addictions such as sex, work, and shopping will want to be attuned to exercise addiction [13,27-29]. There is some evidence that exercise relieves withdrawal symptoms associated with cocaine addiction [48] but use of exercise for this purpose may open the way for an exercise addiction. In the case of behavioral addictions, as the primary addictive behavior decreases in frequency, it is possible that once moderate exercise becomes problematic as this behavior replaces the mood-altering functions of the initial addiction [27]. The need to be attentive to signs of exercise addiction is not limited to those who treat addictions. Some psychotherapy patients will use exercise as a primary form of mood regulation. The challenges of psychotherapy can lead to an increase in this behavior. Being aware of the phases of exercise addiction will help clinicians assess if recreational or at-risk exercise is becoming problematic or fully addictive.

Being attuned to such changes is essential for clinicians who recommend exercise to their patients. Recent research shows that exercise, with its ability to improve mood, can be an important adjunct to many different kinds of treatment [49] including eating disorders [17]. When exercise is recommended for its mood-altering effects, clinicians will want to be sure that the recommended regimen is not exceeded and remain attuned to any of the signs that exercise is becoming problematic.

3.2. Conclusion

The inclusion of behavioral addictions in DSM-5 [1] invigorates the quest to identify which kinds of addictive-like behaviors, other than gambling, should be included under this newly formed diagnostic category. To determine if exercise meets the criteria for an addiction, this paper set out to clarify the primary attributes and distinctive features of exercise addiction. In accomplishing this goal, topics also were addressed that inform clinicians about how to better identify and treat exercise addiction.

While there are several main approaches to identifying exercise addiction [7-11], this paper relied on Hausenblas and Downs' [3,4] approach that is derived from a modification of DSM-IV TR [5] substance dependence criteria. Preference for their approach was largely shaped by the fact that these criteria will most likely be used to define behavioral addictions in future DSM versions. A number of attributes of exercise addiction (e.g., tolerance, obsessive thoughts) overlap with the exercise of committed athletes whose regimen involves intense exercise for long durations. However, as Freimuth [2] has argued, quantity/intensity of a behavior has never been a good measure of addiction.

Instead, one can look to the four phases of addiction to distinguish such "highly engaging" exercise from addictive exercise.

Having presented one set of attributes for identifying exercise addiction, this paper addressed the question of whether exercise addiction is a distinct disorder or simply a manifestation of another disorder. Research was reviewed that demonstrated that exercise addiction, while a problem that commonly co-occurs with eating disorders, exists independent of an eating disorder [31,32]. Exercise addiction cannot simply be reduced to a compulsion or impulse control disorder either. Rather, like other high frequency behaviors that create adverse effects and are maintained by both positive and negatively reinforcement, certain patterns of exercise are best described as an addiction [23].

Although exercise addiction is not included in DSM-5, it is imperative that many kinds of health care providers become familiar with its attributes. The physician may see repetitive injuries and not recognize this as a sign that the compulsion to exercise prevents an injury from full healing. For psychotherapists, the patient committed to exercise may develop an addiction during the course of therapy if exercise is the primary means to manage the emotional demands of change. Addiction specialists also must remain attuned to the signs and symptoms of exercise addiction given its co-occurrence with substance use disorders and other behavioral addictions such as sex, work, and buying/shopping [13,25-27,29].

To date, treatment approaches for exercise addiction draw primarily from cognitive-behavioral principles used to manage other behavioral addictions. Whatever treatment is used, early identification of this problem will make it easier to treat [2]. To this end, the four phases of addiction were reviewed in order to highlight the attributes of an emerging exercise addiction. This system is not only useful for early identification but can be used when defining treatment goals and helping the patient distinguish recreational from addictive exercise.

References

1. American Psychiatric Association. *DSM 5 Development*; 2010. Available online: http://www.dsm5.org/Pages/Default.aspx (accessed on 30 September 2011).
2. Freimuth, M. *Addicted? Recognizing Destructive Behavior before It's too Late*; Rowman & Littlefield Publishers, Inc: Lanham, MD, USA, 2008.
3. Hausenblas, H.A.; Downs, D.S. How much is too much? The development and validation of the Exercise Addiction scale. *Psychology and Health* **2002**, *17*, 387-404.
4. Downs, D.S.; Hausenblas, H.A.; Nigg, C.R. Factorial validity and psychometric examination of the Exercise Dependence Scale-Revised. *Meas. Phys. Educ. Exerc. Sci.* **2004**, *8*, 183-201.
5. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (Text Revision)*, 4th ed.; American Psychiatric Association: Washington, DC, USA, 2000.
6. Aidman, E.V.; Woollard, S. The influence of self-reported exercise addiction on acute emotional and physiological responses to brief exercise deprivation. *Psychol. Sport Exerc.* **2003**, *4*, 225-236.
7. Allegre, B.; Souville, M.; Therme, P.; Griffiths, M. Definitions and measures of exercise dependence. *Addict. Res. Theory* **2006**, *14*, 631-646.

8. Allegre, B.; Therme, P.; Griffiths, M. Individual factors and the context of physical activity in exercise dependence: A prospective study of "ultra-marathoners". *Int. J. Ment. Health Addict.* **2007**, *5*, 233-243.
9. De Coverley Veale, D.M. Exercise addiction. *Br. J. Addict.* **1987**, *82*, 735-740.
10. Terry, A.; Szabo, A.; Griffiths, M. The exercise addiction inventory: A new brief screening tool. *Addict. Res. Theory* **2004**, *12*, 489-499.
11. Zmijewski, C.F.; Howard, M.O. Exercise Addiction and attitudes toward eating among young adults. *Eat. Behav.* **2003**, *4*, 181-195.
12. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority? *Eval. Health Prof.* **2011**, *34*, 3-56.
13. Lejoyeux, M.; Avril, M.; Richoux, C.; Embouazza, H.; Nivoli, F. Prevalence of exercise addiction and other behavioral addictions among clients of a Parisian fitness room. *Comprehensive Psychiatry* **2008**, *49*, 353-358.
14. Meyer, C.; Taranis, L.; Goodwin, H.; Haycraft, E. Compulsive exercise and eating disorders. *Eur. Eat. Disord. Rev.* **2011**, *19*, 174-189.
15. Brewer, J.A.; Potenza, M.N. The neurobiology and genetics of impulse control disorders: relationships to drug addictions. *Biochem. Pharmacol.* **2008**, *75*, 63-75.
16. Grant, J.E.; Potenza, M.N. Compulsive aspects of impulse-control disorders. *Psychiatr. Clin. North Am.* **2006**, *29*, 539-549.
17. Cook, B.; Hausenblas, H.; Tuccitto, D.; Giacobbi, P.R., Jr. Eating disorders and exercise: A structural equation modeling: Analysis of a conceptual model. *Eur. Eat. Disord. Rev.* **2011**, *19*, 216-225.
18. Cox, R.; Orford, J. A qualitative study of the meaning of exercise for people who could be labelled as "addicted" to exercise—can "addiction" be applied to high frequency exercising? *Addict. Res. Theory* **2004**, *12*, 167-188.
19. Iannos, M.; Tiggemann, M. Personality of the excessive exerciser. *Pers. Indiv. Differ.* **1997**, *22*, 775-778.
20. Yates, A. *Compulsive Exercise and Eating Disorders*; Brunner/Mazel: New York, NY, USA, 1991.
21. Rosa, D.A.; De Mello, M.T.; Negrao, A.B.; De Souza-Formigoni, M.L.O. Mood changes after maximal exercise testing in subjects with symptoms of exercise dependence. *Percept. Mot. Skills* **2004**, *99*, 341-353.
22. Scully, D.; Kremer, J.; Meade, M.M.; Graham, R.; Dudgeon, K. Physical exercise and well-being: A critical review. *Br. J. Sports Med.* **1998**, *32*, 111-120.
23. Goodman, A. *Sexual Addiction: An Integrated Approach*; International Universities Press: Madison, CT, USA, 1998.
24. Christenson, G.A.; Faber, R.J.; de Zwaan, M.; Raymond, C.; Specker, M.; Ekern, D.; Mitchell, J.E. Compulsive buying: Descriptive characteristics and psychiatric comorbidity. *J. Clin. Psychiatr.* **1994**, *55*, 5-11.
25. George, A.J. Central nervous system stimulants. *Best Practice & Research Clinical Endocrinology & Metabolism* **2000**, *14*, 79-88.

26. National Institute on Drug Abuse. *InfoFacts: Steroids (Anabolic-Androgenic)*; National Institutes of Health: Washington, DC, USA, 2009; pp. 1-4. Available online: http://www.drugabuse.gov/PDF/Infofacts/Steroids09.pdf (accessed on 22 August 2011).
27. Carnes, P.J.; Murray, R.E.; Charpentier, L. Bargains with chaos: Sex addicts and addiction interaction disorder. *Sexual Addiction and Compulsivity* **2005**, *12*, 79-120.
28. Haylett, S.A.; Stephenson, G.M.; LeFever, R.M.H. Covariation of addictive behaviors: A study of addictive orientation using the Shorter Promis Questionnaire. *Addict. Behav.* **2004**, *29*, 61-71.
29. MacLaren, V.V.; Best, L.A. Multiple addictive behaviors in young adults: Student norms for the Shorter PROMIS questionnaire. *Addict. Behav.* **2010**, *35*, 252-255.
30. Bamber, D.J.; Cockerill, I.M.; Rodgers, S.; Carroll, D. Diagnostic criteria for exercise addiction in women. *Br. J. Sports Med.* **2000**, *37*, 393-400.
31. Klein, D.A.; Bennett, A.S.; Schebendach, J.; Foltin, R.W.; Devlin, M.J.; Walsh, B.T. Exercise "addiction" in anorexia nervosa: Model development and pilot data. *CNS Spectrums* **2004**, *9*, 531-537.
32. An Overview of Activity Anorexia. In *Activity Anorexia: Theory, Research, and Treatment*; Epling, W.F., Pierce, W.D., Eds.; Lawrence Erlbaum Associates: Mahwah, NJ, USA, 1996; pp. 3-11.
33. O'Dea, J.A.; Abraham, S. Eating and exercise disorders in young college men. *J. Am. Coll. Health* **2002**, *50*, 273-278.
34. Griffiths, M.D. A "components" model of addiction within a biopsychosocial framework. *J. Subst. Use* **2005**, *10*, 191-197.
35. Thornton, E.W.; Scott, S.E. Motivation in the committed runner: Correlation between self-report scales and behavior. *Health Promot. Int.* **1995**, *10*, 177-184.
36. Griffiths, M. Exercise addiction: a case study. *Addict. Res.* **1997**, *5*, 161-168.
37. Craft, L.L.; Perna, F.M. The benefits of exercise for the clinically depressed. *Prim. Care Companion J. Clin. Psychiatry* **2004**, *6*, 104-111.
38. Stahl, S.M. *Stahl's Essential Psychopharmacology: Neuroscientific Basis and Practical Applications*, 3rd ed.; Cambridge University Press: New York, NY, USA, 2008.
39. Adams, J. Understanding exercise addiction. *J. Contemp. Psychother.* **2009**, *39*, 231-240.
40. Charlton, J.P. A factor-analytic investigation of computer "addiction" and engagement. *Br. J. Psychol.* **2002**, *93*, 329-344.
41. Crabbe, J.C. Genetic contributions to addiction. *Annu. Rev. Psychol.* **2002**, *53*, 435-462.
42. Gapin, J.; Etnier, J.; Tucker, D. The relationship between frontal brain asymmetry and exercise addiction. *J. Psychophysiol.* **2009**, *23*, 135-142.
43. Larose, R.; Lin, C.A.; Eastin, M.S. Unregulated internet usage: Addiction, habit, or deficient self-regulation? *Media Psychol.* **2003**, *5*, 225-253.
44. Johnston, O.; Reilly, J.; Kremer, J. Excessive exercise: From quantitative categorisation to a qualitative continuum approach. *Eur. Eat. Disord. Rev.* **2011**, *19*, 237-248.
45. Adams, J.; Kirkby, R.J. Excessive exercise as an addiction: A review. *Addict. Res. Theory* **2002**, *10*, 415-437.
46. Kohut, H. *The Kohut Seminars on Self-Psychology and Psychotherapy with Adolescents and Young Adults*; Elson, M., Ed.; Norton: New York, NY, USA, 1987.

47. Brown, R.I.F. A theoretical model of the behavioural addictions—Applied to offending. In *Addicted to Crime?*; Hodge, J.E., McMurran, M., Hollins, C.M., Eds.; John Wiley: Chichester, UK, 1997.
48. Drug Addiction. Exercise may be beneficial for overcoming cocaine addiction. In *Drug Addiction Treatment.* Available online: http://www.drugaddictiontreatment.com/drug-addiction-treatments/exercise-may-be-beneficial-for-overcoming-cocaine-addiction/ (accessed on 3 December 2010).
49. Herring, M.P.; O'Connor, P.J.; Dishman, R.K. The effect of exercise training on anxiety symptoms among patients: A systematic review. *Arch. Intern. Med.* **2010**, *170*, 321-331.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Bergmark, K.H.; Bergmark, A.; Findahl, O. Extensive Internet Involvement—Addiction or Emerging Lifestyle? *Int. J. Environ. Res. Public Health* **2011**, *8*, 4488-4501; doi:10.3390/ijerph8124488.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Extensive Internet Involvement—Addiction or Emerging Lifestyle?

Karin Helmersson Bergmark [1,*], Anders Bergmark [2] and Olle Findahl [3]

[1] Department of Sociology & Addiction Research Group, Stockholm University, S-10691 Stockholm, Sweden
[2] Department of Social Work & Addiction Research Group, Stockholm University, S-10691 Stockholm, Sweden; E-Mail: anders.bergmark@socarb.su.se
[3] World Internet Institute, P.O. Box 975, S-80133 Gavle, Sweden; E-Mail: olle.findahl@wii.se

* Author to whom correspondence should be addressed; E-Mail: karin.bergmark@sociology.su.se; Tel.: +46-8-163-187; Fax: +46-8-612-5580.

Received: 24 October 2011; in revised form: 25 November 2011 / Accepted: 28 November 2011 / Published: 2 December 2011

Abstract: In the discussions for the future DSM-5, the Substance-Related Disorders Work Group has been addressing "addiction-like" behavioral disorders such as "Internet addiction" to possibly be considered as potential additions for the diagnostic system. Most research aiming to specify and define the concept of Internet addiction (or: Excessive/Compulsive/Problematic Internet Use—PIU), takes its point of departure in conventional terminology for addiction, based in established DSM indicators. Still, it is obvious that the divide between characteristics of addiction and dimensions of new lifestyles built on technological progress is problematic and far from unambiguous. Some of these research areas are developing from the neurobiological doctrine of addiction as not being tied to specific substances. The concept of "behavioral addictions", based on biological mechanisms such as the reward systems of the brain, has been launched. The problems connected to this development are in this study discussed and reflected with data from a Swedish survey on Internet use (n = 1,147). Most Swedes (85%) do use the Internet to some degree. The prevalence of excessive use parallels other similar countries. Respondents in our study spend (mean value) 9.8 hours per week online at home, only 5 percent spend more than 30 hours per week. There are both positive and negative social effects at hand. Many respondents have more social contacts due to the use of Internet, but there is a decline in face-to-face contacts. About 40% of the respondents indicate some

Int. J. Environ. Res. Public Health **2011**, *8*

experience of at least one problem related to Internet use, but only 1.8% marked the presence of all problems addressed. Most significant predictors for problem indicators, except for age, relate to "time" and time consuming activities such as gaming, other activities online or computer skills.

Keywords: Internet; addiction; behavioral addiction

1. Introduction

1.1. Background

The concepts "addiction" and "dependence" were originally developed and elaborated to handle the diagnosis of individuals displaying excessive and problematic use of alcohol and other drugs (e.g., opiates). Presently the two concepts are often used interchangeably [1]. Indicators for the concept include tolerance, withdrawal, preoccupation, craving, impaired control and continued use despite clear evidence of adverse consequences. Lately these indicators have been applied to not only the intake of substances but also to different behaviours and practices. In the discussions for the future DSM 5, the Substance-Related Disorders Work Group has been addressing "addiction-like" behavioural disorders such as "Internet addiction" to possibly be considered as potential additions for the diagnostic system. An expansion of the addiction concept is also advocated from different corners of the research community (see e.g., [2]).

Most research aiming to specify and define the concepts of Internet addiction (or excessive, compulsive or pathological Internet use), takes its point of departure in conventional terminology for addiction that is based in established DSM indicators. Still, it is obvious that the divide between characteristics of addiction and dimensions of new lifestyles built on technological progress is problematic and far from unambiguous. Some of these research areas are developing from the neo-biological doctrine of addiction as not being tied to specific substances. The concept of "behavioural addictions", based on biological mechanisms such as the reward systems of the brain, has been launched. In this paper the notion of Internet addiction is discussed and illuminated with data from a Swedish general population survey on Internet use. In comparison to studies that have made use of self-selected convenience samples, such an approach opens up for a broader discussion on the prevalence and characteristics of Internet addiction (or problems related to extensive Internet involvement).

We expected to be able to provide some indications of how experiences of Internet use problems are related to such variables as age, specific Internet activities, general computer skills and time online. Besides the inclusion of more conventional background factors (as age, sex, educational level and living conditions), we chose to include possible predictors for Internet use problems. We assumed that some specific Internet activities (e.g., gaming), general computer skills and time online would display positive correlations to problem indicators. In the case of computer skills we assumed that this reflects a general technological interest that enables the individual to use the Internet in more elaborated ways and in this way might be related to the prevalence of problems.

Int. J. Environ. Res. Public Health **2011**, *8*

1.2. Swedes and Internet

The significance of the Internet has gone from being a work-related tool for certain groups to something most Swedes use for many activities [3]; from gaming to bank transactions and romantic interactions. Internet gives many opportunities for interaction between individuals, without any limitations set by geography or social ties. Gaming and other social activities form a large part of many Swedes' life online. In addition, aspects of our social life and relations have shifted—or expanded—through the upsurge of digital arenas. During the last ten years the share of Swedes who go online on a regular day has risen from 21% to 65% [4]. In 2010, 85% of the population had access to Internet in their homes [5].

1.3. Internet Addiction

During the last decade different forms of Internet use have been in focus for substantial research efforts. Some studies include positive aspects as well as negative. Shen and Williams [6] analysed survey and game-based data for over 5,000 EverQuest gamers and conclude that such games can have both positive and negative consequences for sociability and family life. Pre-existing social ties were reinforced via online contacts. The game also offered an alternative possibility for family communication. Gaming together in the family was positive in terms of solidifying family relations. On the other hand, gamers who did not play with family members displayed, in comparison, higher levels of loneliness. Purposes for gaming, contexts and individual features of the gamer, delineates according to these authors consequences of extended life online—as time displacement or social augmentation.

There has also been an upsurge of problem-oriented studies that advocate concepts such as "behavioural addiction". While neurobiological scholars advocating the concept of addiction previously used to limit discussions to "drug and alcohol addiction" (e.g., [7]), it is at present increasingly argued that many behaviours are as apt to produce short term reward, and provoke continued behaviour and diminished control [8]. The focal point is an attraction, residing in the reward system of the brain, that makes it impossible to resist an impulse to use or do a behaviour of some type. An aggravating circumstance is that comorbidity (behavioural and substance addictions) is claimed to be common [8-11]. This development has also opened the door for a widening of the boundaries concerning which activities that can be considered plausible to develop into an addiction. Some authors argue that food [12] can act as a trigger to addiction in ways similar to alcohol or tobacco. Excessive exercise has been described in similar ways [13,14]. References to pathological gambling are more common [15]. Pathological gambling is included in DSM lV and is diagnosed via indicators for tolerance, withdrawal, preoccupation, a strong desire, impaired control and continued use despite adverse consequences.

In research from the field of "Internet addiction", one key reference is Kimberly Young [10] and her study from the 1990s [16,17]. Drawing from DSM definitions for pathological gambling and her own empirical studies she participated in the development of the concept "Internet addiction" and in forming an instrument for assessment. In a more recent paper, Young claims that Internet based gaming constitutes a now rapidly growing form of Internet addiction, especially among children and

teenagers [18]. The indicators Young uses measure salience, mood modification, tolerance, withdrawal, conflict and relapse (see also [19]) and are parcelled in eight questions.

Respondents who answered yes to five or more of the questions were classified as addicted [16]. Questions referred to all sorts of online activity. The study group was defined by self-selection from different sources; via newspaper advertisements, flyers on college campuses, Internet addiction support groups, and posted online for web-searches on "Internet addiction". Six out of ten respondents were women (middle-aged; older than male respondents). Four out of ten had no vocational background. The support groups included "the Internet Addiction Support Group" and "Webaholics Support Group". When the 12-step language, programme and ideology is established in the group from where respondents are found, it should not be surprising if respondents spontaneously present narratives on e.g., withdrawal, loss of control and craving. Still, Young concludes that she could distinguish a group of addicts who spent long time online "for pleasure" (mean time 38.5 hours per week), who preferred interactive sites more than others and who describe themselves as "completely hooked". She claims that the addicts reported lots of life problems but there are no records of how this was measured.

Gilbert *et al.* [9] used the Internet addiction test suggested by Young in a study based on a convenience sample of Second Life-users. Van Rooij *et al.* [20] developed a similar list of questions from a manual for CIU (Compulsive Internet Use). Shapira *et al*. [21] present a general definition of Internet addiction based on one symptom: loss of control and two "signs": distress and impairment of daily routines.

Weinstein and Lejoyeaux [10] suggest four components for the diagnosis of Internet addiction (dependence). Excessive Internet use and loss of sense of time or basic drives is the first. The second is based on indications of withdrawal; tension or depression when unable to be online. The third component is tolerance; e.g., need for more time online and the forth include adverse consequences such as arguments and social isolation. These authors are, however, quite critical of previous use of diagnostic tools for Internet Addiction and the main conclusion is that "more research is needed".

Given the fact that there are several rather obvious problems connected to the concept of "Internet addiction" it is somewhat surprising that the Substance-Related Disorders working Group for the upcoming DSM 5 has identified Internet addiction as the main candidate for future inclusion under the new section "Addiction and Related Disorders". O'Brien (p. 2, [22]) notes that "Other non-pharmacological addictions were also reviewed, but only gambling met the criteria for inclusion at this time: Internet addiction will be recommended for the Appendix in order to encourage further research". A major problem related to the ambition to substantiate and delimit Internet addiction is the fact that the Internet is a technology that enables individuals to engage in an almost endless number of activities, but it is not an activity in itself. To the extent that anyone is considered to be addicted to his or her activities that are made possible through the use of the Internet, it is most likely to be activities such as online gaming, gambling, chatting or network communicating (as e.g., Facebook).

Thus, it can be claimed that the concept of internet addiction lacks specificity. Sussman *et al*. [23] have suggested that "addiction specificity", *i.e.*, why one pattern of addictive behaviours may be acquired whereas another is not, can be illuminated within a model that identifies four dimensions; pragmatics, attraction; communication and expectation (PACE). The first component, pragmatics, concerns the accessibility of a particular addictive behaviour. There must be a supply of the object of addiction, in the case at hand the Internet must be established in order to supply Internet gaming,

gambling or chatting. An individual also needs to be aware of this supply of activities as well as have the acquisition skills of how to use computers and connect to the Internet. Lastly the individual needs to have the means of exchange in order to get access to the object of addiction (*i.e.*, economic resources that allows the individual to have an Internet connection). In the perspective of the PACE model of addiction specificity, the idea of Internet addiction leaves out the aspects of attraction, communication and expectation. The idea of Internet addiction lacks a specification of the actual activity that is the object of attraction (as e.g., gaming, gambling or chatting) and consequently the characteristics of the remaining dimensions of the PACE model—communication and expectation—cannot be identified.

Moreover, the activities that are possible to engage in do not constitute a stable configuration; on the contrary, there is a constant transformation of activities that are provided through the Internet. An attempt to solve this type of problem is to use a terminology that has a more limited scope, e.g., the specified category of "gaming addiction". Although such a change represents a step in the right direction it does not solve all problems. There are many different types of games and different way of playing them. Shen and Williams (p. 125, [6]) identifies "a universe of playable titles now over 30,000 dozens of genres, and myriad play options within most titles".

2. Methodology

2.1. This Study

Research on Internet and virtual worlds is currently at hand in many disciplines. Most often this research is based on ethnographic data or surveys based on self-selected study groups [24]. We searched for more representative data where indicators used by Young [16] were included. Therefore we have, for this study, chosen to use a survey from 2009; "Swedes and Internet" [25], the Swedish contribution to the World Internet Project (WIP). In Sweden these surveys on Internet use have, in the form of a revolving panel study, been carried through almost annually since the Year 2000. Respondents are interviewed via telephone or the web (respondents' choice). The survey from 2009 was selected since this survey included five indicators related to "Internet Addiction". Internal dropout was for most variables not higher than 5%. Listwise deletion was used for analyses. Only Internet users (83.2% of all respondents) were included in this study; n = 1,147. The group of Internet users demonstrated similar distributions on background variables as the whole group of respondents. There is no information available on external drop-outs and, for a survey with this kind of design, drop-out rates are difficult to calculate. We would expect overrepresentation of respondents with an interest for computers and Internet. On the other hand, the frequency of Internet users in this study parallels other sources for national Internet statistics [26]. Also, background characteristics are comparable to other national Internet data.

2.2. Methodology and Analyses

For this study we included a number of independent variables. Demographics included sex (gender), year of birth and residence (urban/rural). Educational level was measured with highest degree obtained. For occupation we constructed an indicator with two positions (1: working or student; 2:

unemployed, on parental leave, on sick leave, or on pension leave). An indicator of household income was deleted due to high missing data on that question (*i.e.*, 35%).

Gaming experiences were assessed with the item "How often, if ever, do you use Internet for gaming" (never–several times per day) and gambling experiences was assessed with "How often, if ever, do you use Internet for gambling" (never–several times per day). Membership in communities on Internet (e.g., Facebook) as well as other kinds of online activities were assessed. Computer skills was assessed with the items, "How skilled do you consider yourself to be when it comes to computer use?" (not at all skilled–very skilled) and "Do you know how to install an operating system on a computer?" (no–yes). Indicators of social life was assessed with the items, "Has the use of Internet led to less/the same/more contact with your family/friends?" (much less–much more) and "Would you say that since you started to use the Internet you spend less/the same/more time in face-to-face contact with your family/friends?" (less time–more time).

As a measure for time online we used a question on how much time (in hours and minutes) during a regular week the respondent spends on Internet at home. This does not directly correspond to Young's data on time "for pleasure" online but is the closest we can get though not including online activities at work or in school.

As indicators of Internet related problems we included all five indicators available. These were: "Have you ever spent too much time on Internet", "Do you ever come to quarrel with your family over (your) excessive Internet use", "Do you ever feel depressed, irritated or annoyed because you cannot be online", "Did you ever forget about usual needs such as eating or sleeping due to Internet", and "Have you ever tried and failed to cut down on time on Internet?" All five indicators include four response alternatives: never, sometimes, often, very often. For the analysis the indicators were, however, recoded into occurrence (no/yes) due to the imprecision of the alternative answers and resulting skewness of data distributions (*i.e.*, the distance between "sometimes" and "often" is not necessarily the same as between "often" and "very often").

Linear regression models were used to explore predictors of Internet related problems. For the first analysis, occurrence of the five problem indicators was summed. The constructed dependent variable was skewed and therefore transformed to logarithmic form for the analysis. Following this, a similar regression model using "time on Internet", also in logarithmic form due to skewness, was carried through. Both regression models were tested for collinearity, with satisfactory results (see Tables).

3. Results

3.1. Sample Characteristics

In Table 1, background variables for the study group are presented. Half the group was female and a majority (64.8%) lived in urban settings. The oldest respondent was born in 1917 and the youngest in 1996, with a median and mean age of 45. Approximately, twenty-five percent held a university degree while approximately 20% reported only obtaining elementary school education. A majority of respondents (64.1%) were working.

Int. J. Environ. Res. Public Health **2011**, *8*

Table 1. Respondents.

Gender male/female	50.4%/49.6%
Age	Mean = 45 Md = 45
Urban/rural residence	64.8%/35.2%
Educational level	22.3% Elementary school exam only 55.5% Secondary level 27.2% University degree
Occupation	64.1% Work 12.0% Students 13.1% Retired 10.8% Unemployed, sick- or parental leave

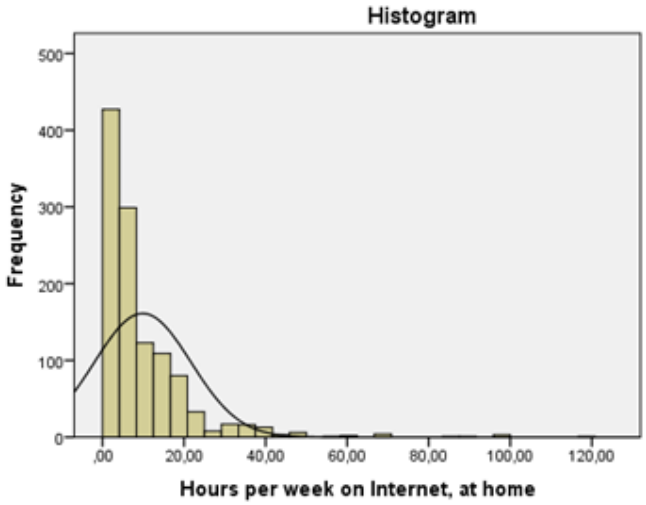
Among Internet users, the mean time length on Internet, at home, was 9.8 hours per week (median= 7 hours; see Table 2). The distribution is heavily skewed (see Figure 1). One out of three used the Internet for gaming while it was quite unusual to use Internet for gambling (11.4%). Internet was also used for many other kinds of activities. None of these other activities that were addressed (*i.e.*, instant messaging, chatting, unspecified Internet surfing, surfing sex sites, posting photos) correlated significantly with the problem indicators used for analyses when introduced in regression models and they were not included in further analyses. Approximately 75% considered themselves as having good or even very good skills in computer use and 45% reported that it was easy or very easy to solve computer related problems and claimed to know how to install a new operating system in a computer.

Table 2. Internet use and computer skills among Internet users.

Hours spent on Internet at home a regular week	9.8 (mean value) 7.0 (median value)
Hours spent on Internet (all locations) a regular week (includes school/work)	15.6 (mean value)
Internet gaming; ever	33.7%
Hours spent on gaming a regular week among regular Internet gamers	6.1 (mean value)
Internet gambling; ever	11.4%
Instant messaging; ever	48.5%
Internet chatting; ever	16.3%
Internet surfing (unspecified); ever	75.2%
Internet sex sites; ever	18.3%
Posting photos on Internet; ever	33.8%
Skills in computer use; good or very good	73.3%
Knows how to install a new operating system	45.0%
Member of Internet based community	29.4%

Int. J. Environ. Res. Public Health **2011**, *8*

Figure 1. Hours per week on Internet, at home (Mean = 9.86, Std. Dev = 11.823, N = 1,147).



Almost one out of three respondents was member in at least one community on the Internet. More said (see Table 3) that use of Internet had led to more contact, both with family members and with friends, than the opposite. More respondents endorsed, however, the fact that Internet use had led to less face-to-face contact than the opposite, especially with other family members.

Table 3. Internet and social life among Internet users.

Has the use of Internet led to less/more contact with family members?	5.5% less 28.5% more
Has the use of Internet led to less/more contact with friends?	4.3% less 39.9% more
Have you spent less/more time in face-to-face interaction with your family since you got Internet at home?	27.5% less 3.2% more
Have you spent less/more time in face-to-face interaction with your friends since you started to use Internet?	9.8% less 3.9% more

3.2. Regression Analyses

In the first regression analysis (Table 4), the summed occurrence of problem indicators was tested against background variables and Internet related variables. Among the background variables only (young) age resulted in a significant prediction. The most powerful predictor was prevalence of membership in online communities, followed by time on Internet and prevalence of gaming. Also, prevalence of gambling predicted more problems. More skills in computer handling were also significant predictors, as were spending less time face-to-face with family and friends since the Internet was introduced in the home, while degree of contact with friends (or family) did not predict problems.

Int. J. Environ. Res. Public Health **2011**, *8*

Table 4. Linear regression analysis; (ln) summed occurrence of problem indicators.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Collinearity Statistics	
	B	Std. Error	Beta			Tolerance	VIF
(Constant)	−4.851	2.259		−2.147	0.032		
Woman	0.010	0.030	0.009	0.330	0.741	0.785	1.274
Year of birth (young age)	0.003	0.001	0.080	2.345	0.019	0.554	1.806
Educational level	0.010	0.011	0.025	0.918	0.359	0.889	1.125
Rural living	−0.008	0.029	−0.007	−0.268	0.788	0.945	1.058
No engagement (no work/stud.)	0.030	0.037	0.024	0.789	0.431	0.717	1.394
Internet gaming	0.056	0.012	0.135	4.724	0.000	0.784	1.276
Internet gambling	0.077	0.022	0.095	3.544	0.000	0.899	1.112
Degree of skills related to computer use	0.069	0.023	0.093	3.051	0.002	0.687	1.456
Knows how to install operative systems	0.109	0.034	0.103	3.243	0.001	0.637	1.571
Member in community/-ies	0.199	0.035	0.172	5.703	0.000	0.704	1.421
Less or more FTF with friends	−0.154	0.040	−0.106	−3.819	0.000	0.834	1.200
Less or more FTF with family	−0.065	0.030	−0.061	−2.176	0.030	0.822	1.216
Less or more contact with family	−0.043	0.025	−0.065	−1.763	0.078	0.470	2.126
Less or more contact with friends	0.014	0.023	0.023	0.598	0.550	0.446	2.240
Hours/week on Internet, at home	0.000	0.000	0.149	5.096	0.000	0.750	1.333

Dependent Variable: ln problem indicators in dummy form and summed. Adj. $R^2 = 0.272$.

In the second regression analysis (Table 5), time in hours per week on Internet at home was tested. Young age and unemployment (no work/studies) predicted more time on Internet, which might be as expected. Gaming constituted the most powerful predictor, but skills with computers and membership in Internet based communities also predicted more time spent on the Internet. Stating that the Internet has led to more contact with friends predicted more time on Internet while there was no significant prediction value in changes of face-to-face contacts.

Int. J. Environ. Res. Public Health **2011**, *8*

Table 5. Linear regression analysis; (ln) summed hours per week on Internet, at home.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Collinearity Statistics	
	B	Std. Error	Beta			Tolerance	VIF
(Constant)	−12.650	5.282		−2.395	0.017		
Woman	−0.115	0.071	−0.046	−1.620	0.106	0.785	1.273
Year of birth (young age)	0.008	0.003	0.100	2.998	0.003	0.558	1.792
Educational level	0.005	0.026	0.005	0.179	0.858	0.890	1.124
Rural living	−0.104	0.067	−0.039	−1.542	0.123	0.948	1.055
No engagement (no work/stud.)	0.351	0.087	0.117	4.021	0.000	0.728	1.373
Internet gaming	0.189	0.027	0.192	7.046	0.000	0.838	1.193
Internet gambling	0.057	0.051	0.029	1.123	0.262	0.901	1.109
Degree of skills related to computer use	0.242	0.053	0.138	4.615	0.000	0.697	1.434
Knows how to install operative systems	0.413	0.078	0.163	5.265	0.000	0.649	1.542
Member in community/-ies	0.235	0.082	0.085	2.864	0.004	0.706	1.417
Less or more FTF with friends	0.110	0.095	0.031	1.156	0.248	0.834	1.199
Less or more FTF with family	−0.005	0.070	−0.002	−0.075	0.940	0.823	1.215
Less or more contact with family	0.093	0.058	0.058	1.609	0.108	0.472	2.117
Less or more contact with friends	0.282	0.054	0.194	5.227	0.000	0.450	2.223

Dependent Variable: ln hours per week on Internet, at home, Adj. $R^2 = 0.296$.

4. Conclusions and Discussion

Much of the previous research in the field of Internet and other behavioral addictions is based on selected samples; in the case of Internet addiction, the research has been biased towards high frequency users of Internet. In the present study this is not the case. As could be expected when using more representative data, we find in our data a more balanced distribution of positive and negative experiences related to Internet use and fewer reports of problems related to Internet use.

The recent development within addiction studies to consider the inclusion of non-substance or behavioural addictions—e.g., Internet addiction—as formal diagnosis in a nomenclature such as the DSM 5 is a clear sign of an ongoing transformation of the conceptual framework for the underlying phenomena. Although Petry (p. 142, [27]) might be correct in her claims that behavioural addictions "share many features" with addictions related to the consumption of substances such as heroin or cocaine, it is nevertheless obvious that there is also a distinct difference between a substance addiction

and a behavioural addiction, in the sense that the former category has been identified as addiction due to specific characteristics of a limited set of substances. In the beginning of the 1970's Smith and Gay [28] published a book with the title "It's so good do not even try it once—Heroin in perspective". The price for the expansion of addiction to encompass various other non-substance-related behaviors is a considerable switch, possibly undermining or at least shifting attention away from the presumption that certain substances in and of themselves are capable of enslaving those who consume them due to inherent qualities of the substances.

The distribution of hours per week on Internet at home in our study (Figure 1) demonstrates similarity with the well-known distribution of alcohol consumption, *i.e.*, the distribution is positively skewed with the tail consisting of individuals that have an extensive Internet involvement. This observation underlines that many of the studies that report alarming prevalence rates of extensive Internet involvement/addiction (see e.g., [16,17]) are likely to be huge overestimations of what proportions of Internet users that might considered to exhibit problematic use. It seems quite clear that this type of overestimation in the general case is a consequence of the fact that many studies has relied on convenience samples that are self-selected through advertisements on the Internet. While the respondents identified as "dependent" (80 percent of the total) in Young's [16,17] study spent an average of 38.5 hours per week on the Internet "for pleasure", the mean value for similar activity in the sample presented in this study was 10.7 hours per week and only 5 percent spent more than 30 hours per week online.

As pointed out in the foregoing, it is of vital importance to reiterate that "Internet use" is a highly abstract and non-specific [23] category, and that it is by no means self-evident what functions different activities, made possible through the Internet, have for different types of individuals. Shen and Williams [6] identify two basic perspectives on the effects of Internet use. On the one hand there is a "compensation model" in which it is postulated that Internet use provides social options that are most beneficial for individuals who are less socially competent in "real life". On the other hand there is also a model which suggests a polarization between resourceful and more disadvantaged individuals, *i.e.*, a model proposing that the "rich get richer" while at the same time the "poor get poorer." Although the data in the present study does not allow for a more detailed analysis of these models, or other models, we can at least find some support for the presence of both positive and negative social effects. Regarding positive effects, a substantial number of individuals have had more social contacts with both family members as well as friends due to their use of Internet. Regarding negative effects, there is a decline in face-to-face contacts with both of these groups. However, taken together, there is a net sum increase in social contacts of 28% (given that we consider it possible to group the both categories of contacts together).

About 50% of the respondents have indicated some experience of at least one problem related to their use of the Internet, but only 2.5% have marked the presence of all five problem items. In the regression analysis based on the summed occurrence of problem indicators there are three significant predictors that, besides weekly hours on Internet, relate to "time". Gaming and membership in online communities are both time consuming activities. The significant effect of gambling, despite its relative low prevalence in the sample, points in the direction of a different type of relation to the dependent variable than those of gaming and membership in online communities, *i.e.*, gambling is closer to being a problem in itself.

Int. J. Environ. Res. Public Health **2011**, *8*

In possibly the most well developed field of addiction studies, that of alcohol addiction, the main general limitation in the "epidemiological approach"—that we have relied on in this study—is known to be the relative difficulty of reaching individuals with a high level of problems through the distribution of a survey. It is reasonable to expect that a similar self-selection effect is present in studies on problems related to Internet use, but it is not self-evident that the magnitude of such a problem will be the same as in alcohol studies. It is general knowledge that the social stigma associated with alcohol addiction is not present in the same way for individuals with problems related to extensive Internet use. Hence, they could be expected to be more inclined to participate in surveys (compared to individuals with alcohol problems). At the same time the classical divergence between "treatment seeking populations and larger realities" in the alcohol field [29] is not present when it comes to problems related to Internet use. This is due to the fact that a majority of the selected samples that have been used to illuminate problematic Internet use does not consist of treatment seeking individuals. Instead recruitment is often done through web-searches related to Internet use. Even though the epidemiological approach is associated with some methodological problems, it nevertheless seems to be worthwhile to continue to study the distribution of internet use and problems in the general population, and thereby provide a wider perspective on problems related to Internet use.

A fundamental component of most perspectives on the addiction phenomena is that addiction is persistent, *i.e.*, it is characterised by the fact that it has a strong tendency to not go away. This aspect of the addiction phenomenon cannot be addressed with reference to the empirical material presented in the present paper. However in the study by Van Roij *et al*. [20], a design with repeated cross-sectional surveys opened up for the identification of longitudinal cohort of online adolescent gamers (n = 467). Although the time frame for this group covered just one year (from 2008 to 2009) only half of the respondents that were classified as addicted in 2008 could be identified in the same category in 2009. This finding implies that extensive online gaming may be characterized as transitory rather than persistent, and hence does not have a clear connection to one of the most central characteristics of a traditional understanding of addiction. On the other hand, other addictions do tend to be more transitory among adolescents than adults. Thus, that study may not be applicable to the current sample, or to addiction phenomena among adults.

One limitation in the present study is that we attempt to contrast our results with Young's although we do not have data for a "full" replication of the Young study. In the survey we used for analyses only five of the eight indicators used by Young were included. We would of course have preferred to have employed all eight indicators to work with. Due to survey space limitations, this was not possible. Another limitation is embedded in the cross-sectional design of the survey. Hence, we do not know how users of Internet develop extensive preferences or problems. Nor do we know the time order of predictors and dependent variables. There is much to be done in future studies using general population samples.

Acknowledgments

We gratefully acknowledge comments and suggestions of two anonymous reviewers and the guest editor Steve Sussman. The study was conducted with funding from FAS—the Swedish Council for Working Life and Social Research.

Int. J. Environ. Res. Public Health **2011**, *8*

Conflict of Interest

The authors declare no conflict of interest.

References

1. Bergmark, K.H.; Bergmark, A. The diffusion of addiction to the field of MMORPGs. *Nord. Stud. Alcohol Drug.* **2009**, *26*, 415-426.
2. Alexander, B. The globalization of addiction. *Addict. Res*. **2000**, *6*, 501-526.
3. Eklund, L.; Bergmark, K.H. *Ungdomars Medieanvänding och Föräldrakontroll (Youth's Media Use and Parental Control)*; Swedish Media Council: Stockholm, Sweden, 2010. Available online: http://www.medierådet.se/Kunskapsbanken/Ovrigt/Foraldrars-kontroll-av-mediekonsumtion (accessed on 28 November 2011).
4. Nordicom-Sveriges. *Internetbarpmeter 2009*; Goteborgs Universitet: Sverige, Sweden, 2009. Available online: www.nordicom.gu.se/common/publ_pdf/323_internetbarometer2009.pdf (accessed on 28 November 2011).
5. Findahl, O. *Svenskarna och Internet 2010 (Swedes and Internet 2010)*; World Internet Institute: Gavle, Sweden, 2010.
6. Shen, C.; Williams, D. Unpacking time online: Connecting Internet and massively multiplayer online game use with psychosocial well-being. *Comm. Res.* **2010**, *38*,123-149.
7. Wong, C.Y.; Mill, J.; Fernandes, C. Drugs and addiction: An introduction to epigenetics. *Addiction* **2011**, *106*, 480-489.
8. Grant, J.E.; Potenza, M.N.; Weinstein, A.; Gorelick, D.A. Introduction to behavioral addictions. *Am. J. Drug Alcohol Abuse* **2010**, *36*, 233-241.
9. Gilbert, R.L.; Murphy, N.A.; McNally, T. Addiction to the 3-dimensional Internet: Estimated prevalence and relationship to real world addictions. *Addict. Res. Theory* **2011**, *19*, 380-390.
10. Weinstein, A.; Lejoyeux, M. Internet addiction or excessive Internet use. *Am. J. Drug Alcohol Abuse* **2010**, *36*, 277-283.
11. Kuss, D.J.; Griffiths, M.D. Online social networking and addiction—A review of the psychological literature. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3528-3552.
12. Gearhardt, A.N.; Grilo, C.M.; DiLeone, R.J.; Brownell, K.D.; Potenza, M.N. Can food be addictive? Public health and policy implications. *Addiction* **2011**, *106*, 1208-1212.
13. Adams, J.; Kirkby, R.J. Excessive exercise as an addiction: A review. *Addict. Res. Theory* **2002**, *10*, 415-437.
14. Freimuth, M.; Moniz, S.; Kim, S.R. Clarifying excercise addiction: Differential diagnosis, co-occurring disorders, and phases of addiction. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4069-4081.
15. Lorains, F.K.; Cowlishaw, S.; Thomas, S.A. Prevalence of comorbid disorders in problem and pathological gambling: Systematic review and meta-analysis of population surveys. *Addiction* **2011**, *106*, 490-498.
16. Young, K. Internet addiction: The emergence of a new clinical disorder. *Cyberpsychol. Behav.* **1996**, *1*, 237-244.

17. Young, K. *Caught in the Net: How to Recognize the Signs of Internet Addiction and a Winning Strategy for Recovery*; John Wiley & Sons, Inc.: New York, NY, USA, 1996.
18. Young, K. Understanding online gaming addiction and treatment issues for adolescents. *Am. J. Fam. Ther.* **2009**, *37*, 355-372.
19. Griffiths, M.D. Online therapy for addictive behaviors. *Cyberpsychol. Behav.* **2005**, *8*, 555-561.
20. van Rooij, A.J.; Schoenmakers, T.M.; Vermulst, A.A.; van der Eijnden, R.J.J.M.; van de Mheen, D. Online video game addiction: Identification of addicted adolescent gamers. *Addiction* **2010**, *106*, 205-212.
21. Shapira, N.; Lessig, M.; Goldsmith, T.; Szabo, S.; Lazoritz, M.; Gold, M.; Stein, D. Problematic Internet use: Proposed classification and diagnostic criteria. *Depress. Anxiety* **2003**, *17*, 207-216.
22. O'Brien, C. Addiction and dependence in DSM-V. *Addiction* **2011**, *106*, 866-867.
23. Sussman, S.; Leventhal, A.; Bluthenthal, R.; Freimuth, M.; Forster, M.; Ames, D. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
24. Byun, S.; Ruffini, C.; Mills, J.E.; Douglas, A.C.; Niang, M.; Stepchenkova, S.; Lee, S.K.; Loufti, J.; Lee, J.-K.; Atallah, M.; Blanton, M. Internet addiction: Metasynthesis of 1996–2006 quantitative research. *Cyberpsychol. Behav.* **2009**, *12*, 203-207.
25. Findahl, O. *Svenskarna och Internet 2009 (Swedes and Internet 2009)*; World Internet Institute: Gavle, Sweden, 2009.
26. Findahl, O. *Vad Säger Internetstatistiken? En Vägledning för Analys och Tolkning av Nationell och Internationell Statistik (What says the Internet Statistics? A Guide for Analysis and Interpretation of National and International Statistics)*; Stiftelsen för Internetinfrastruktur: Stockholm, Sweden, 2008.
27. Petry, N. Should the scope of addictive behaviours be broadened to include pathological gambling. *Addiction* **2006**, *101*, 152-160.
28. Smith, D.; Gay, G. *It's so Good do not Even Try it Once—Heroin in Perspective*; Prentice-Hall: Eaglewoods Cliffs, NJ, USA, 1972.
29. Room, R. Treatment seeking populations and larger realities. In *Alcoholism Treatment in Transition*; Edwards, G., Grant, M., Eds.; Croom Helm: London, UK, 1980; pp. 205-224.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Sun, P.; Johnson, C.A.; Palmer, P.; Arpawong, T.E.; Unger, J.B.; Xie, B.; Rohrbach, L.A.; Spruijt-Metz, D.; Sussman, S. Concurrent and Predictive Relationships Between Compulsive Internet Use and Substance Use: Findings from Vocational High School Students in China and the USA. *Int. J. Environ. Res. Public Health* **2012**, *9*, 660-673; doi:10.3390/ijerph9030660.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Concurrent and Predictive Relationships Between Compulsive Internet Use and Substance Use: Findings from Vocational High School Students in China and the USA

Ping Sun [1,*], Carl Anderson Johnson [2], Paula Palmer [2], Thalida E. Arpawong [1], Jennifer B. Unger [1], Bin Xie [2], Louise A. Rohrbach [1], Donna Spruijt-Metz [1] and Steve Sussman [1]

[1] Institute for Health Promotion and Disease Prevention Research, Department of Preventive Medicine, Keck School of Medicine, University of Southern California, 2001 N. Soto Street, Los Angeles, CA 90032, USA; E-Mails: arpawong@usc.edu (T.E.A.); unger@usc.edu (J.B.U.); rohrbac@usc.edu (L.A.R.); dmetz@usc.edu (D.S.-M.); ssussma@usc.edu (S.S.)

[2] School of Community & Global Health, Claremont Graduate University, 160 East 10th Street, Claremont, CA 91711, USA; E-Mails: andy.johnson@cgu.edu (C.A.J.); paula.palmer@cgu.edu (P.P.); bin.xie@cgu.edu (B.X.)

* Author to whom correspondence should be addressed; E-Mail: sping@usc.edu; Tel.: +1-323-442-8221; Fax: +1-323-442-8201.

Received: 29 December 2011; in revised form: 25 January 2012 / Accepted: 17 February 2012 / Published: 23 February 2012

Abstract: *Purpose:* Compulsive Internet Use (CIU) has increasingly become an area of research among process addictions. Largely based on data from cross-sectional studies, a positive association between CIU and substance use has previously been reported. This study presents gender and country-specific longitudinal findings on the relationships between CIU and substance use. *Methods:* Data were drawn from youth attending non-conventional high schools, recruited into two similarly implemented trials conducted in China and the USA. The Chinese sample included 1,761 students (49% male); the US sample included 1,182 students (57% male) with over half (65%) of the US youth being of Hispanic ethnicity. Path analyses were applied to detect the concurrent and predictive relationships between baseline and one-year follow-up measures of CIU level, 30-day cigarette smoking, and 30-day binge drinking. *Results:* (1) CIU was not positively related with substance use at baseline. (2) There was a positive predictive relationship between baseline CIU and change in substance use among female, but not male students.

Int. J. Environ. Res. Public Health **2012**, *9*

(3) Relationships between concurrent changes in CIU and substance use were also found among female, but not male students. (4) Baseline substance use did not predict an increase in CIU from baseline to 1-year follow-up. *Conclusions:* While CIU was found to be related to substance use, the relationship was not consistently positive. More longitudinal studies with better measures for Internet Addiction are needed to ascertain the detailed relationship between Internet addiction and substance use.

Keywords: compulsive internet use; internet addiction; youth; cigarette smoking; binge drinking; addiction syndrome; addiction specificity

1. Introduction

Although it is still debatable whether Internet Addiction (IA) should be labeled as an addictive disorder [1–4], the detrimental effects of IA have been widely observed and documented [3,5,6]. Furthermore, while IA may not be as widely recognized as an addictive behavior in the United States (US), the harmful effect of IA has been recently demonstrated in China and other Asian countries [7]. It has been estimated that 14.1% of adolescents—roughly 20 million people—met the diagnostic criteria for IA in China, and the prevalence of IA is highest among the 18–23 year old age group [8]. National laws are being implemented in China with the aim of reducing IA among youth, especially through uncontrolled use in cyber cafés [3]. To effectively help those who are in need of assistance, evidence based school-level or student-level IA intervention methods are being requested by parents and health educators [9].

In order to develop evidence-based programs aimed at controlling IA, it is essential to fully understand its etiological pathway and patterns of psychiatric and behavioral co-morbidities. IA has been found to be a co-morbidity with psychiatric symptoms [10,11] and other risk factors [12] of substance use, or substance use dependencies [13]. Research suggests that addiction to internet use is similar to addiction to other compulsive behaviors such as substance use, binge eating [14] (page 479), compulsive gambling [15], excessive shopping [16], and compulsive sex behavior [17]; they are all symptoms of maladaptive process addictions. A syndrome hypothesis of addiction [18] posits that addiction may not be inextricably specific to a particular substance or behavior. For example, various addictions are found to co-exist [19] with addicts often alternating between substances [20]. Furthermore, it is common that addicts may switch from substance use to a compulsive behavior [21]. Thus, with respect to uncovering etiological patterns of IA as a current and lifetime co-morbidity, it will be important to examine the complete concurrent and longitudinal relationships between IA and substance use.

Currently, available data that relates IA with other addictive behaviors (substance abuse) is very limited [22]. Analyses of cross-sectional data have revealed that IA was positively related to substance use among youth [23–27]. IA was also positively correlated with certain risk factors of substance use behavior (e.g., intention to use drugs, pro-drug use attitudes, perceived social norm of drug use) [26]. However, there is a dearth of longitudinal studies that contribute to understanding not only the

cross-sectional concurrence between IA and substance use, but also the other hypothesized relationships over time.

Research focusing on IA has been much more prominent in Asian countries [3], which may reflect the fact that consequences of IA are perceived as more harmful in Asian countries than in the western countries [3]. Results of one study suggest that there are differences in IA by race [28]; thus, it will be important to compare findings from similar studies conducted in both Eastern and Western countries.

In general, hypotheses regarding the comprehensive nature of the relationships between IA and substance use may include the following: (1) IA is a causal risk factor for substance use; (2) substance use is a causal risk factor for IA; (3) IA and substance use reinforce each other and thus have a reciprocal relationship; and (4) concurrent relationships exist between measures of IA and substance use. If there are no relationships found to support hypotheses 1, 2, or 3; but there is a relationship that supports hypothesis 4, there would be no true causal relationship between IA and substance use; rather the observed concurrent relationships between the cross-sectional measures, or between parallel changes during the same time span could be spurious (*i.e.*, caused by a third factor).

There is currently a lack of universal diagnostic criteria for IA. Viewing IA as an impulse-control disorder that does not involve an intoxicant, researchers consider IA as a multi-dimensional disorder [7,29,30]. Together with other aspects of IA, compulsive internet use (CIU) is a commonly accepted dimension of IA. The relationships between CIU, cigarette smoking, and binge drinking are the focus of this study.

Objectives of this study are uniquely comprehensive in that we will examine all four hypothesized relationships between CIU and substance use using longitudinal data. Namely, we will depict the possible concurrent and reciprocal predictive relationships between CIU, cigarette smoking, and binge drinking behaviors among high-risk youth. The concurrent relationships investigated will include both the cross-sectional (baseline with baseline) and longitudinal ones (e.g., how the change in CIU from baseline to follow-up correlate with the change in cigarette smoking from baseline to follow-up). All change scores will be calculated as the value at 1-year follow-up survey minus the value at baseline. All possible bidirectional relationships will be investigated such that baseline behaviors will be used to predict change in behavior from baseline to one-year follow-up. All analyses will be conducted separately in US and Chinese samples, and separately for each gender. Therefore, analyses will also be able to distinguish the possible differences in the hypothesized relationships between the two countries, and by gender.

2. Methods: Study Design and Sample Description

Data for the analyses were drawn from two similar school-based longitudinal trials, one conducted in Chengdu, China and the other in Los Angeles, California, USA. The study in China, the "Healthy Lives, Successful Lives" (HLSL) trial, was a two-condition controlled intervention conducted to test the impact of a 10 lesson (45 minutes each) intervention on tobacco use and alcohol use among high school students over two years. The US study, a "Towards No Drug Abuse" (TND) trial, was a three-condition controlled intervention implemented to test the impact of TND programming *vs.* "a standard care control program" over three years. Each study employed a design where program interventions were applied between a pre-intervention baseline measure and several post-intervention

follow-up measures. Data for these analyses were drawn from measures collected at two time points: baseline and one-year follow-up. The US study included 1,182 students recruited from 24 continuation high schools (CHS) in California. The China study included 1,761 10th grade students from 12 vocational high schools (VHS). Both types of schools—CHS in California and VHS in China—are high schools that differ from regular high schools because of their student enrollment methods. In California, students who are unable to remain in the regular high school system due to functional reasons (e.g., did not fulfill credit requirement for graduation, consistent use of substances, truancy) are transferred to CHS. In China, students who are unable to perform academically on the high school admittance test or are enduring household economic challenges typically end up in VHS. The details of the study design and data collection procedures for these two studies were introduced elsewhere [31,32]. All study procedures, including informed consent, for both studies were approved by the University of Southern California's Institutional Review Board (IRB). Procedures for the China study gained additional approvals from the local IRB in China.

The average age (years, mean ± std) was 16.8 ± 0.93 and 15.9 ± 0.76 for the students in the TND, and HLSL study, respectively. The TND study included slightly more male students than the HLSL study (56.6% *vs.* 49.1%). In TND, more than half of the students were Hispanic (64.9%), with 11.7% being Non-Hispanic White, 4.3% African-American, 13.0% Mixed ethnicity, and 6.1% other Ethnicity. In the HLSL study, almost all youth were of Han ethnicity.

3. Measures

This paper focuses on the concurrent and predictive relationships between CIU, cigarette smoking, and binge drinking. The key analytical variables included CIU, 30-day cigarette use, 30-day binge drinking, and other covariates that are adjusted for in the analyses.

Various methods have been used to assess problematic internet use [7,29,33]. Our measure of CIU was based on the Davis Scale for problematic internet use [30,33]. In this scale, Davis proposed four dimensions with which to assess consequential CIU: diminished impulse control, loneliness/depression, social conformity, and distraction. Limited by the number of items that could be placed on the surveys, we chose to focus on assessing diminished impulse control with the four items that may be best suited for revealing shared pathways with other addictive behaviors (e.g., substance use [34,35]); we labeled the measure "Compulsive Internet Use" (CIU). The four items comprising CIU were "I use the Internet more than I ought to", "I usually stay on the Internet longer than I had planned", "Even though there are times when I would like to, I can't cut down on my use of the Internet", and "My use of the Internet sometimes seems beyond my control." Response options were provided on an ordinal scale, including (1) Strongly disagree, (2) Disagree, (3) Neutral, (4) Agree, and (5) Strongly agree. The mean of all four items was used as a continuous measure of CIU. The CIU scale showed a high inter-item consistency for the four items with a Cronbach alpha of 0.84 and 0.80 among the Chinese and US sample, respectively. The test-re-test correlation for CIU between baseline and 2-month follow-up was 0.48 for Chinese and 0.61 for US youth; and between baseline and 1-year follow-up, it was 0.39 for Chinese and 0.48 for US youth.

Cigarette smoking and binge drinking in last 30 days were assessed slightly differently in TND and HLSL studies. In TND, cigarette smoking was assessed by the question, "How many times in the last

month have you used cigarettes?", with a 12-point scale response option, starting at "0 times", increasing in intervals of 10 (e.g., "1–10 times", "11–20 times") with the last (12th) category being "over 100 times"; binge drinking was assessed by the question, "How many days have you had 5 or more alcoholic drinks within a 5 hour period over the last 30 days?" with a similar 12-point scale response option. In the HLSL study, cigarette smoking was assessed by the question "During the last 30 days, on how many days did you smoke cigarettes?", with a 7-level answer option ranging from 1: "0 day", 2: "1 or 2 days", …, 6: "20–29 days", to 7: "all 30 days". The binge drinking behavior was assessed by asking the participants "During the past 30 days, on how many days did you have 5 or more drinks of alcohol in a row, that is, within a couple of hours?" with the 7-level answer options ranging from 1: "0 day", 2: "1 day",…, to 7: "20 or more days". Dichotomous transformation of the substance use measures were applied such that any use on one or more days is coded as "Yes" for use (*i.e.*, presence of cigarette smoking or binge drinking in past 30 days).

To assess any differential relationship across gender, analyses were conducted separately for boys and girls. Analyses were adjusted for potential demographic and environmental confounders. The covariates for the Chinese sample included age, students' birthplace (urban *vs.* rural or suburban), parents' education (highest among both parents with seven levels ranging from 1: 'lower than elementary school' to 7: '4-year or higher'), allowance (a 10-level answer for weekly amount, ranging from 1: 'none', to 9: 'more than 90 Yuan'), self-reported academic performance (5-levels, ranging from 1: 'F or below', to 5: 'A or above'), a dummy variable indicating intervention status (program *vs.* control), and a propensity score for attrition. Covariates for the US sample included age, ethnicity, a dummy variable indicating intervention status (program *vs.* control), and a propensity score for attrition. The purpose for including a propensity score for attrition as a covariate is to adjust for the potential bias introduced by non-random attrition at follow-up [36]. The propensity score was generated by using logistic regression analysis, such that selected baseline measures were used to predict the dichotomous attrition status at one-year follow-up. A continuous propensity score for attrition was then calculated from the estimated formula.

4. Statistical Modeling

Due to the multi-level sampling method employed in both studies (targeted school, and students within the targeted schools), multi-level random coefficients modeling was applied to the analysis, in which a school-level random effect was assumed in the model. Structural equation modeling was employed to assess all concurrent and predictive relationships in one SEM model. Mplus [37] was used to construct the SEM modeling, which estimates the independent relationships among the three behaviors targeted. All estimates from the model could then be viewed as the relationships between the residuals of the variance, after adjusting for all other relationships in the pathway as well as the contributions from covariates. Standardized coefficients for the relationships (partial correlations) were reported for the path diagrams. Additional analyses were also conducted to examine if the relationships were moderated by intervention status, such that interaction terms were computed and modeled in the analyses.

Int. J. Environ. Res. Public Health **2012**, *9*

5. Results

The mean (±std) for CIU was 2.27 (±0.92), 2.71 (±1.02), 2.45 (±0.96), and 2.32 (±0.93) among the Chinese female, Chinese male, US female, and US male students, respectively. While this continuous measure is not a suitable estimate of CIU prevalence, if assuming that an average score of 4 ("Agree" or "Strongly agree" with the CIU statements) is the cut-off for determining the binary status of having CIU problem, the prevalence of CIU would be 5.8%, 15.7%, 9.7%, and 7.3% among the Chinese female, Chinese male, US female, and US male students, respectively. Other studies in China have estimated the prevalence of Internet addiction among Chinese youth to be between 5% and 15% [7,8,38], which is consistent with our estimations for the Chinese youth.

The prevalence of 30-day cigarette smoking was 13.7%, 46.1%, 36.6%, and 46.3% among the Chinese females, Chinese males, US females, and US males, respectively. The prevalence for binge drinking was 3.7%, 10.9%, 34.2%, and 38.8% among the Chinese females, Chinese males, US females, and US males, respectively. Chinese females therefore had the lowest prevalence of both cigarette smoking and binge drinking, while Chinese boys had a lower prevalence of binge drinking compared to both US boys and girls.

The intervention did not generate statistically significant (tailed alpha = 0.05) effects on CIU, and prevalence of cigarette smoking and binge drinking. Additional analysis also failed to detect any statistically significant interaction between the intervention and the focused relationships between the three addictive behaviors. Thus, reported below were the results from the analyses involving subjects in both "program" and "control" conditions, with intervention status adjusted as a covariate.

The concurrent and predictive relationships between CIU, cigarette smoking, and binge drinking are depicted separately for females (Figure 1) and males (Figure 2) since results were found to vary by gender. Two pathway diagrams are included side-by-side in each figure, with the left diagram revealing findings among the US youth, and the right for the Chinese. Chi-square tests for the unrestricted and fitted models indicate a good model fit for all four models (dof = 80, $\chi^2 > 500$ for the models of US females and US males; dof = 75, $\chi^2 > 4{,}000$ for the models of Chinese females and Chinese males). In all figures, only statistically significant ($p < 0.05$) relationships are illustrated, with the curved double-headed arrows depicting concurrent relationships, and straight single-headed arrows depicting predictive relationships. Four major findings emerged from the pattern of relationships.

Figure 1. Female students only: Concurrent and longitudinal predictive relationships among CIU, cigarette smoking, and binge drinking.

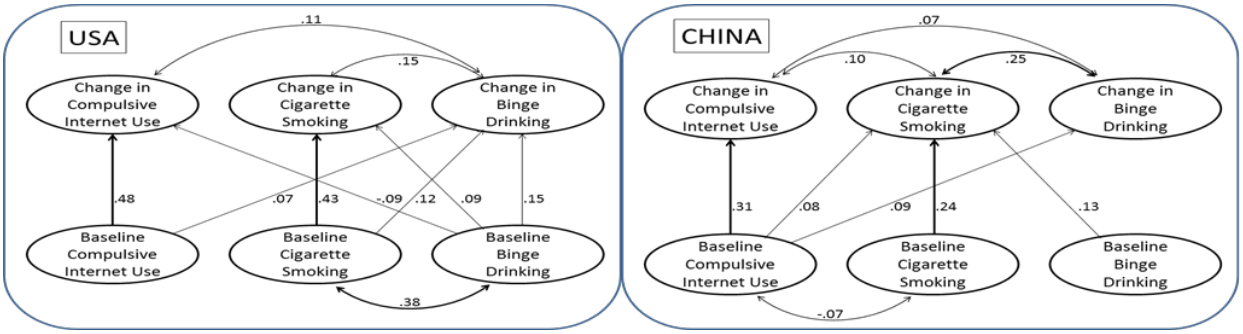
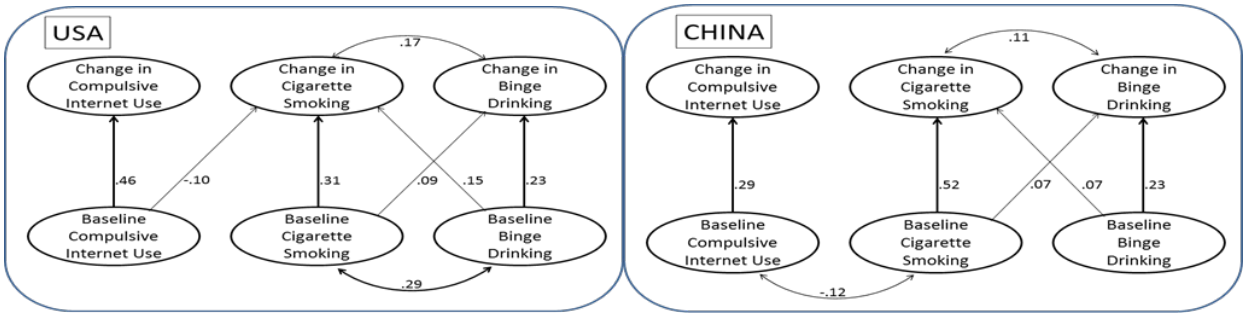


Figure 2. Male students only: Concurrent and longitudinal predictive relationships among CIU, cigarette smoking, and binge drinking.



Notes: (1) Only statistically significant relationships ($p < 0.05$) from SEM modeling are shown. (2) Coefficients in the figure are adjusted correlations. (3) Covariates for modeling in the US sample include age, ethnicity, dummy coding indicating intervention status (two program, and control), and a propensity score for attrition. (4) Covariates for modeling in the Chinese sample include age, students' birthplace (urban *vs.* rural or suburb), parents' education (highest among both parents, 7 levels ranging from 1: 'lower than elementary school' to 7: '4-year or higher'), allowance (a 10-level answer for weekly amount, ranging from 1: 'none', to 9: 'more than 90 Yuan'), self-reported academic performance (5-levels, ranging from 1: 'F or below', to 5: 'A or above), a dummy variable indicating intervention status (program *vs.* control), and a propensity score for attrition.

First, data from both studies revealed the lack of positive cross-sectional relationships between CIU and substance use. Differences between US and Chinese youth were found in the relationships between baseline measures of CIU, cigarette use, and binge drinking. Baseline CIU was negatively correlated with baseline cigarette smoking among Chinese female and male youth (r = −0.07 and −0.12, respectively), but not among US female or male youth.

Second, longitudinally, higher baseline CIU predicted elevated substance use in female students, but not in male students. Among females, higher baseline CIU levels resulted in more binge drinking at 1-year follow-up among US students (r = 0.07), and in more cigarette smoking (r = 0.08) and more binge drinking at 1-year follow-up (r = 0.09) among Chinese students. However, among males, the only predictive relationship between CIU and substance use was that among US males, in which higher baseline CIU levels resulted in less cigarette smoking at 1-year follow-up (r = −0.10).

Third, change in CIU was positively related with change in substance use among females, but not males. More specifically, change in CIU was positively related with change in binge drinking among both US (r = 0.11) and Chinese (r = 0.07) female students, and with change in cigarette smoking among Chinese female students (r = 0.10) only. However, none of the relationships between concurrent changes were statistically significant among male students.

Fourth, baseline substance use did not predict the increase in CIU over 1 year. Regarding cigarette use and binge drinking among female and male US and Chinese students, the only statistical finding was that baseline binge drinking predicted a reduction in CIU over 1 year among the US female students (r = −0.09).

Overall, a complete pattern of positive relationships emerged between cigarette smoking and binge drinking among US youth. The patterns are referred to as complete because there were significant

relationships between all possible pathways. Concurrently, baseline cigarette smoking and binge drinking were found to be positively correlated (r = 0.38 in US females, and r = 0.29 in US males), as were their change scores a year later (r = 0.15 in US females, and r = 0.17 in US males). Predictively, baseline cigarette smoking predicted change in binge drinking (r = 0.12 and 0.09 in US female and males, respectively); while baseline binge drinking also predicted change in cigarette smoking (r = 0.09 and 0.15 in US female and males, respectively). In comparison, the pattern of relationships between cigarette smoking and binge drinking were less complete among Chinese youth, and they were the least complete between CIU and any of the substance use measures.

6. Discussion

This study documents both the concurrent and predictive longitudinal relationships among CIU, cigarette smoking, and binge drinking among non-traditional high school students in the US and China. Viewing CIU as a process addiction behavior, and cigarette smoking and binge drinking as substance use addiction behaviors, the findings help document the relationships between a process addiction and two substance use addictions, with the relationships between the two types of substance use addiction serving as a reference for comparison.

Five major findings can be summarized from the data: (1) CIU was not positively related with substance use at baseline. (2) There was a positive predictive relationship between baseline CIU and change in substance use among female, but not male students. (3) Relationships between concurrent changes in CIU and substance use were also found among female, but not male students. (4) Baseline substance use did not predict an increase in CIU from baseline to 1-year follow-up. (5) A complete reciprocally predictive relationship was found among US females and males for the relationship between cigarette smoking and binge drinking; but no reciprocally predictive relationship was found between CIU and either type of substance use among either US or Chinese youth.

While the relationships are complex, these unique findings can be applied to test theoretical models of addiction. For example, a Syndrome Model of Addiction suggests that substance addiction and process addiction are all specific expressions of underlying shared manifestations and sequelae for a general addiction syndrome [18]. Guided by the syndrome hypothesis, different manifestations (addiction types) of an underlying addiction syndrome may be correlated, and the change in one addiction manifestation should co-vary (concurrent or substitute manifestations) with the change in another manifestation. Our findings regarding cigarette smoking and binge drinking among US youth shows a strong pattern of concurrent manifestations of addiction, and is thus consistent with the syndrome model. However, due to the overall pattern of results showing the lack of any cross-sectional, predictive, and concurrent relationships between CIU and substance use among male students, our data offers evidence that is inconsistent with the Syndrome Model of Addiction between CIU and substance use among male students from either the US or China. The relationship between CIU and cigarette smoking among Chinese females may indicate a certain level of substitute addiction occurring between CIU and cigarette use. Chinese females with a higher "general" addiction level may vacillate between CIU or cigarette smoking, thereby resulting in a small negative correlation between the two behaviors at baseline. However, because of the "general" addiction tendency, females with higher CIU levels at baseline may also have a higher likelihood of smoking at one-year follow-up.

The "PACE" model for addiction hypothesizes that Pragmatics, Attraction, Expectations, and Communication are the etiologic elements that explain a person's addiction to an array of addiction behaviors or to a specific addictive behavior [39]. While consistent with the syndrome model of addiction in that shared factors may cause an array of addictions, the PACE model emphasizes the fact that people with addiction syndrome do not randomly choose one or an array of addictions, or alternate from one to another. While there are shared biological and environmental factors behind the concurrent manifestations of addiction, the specific expressions of addiction may be studied in the framework of the PACE model involving pathways that link various PACE elements [39]. Guided by the PACE model, the lack of overall correlation between CIU and substance use (in reference to the comprehensive correlations between smoking and binge drinking) may indicate the need to study the specific risk factors and pathways for CIU and use of other substances. For example, the availability of supply and acquisition skills (Pragmatics) may be different; the appetitive effects (Attraction) may also be different; taken together with the synergy of preferentially socializing with different sub-groups of students (Communication), the Expectations and therefore the actual adoption of internet or substance addiction may be different.

With regard to the positive associations between baseline CIU and change in substance use, and between change in CIU and change in substance use, the positive associations are detected among female, but not male students. Previous studies have reported a trend of positive associations between internet addiction and risk factors for substance use or substance use dependencies [10–13], although none of them conducted analyses separately by gender. Thus, our finding indicates that the association between CIU and substance use may differ by gender. This is consistent with other research that has identified gender as an important factor that moderates the neurobiological, cognitive, and psychosocial mechanisms for both cigarette use [40] and binge drinking [41]. In general teenage males binge drink more frequently than females, and teen binge drinking is associated with a variety of problem prone behaviors [42]. Binge drinking may reflect a greater masculinity-based gender role orientation, related to the externalization of behaviors [43]. Therefore, males who engage in CIU may not be any more likely to binge drink in the future compared to males who do not engage in CIU and are otherwise similar in levels of masculinity. Females who engage in CIU, on the other hand, may be more likely to demonstrate masculine gender roles (e.g., if they are online game players), and hence, be more likely to binge drink in the future compared to other females who are higher in femininity. This and other explanations of CIU as a predictor of prospective binge drinking among females in both countries should be examined in future studies.

In addition, previous studies have revealed a possible difference in types of CIU by gender. Huang *et al.* [32] reported that the most prevalent type of internet use differed for Chinese female and male youth, with females being more likely to use online social networking sites, and males being more likely to use online gaming programs. Future studies would be needed to reveal the mechanisms by which the associations between CIU and substance use differ by gender, as well as examine the types of internet use, and their specific roles in the addictive behaviors of females and males.

One possible explanation for the differences found among US and Chinese youth is that the same behavior may be perceived differently in different cultures, in addition to being different between females and males. For example, binge drinking by youth at a social gathering may not be considered deviant behavior in China, while any youth alcohol consumption is prohibited by laws in the US.

Further, both the prevalence and relationships between cigarette smoking and binge drinking were found to differ between the US and Chinese samples, and these differences may reflect the differential meaning of cigarette smoking and binge drinking across race and gender. The vast differences in smoking prevalence, and reasons for smoking, among male and female youth in China have been previously documented [44]. In addition, internet use and its addiction may also bear different meanings and consequences between US and Chinese youth. For example, internet access in cyber cafés is much more common in China than in the US; therefore, the relationship between CIU and substance use among Chinese youth may be confounded by the physical and social settings for internet use. Thus, our finding supports the notion that the overall cultural context plays an important role in studying the current and lifetime co-morbidity and prevention of addictive behaviors.

Findings from previous cross-sectional studies have revealed that CIU is positively correlated with substance use [23,24], and has similar relationships with some common risk factors for substance use (e.g., hostility, depression [10], family factors [45]). However, in this study, baseline CIU was not related to baseline substance use among US youth, and was negatively correlated with baseline cigarette smoking among Chinese youth. This difference in findings by country may stem from the uniqueness of the samples, study design, or measures used. Nevertheless, our finding is salient because it offers evidence for a substitute model of addiction, such as the syndrome model or "PACE" model for addictions. For example, due to there being a common risk factor profile for an general level of addiction, a person may have a higher tendency to indulge in one or more addictive behaviors in a specific time period and environment; however, the addictive behavior may switch from one to another along with the change in time period or environment. Thus, although CIU may share common risk factors with substance use, CIU as a behavior may not be correlated, or even may be negatively correlated with substance use depending upon the contextual factors.

Results of this study show that prospectively, baseline substance use reduces CIU among US youth when assessed one year later, but no significant relationship exists between baseline substance use and change in CIU among Chinese youth. Reciprocally, baseline CIU increases substance use among Chinese females, yet reduces cigarette smoking among US males. While the data revealed a small prospective effect between CIU and substance use, the data did not suggest clear and substantively significant directionality in the pattern of relationships. One possible explanation for this is that the effects of CIU and the effect on CIU may be closely coupled with the particular type of internet use in which youth frequently engage. An alternative hypothesis proposed for further study is that addiction to particular types of websites may increase substance use, or that particular types of CIU may be promoted by substance use.

This study draws data from two longitudinal trials with both CIU and substance use behaviors assessed simultaneously at baseline and one-year follow-up. Thus, the data offered an unprecedented opportunity to examine the concurrent and predictive relationships between CIU and substance use behaviors, and differences by country and gender.

Nevertheless, the study has inherent weaknesses. The first weakness is that the studies from which the data were drawn were not designed to focus on CIU. The assessment of IA needed to be shortened to focus on the compulsive use dimension, which may not be representative of the full spectrum of IA behavior [7]. Our measure of IA is thus labeled as Compulsive Internet Use (CIU), which was an un-validated sub-scale adapted from a 4-dimensional measure of Problematic Internet Use suggested

by Davis *et al.* [33]. Also, we are not aware of any study that has reported on the validity of the Davis "Problematic Internet Use" scale among high-risk US or Chinese youth. Therefore, unique findings from this report between CIU and substance use may not be generalized to validate or invalidate other findings where IA was assessed differently. The second weakness could be due to the two studies being conducted in US and China, separately. Our aim was to compare the results from data drawn from two countries, but the comparability may be compromised by several major differences in the two studies. First, although youth in both studies were considered "higher risk" high school students enrolled in non-regular high schools, the US students were enrolled in "Continuation High Schools" often due to behavioral problems, while the Chinese students were enrolled in "Vocational High Schools" mainly due to academic or economic reasons. Second, although the CIU items were coordinated to be examined in the same way, the overall study designs and other measures were not exactly the same. Thus, the cross-country comparison of findings may be informative, but we are not able to conclude that the differences in findings are attributable to the differences between the Eastern and Western cultural context. Regarding the study samples, there are both strengths and weaknesses that stem from this study being conducted among high-risk youth. The strengths are due to the samples having a higher prevalence of addictive behaviors, which thereby allows for greater statistical power for the study, and yields results that are directly applicable to the needs of the sub-group of youth who need the most help. Conversely, a weakness is that the conclusions may not be extended to all youth, most of whom attend regular academic schools, and are at lower-risk for substance use.

In summary, the harmful effects of Internet Addiction are starting to be recognized in the media and the research field, yet the scientific understanding of the behavior is still in its infancy. The findings from this study suggest that the etiologic pathway to Internet Addiction may share common aspects with the pathways for substance use, although the unique aspects of Internet Addiction warrant clearer examination in future studies.

Conflict of Interest

The authors declare no conflict of interest.

References

1. Young, K. Internet addiction over the decade: A personal look back. *World Psychiatry* **2010**, *9*, 91.
2. Widyanto, L.; Griffiths, M. 'Internet addiction': A critical review. *Int. J. Mental Health Addict.* **2006**, *4*, 31–51.
3. Block, J.J. Issues for DSM-V: Internet addiction. *Am. J. Psychiatry* **2008**, *165*, 306–307.
4. Suler, J. Computer and cyberspace "addiction". *Int. J. Appl. Psychoanal. Stud.* **2004**, *1*, 359–362.
5. Chou, C.; Condron, L.; Belland, J.C. A Review of the research on Internet addiction. *Educ. Psychol. Rev.* **2005**, *17*, 363–388.
6. Young, K.S. Internet addiction: The emergence of a new clinical disorder. *CyberPsychol. Behav.* **1998**, *1*, 237–244.
7. Tao, R.; Huang, X.; Wang, J.; Zhang, H.; Zhang, Y.; Li, M. Proposed diagnostic criteria for Internet addiction. *Addiction* **2010**, *105*, 556–564.

8. China Youth Internet Association. *Net Addiction among Chinese Youth*; 2009. Available online: http://www.zqwx.youth.cn/web/zuizhong.jsp?id=853 (accessed on 17 February 2012).
9. China Internet-Based Information Center. *Profile of Internet Use among Chinese Youth*; 2006. Available online: http://www.china5000.org.cn/wm/Research/200809/P020080919340560208240.pdf (accessed on 17 Feruary 2012).
10. Yen, J.Y.; Ko, C.H.; Yen, C.F.; Chen, S.H.; Chung, W.L.; Chen, C.C. Psychiatric symptoms in adolescents with Internet addiction: Comparison with substance use. *Psychiatry Clin. Neurosci.* **2008**, *62*, 9–16.
11. Morrison, C.M.; Gore, H. The relationship between excessive Internet use and depression: A questionnaire-based study of 1,319 young people and adults. *Psychopathology* **2010**, *43*, 121–126.
12. Ko, C.H.; Hsiao, S.; Liu, G.C.; Yen, J.Y.; Yang, M.J.; Yen, C.F. The characteristics of decision making, potential to take risks, and personality of college students with Internet addiction. *Psychiatry Res.* **2010**, *175*, 121–125.
13. Frangos, C.C.; Sotiropoulos, I. Problematic Internet use among Greek university students: An ordinal logistic regression with risk factors of negative psychological beliefs, pornographic sites, and online games. *Cyberpsychol. Behav. Soc. Netw.* **2011**, *14*, 51–58.
14. Lowinson, J.H.; Ruiz, P.; Millman, R.B.; Langrod, J.G. *Substance Abuse: A Comprehensive Textbook*, 4th ed.; Lippincott Williams & Wilkins: Philadelphia, PA, USA, 2004.
15. Shaffer, H.J.; Stanton, M.V.; Nelson, S.E. Trends in gambling studies research: Quantifying, categorizing, and describing citations. *J. Gambl. Stud.* **2006**, *22*, 427–442.
16. Dell'Osso, B.; Marazziti, D.; Hollander, E.; Altamura, A.C. Traditional and newer impulse control disorders: A clinical update. *Clin. Neuropsychiatry* **2007**, *4*, 30–38.
17. Storholm, E.D.; Fisher, D.G.; Napper, L.E.; Reynolds, G.L.; Halkitis, P.N. Proposing a tentative cut point for the compulsive sexual behavior inventory. *Arch. Sex. Behav.* **2011**, *40*, 1301–1308.
18. Shaffer, H.J.; LaPlante, D.A.; LaBrie, R.A.; Kidman, R.C.; Donato, A.N.; Stanton, M.V. Toward a syndrome model of addiction: Multiple expressions, common etiology. *Harv. Rev. Psychiatry* **2004**, *12*, 367–374.
19. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority? *Eval. Health Prof.* **2011**, *34*, 3–56.
20. Conner, B.T.; Stein, J.A.; Longshore, D.; Stacy, A.W. Associations between drug abuse treatment and cigarette use: Evidence of substance replacement. *Exp. Clin. Psychopharmacol.* **1999**, *7*, 64–71.
21. Blume, S.B. Pathological gambling and switching addictions: Report of a case. *J. Gambl. Stud.* **1994**, *10*, 87–96.
22. Hollander, E. Behavioral addictions and dirty drugs. *CNS Spectr.* **2009**, *14*, 60–61.
23. Yen, J.-Y.; Ko, C.-H.; Yen, C.-F.; Chen, C.-S.; Chen, C.-C. The association between harmful alcohol use and Internet addiction among college students: Comparison of personality. *Psychiatry Clin. Neurosci.* **2009**, *63*, 218–224.
24. Ko, C.H.; Yen, J.Y.; Yen, C.F.; Chen, C.S.; Weng, C.C.; Chen, C.C. The association between Internet addiction and problematic alcohol use in adolescents: The problem behavior model. *Cyberpsychol. Behav.* **2008**, *11*, 571–576.

25. Ko, C.-H.; Yen, J.-Y.; Chen, C.-C.; Chen, S.-H.; Wu, K.; Yen, C.-F. Tridimensional personality of adolescents with Internet addiction and substance use experience. *Can. J. Psychiatry Rev. Can. Psychiatr.* **2006**, *51*, 887–894.
26. Gong, J.; Chen, X.; Zeng, J.; Li, F.; Zhou, D.; Wang, Z. Adolescent addictive Internet use and drug abuse in Wuhan, China. *Addic. Res. Theory* **2009**, *17*, 291–305.
27. Liu, T.C.; Desai, R.A.; Krishnan-Sarin, S.; Cavallo, D.A.; Potenza, M.N. Problematic Internet use and health in adolescents: Data from a high school survey in Connecticut. *J. Clin. Psychiatry* **2011**, *72*, 836–845.
28. Barry, D.T.; Steinberg, M.A.; Wu, R.; Potenza, M.N. Differences in characteristics of Asian American and white problem gamblers calling a gambling helpline. *CNS Spectr.* **2009**, *14*, 83–91.
29. Young, K.S. Evaluation and treatment of Internet addiction. *VandeCreek Leon* **1999**, *17*, 19–31.
30. Davis, R.A. Problematic internet use: Structure of the construct and association with personality, stress, and coping. *Diss. Abstr. Int. Sect. B* **2004**, *65*, 472.
31. Sussman, S.; Sun, P.; Rohrbach, L.A.; Spruijt-Metz, D. One-year outcomes of a drug abuse prevention program for older teens and emerging adults: Evaluating a motivational interviewing booster component. *Health Psychol.* **2011**, doi:10.1037/a0025756.
32. Huang, G.C.; Okamoto, J.; Valente, T.W.; Sun, P.; Wei, Y.; Johnson, C.A.; Unger, J.B. Effects of social media and social standing on smoking behaviors among adolescents in China. *J. Child. Media* **2012**, *6*, 100–118.
33. Davis, R.A.; Flett, G.L.; Besser, A. Validation of a new scale for measuring problematic Internet use: Implications for pre-employment screening. *CyberPsychol. Behav.* **2002**, *5*, 331–345.
34. Xiao, L.; Bechara, A.; Cen, S.; Grenard, J.L.; Stacy, A.; Gallaher, P.; Wei, Y.; Jia, Y.; Johnson, C.A. Affective decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in 10th grade Chinese adolescent smokers *Nicotine Tob. Res.* **2008**, *10*, 1085–1097.
35. Grant, J.E.; Potenza, M.N.; Weinstein, A.; Gorelick, D.A. Introduction to behavioral addictions. *Am. J. Drug Alcohol Abuse* **2010**, *36*, 233–241.
36. Griswold, M.E.; Localio, A.R.; Mulrow, C. Propensity score adjustment with multilevel data: Setting your sites on decreasing selection bias. *Ann. Intern. Med.* **2010**, *152*, 393–395.
37. Muthen, L.K.; Muthen, B.O. *Mplus Statistical Analysis with Latent Variables*; Muthen & Muthen: Los Angeles, CA, USA, 2011.
38. Xie, Y.B.; Zhou, P.; Xu, L.P.; Peng, Z.W. Prevalence of internet addiction and the related factors in middle school students in Guangzhou. *Nan Fang Yi Ke Da Xue Xue Bao* **2010**, *30*, 1801–1804.
39. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399–3415.
40. Gehricke, J.G.; Loughlin, S.E.; Whalen, C.K.; Potkin, S.G.; Fallon, J.H.; Jamner, L.D.; Belluzzi, J.D.; Leslie, F.M. Smoking to self-medicate attentional and emotional dysfunctions. *Nicotine Tob. Res.* **2007**, *9*, S523–S536.
41. Field, M.; Schoenmakers, T.; Wiers, R.W. Cognitive processes in alcohol binges: A review and research agenda. *Curr. Drug Abuse Rev.* **2008**, *1*, 263–279.

42. Barnes, G.M.; Welte, J.W.; Hoffman, J.H. Relationship of alcohol use to delinquency and illicit drug use in adolescents: Gender, age, and racial/ethnic differences. *J. Drug Issues* **2002**, *Winter*, 153–178.
43. Lengua, L.J.; Stormshak, E.A. Gender, gender roles and personality: Gender differences in the prediction of coping and psychological symptoms. *Sex Roles* **2000**, *43*, 787–820.
44. Weiss, J.W.; Spruijt-Metz, D.; Palmer, P.H.; Chou, C.-P.; Johnson, C.A.; China Seven Cities Study Research Team. Smoking among adolescents in China: An analysis based upon the meanings of smoking theory. *Am. J. Health Promot.* **2006**, *20*, 171–178.
45. Yen, J.-Y.; Yen, C.-F.; Chen, C.-C.; Chen, S.-H.; Ko, C.-H. Family factors of internet addiction and substance use experience in Taiwanese adolescents. *CyberPsychol. Behav.* **2007**, *10*, 323–329.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Kuss, D.J.; Griffiths, M.D. Online Social Networking and Addiction—A Review of the Psychological Literature. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3528-3552; doi:10.3390/ijerph8093528.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Review

Online Social Networking and Addiction—A Review of the Psychological Literature

Daria J. Kuss * and Mark D. Griffiths

International Gaming Research Unit, Psychology Division, Nottingham Trent University, NG1 4BU, UK; E-Mail: mark.griffiths@ntu.ac.uk

* Author to whom correspondence should be addressed; E-Mail: daria.kuss@ntu.ac.uk; Tel.: +44-789-111-9490.

Received: 9 March 2011; in revised form: 12 August 2011 / Accepted: 22 August 2011 / Published: 29 August 2011

Abstract: Social Networking Sites (SNSs) are virtual communities where users can create individual public profiles, interact with real-life friends, and meet other people based on shared interests. They are seen as a 'global consumer phenomenon' with an exponential rise in usage within the last few years. Anecdotal case study evidence suggests that 'addiction' to social networks on the Internet may be a potential mental health problem for some users. However, the contemporary scientific literature addressing the addictive qualities of social networks on the Internet is scarce. Therefore, this literature review is intended to provide empirical and conceptual insight into the emerging phenomenon of addiction to SNSs by: (1) outlining SNS usage patterns, (2) examining motivations for SNS usage, (3) examining personalities of SNS users, (4) examining negative consequences of SNS usage, (5) exploring potential SNS addiction, and (6) exploring SNS addiction specificity and comorbidity. The findings indicate that SNSs are predominantly used for social purposes, mostly related to the maintenance of established offline networks. Moreover, extraverts appear to use social networking sites for social enhancement, whereas introverts use it for social compensation, each of which appears to be related to greater usage, as does low conscientiousness and high narcissism. Negative correlates of SNS usage include the decrease in real life social community participation and academic achievement, as well as relationship problems, each of which may be indicative of potential addiction.

Int. J. Environ. Res. Public Health **2011**, *8*

Keywords: social network addiction; social networking sites; literature review; motivations; personality; negative consequences; comorbidity; specificity

1. Introduction

"I'm an addict. I just get lost in Facebook" replies a young mother when asked why she does not see herself able to help her daughter with her homework. Instead of supporting her child, she spends her time chatting and browsing the social networking site [1]. This case, while extreme, is suggestive of a potential new mental health problem that emerges as Internet social networks proliferate. Newspaper stories have also reported similar cases, suggesting that the popular press was early to discern the potentially addictive qualities of social networking sites (SNS; *i.e.*, [2,3]). Such media coverage has alleged that women are at greater risk than men for developing addictions to SNSs [4].

The mass appeal of social networks on the Internet could potentially be a cause for concern, particularly when attending to the gradually increasing amounts of time people spend online [5]. On the Internet, people engage in a variety of activities some of which may be potentially to be addictive. Rather than becoming addicted to the medium *per se,* some users may develop an addiction to specific activities they carry out online [6]. Specifically, Young [7] argues that there are five different types of internet addiction, namely *computer addiction* (*i.e.*, computer game addiction), *information overload* (*i.e.*, web surfing addiction), *net compulsions* (*i.e.*, online gambling or online shopping addiction), *cybersexual addiction* (*i.e.*, online pornography or online sex addiction), and *cyber-relationship addiction* (*i.e.*, an addiction to online relationships). SNS addiction appears to fall in the last category since the purpose and main motivation to use SNSs is to establish and maintain both on- and offline relationships (for a more detailed discussion of this please refer to the section on motivations for SNS usage). From a clinical psychologist's perspective, it may be plausible to speak specifically of '*Facebook* Addiction Disorder' (or more generally 'SNS Addiction Disorder') because addiction criteria, such as neglect of personal life, mental preoccupation, escapism, mood modifying experiences, tolerance, and concealing the addictive behavior, appear to be present in some people who use SNSs excessively [8].

Social Networking Sites are virtual communities where users can create individual public profiles, interact with real-life friends, and meet other people based on shared interests. SNSs are "web-based services that allow individuals to: (1) construct a public or semi-public profile within a bounded system, (2) articulate a list of other users with whom they share a connection, and (3) view and traverse their list of connections and those made by others within the system" [9]. The focus is placed on established networks, rather than on networking, which implies the construction of new networks. SNSs offer individuals the possibilities of networking and sharing media content, therefore embracing the main Web 2.0 attributes [10], against the framework of their respective structural characteristics.

In terms of SNS history, the first social networking site (*SixDegrees*) was launched in 1997, based on the idea that everybody is linked with everybody else via six degrees of separation [9], and initially referred to as the "small world problem" [11]. In 2004, the most successful current SNS, *Facebook*, was established as a closed virtual community for Harvard students. The site expanded very quickly

and *Facebook* currently has more than 500 million users, of whom fifty percent log on to it every day. Furthermore, the overall time spent on *Facebook* increased by 566% from 2007 to 2008 [12]. This statistic alone indicates the exponential appeal of SNSs and also suggests a reason for a rise in potential SNS addiction. Hypothetically, the appeal of SNSs may be traced back to its reflection of today's individualist culture. Unlike traditional virtual communities that emerged during the 1990s based on shared interests of their members [13], social networking sites are egocentric sites. It is the individual rather than the community that is the focus of attention [9].

Egocentrism has been linked to Internet addiction [14]. Supposedly, the egocentric construction of SNSs may facilitate the engagement in addictive behaviors and may thus serve as a factor that attracts people to using it in a potentially excessive way. This hypothesis is in line with the PACE Framework for the etiology of addiction specificity [15]. Attraction is one of the four key components that may predispose individuals to becoming addicted to specific behaviors or substances rather than specific others. Accordingly, due to their egocentric construction, SNSs allow individuals to present themselves positively that may "raise their spirits" (*i.e.*, enhance their mood state) because it is experienced as pleasurable. This may lead to positive experiences that can potentially cultivate and facilitate learning experiences that drive the development of SNS addiction.

A behavioral addiction such as SNS addiction may thus be seen from a biopsychosocial perspective [16]. Just like substance-related addictions, SNS addiction incorporates the experience of the 'classic' addiction symptoms, namely mood modification (*i.e.*, engagement in SNSs leads to a favourable change in emotional states), salience (*i.e.*, behavioral, cognitive, and emotional preoccupation with the SNS usage), tolerance (*i.e.*, ever increasing use of SNSs over time), withdrawal symptoms (*i.e.*, experiencing unpleasant physical and emotional symptoms when SNS use is restricted or stopped), conflict (*i.e.*, interpersonal and intrapsychic problems ensue because of SNS usage), and relapse (*i.e.*, addicts quickly revert back in their excessive SNS usage after an abstinence period).

Moreover, scholars have suggested that a combination of biological, psychological and social factors contributes to the etiology of addictions [16,17], that may also hold true for SNS addiction. From this it follows that SNS addiction shares a common underlying etiological framework with other substance-related and behavioral addictions. However, due to the fact that the engagement in SNSs is different in terms of the actual expression of (Internet) addiction (*i.e.*, pathological use of social networking sites rather than other Internet applications), the phenomenon appears worthy of individual consideration, particularly when considering the potentially detrimental effects of both substance-related and behavioral addictions on individuals who experience a variety of negative consequences because of their addiction [18].

To date, the scientific literature addressing the addictive qualities of social networks on the Internet is scarce. Therefore, with this literature review, it is intended to provide empirical insight into the emerging phenomenon of Internet social network usage and potential addiction by (1) outlining SNS usage patterns, (2) examining motivations for SNS usage, (3) examining personalities of SNS users, (4) examining negative consequences of SNSs, (5) exploring potential SNS addiction, and (6) exploring SNS addiction specificity and comorbidity.

Int. J. Environ. Res. Public Health **2011**, *8*

2. Method

An extensive literature search was conducted using the academic database Web *of Knowledge* as well as *Google Scholar*. The following search terms as well as their derivatives were entered: social network, online network, addiction, compulsive, excessive, use, abuse, motivation, personality, and comorbidity. Studies were included if they: (i) included empirical data, (ii) made reference to usage patterns, (iii) motivations for usage, (iv) personality traits of users, (v) negative consequences of use, (vi) addiction, (vii) and/or comorbidity and specificity. A total of 43 empirical studies were identified from the literature, five of which specifically assessed SNS addiction.

3. Results

3.1. Usage

Social networking sites are seen as a 'global consumer phenomenon' and, as already noted, have experienced an exponential rise in usage within the last few years [12]. Of all Internet users, approximately one-third participate in SNSs and ten percent of the total time spent online is spent on SNSs [12]. In terms of usage, the results of the Parents and Teens 2006 Survey with a random sample of 935 participants in America revealed that 55% of youths used SNSs in that year [19]. The main reasons reported for this usage were staying in touch with friends (endorsed by 91%), and using them to make new friends (49%). This was more common among boys than girls. Girls preferred to use these sites in order to maintain contacts with actual friends rather than making new ones. Furthermore, half of the teenagers in this sample visited their SNS at least once a day which is indicative of the fact that in order to keep an attractive profile, frequent visits are necessary and this is a factor that facilitates potential excessive use [19]. Moreover, based on the results of consumer research, the overall usage of SNSs increased by two hours per month to 5.5 hours and active participation increased by 30% from 2009 to 2010 [5].

The findings of an online survey of 131 psychology students in the US [20] indicated that 78% used SNSs, and that 82% of males and 75% of females had SNS profiles. Of those, 57% used their SNS on a daily basis. The activities most often engaged in on SNSs were reading/responding to comments on their SNS page and/or posts to one's wall (endorsed by 60%; the "wall" is a special profile feature in *Facebook*, where people can post comments, pictures, and links, that can be responded to), sending/responding to messages/invites (14%), and browsing friends' profiles/walls/pages (13%; [20]). These results correspond with findings from a different study including another university student sample [21].

Empirical research has also suggested gender differences in SNS usage patterns. Some studies claim that men tend to have more friends on SNSs than women [22], whereas others have found the opposite [23]. In addition, men were found to take more risks with regards to disclosure of personal information [24,25]. Furthermore, one study reported that slightly more females used *MySpace* specifically (*i.e.*, 55% compared to 45% of males) [26].

Usage of SNSs has also been found to differ with regards to age group. A study comparing 50 teenagers (13–19 years) and the same number of older *MySpace* users (60 years and above) revealed that teenagers' friends' networks were larger and that their friends were more similar to themselves

with regards to age [23]. Furthermore, older users' networks were smaller and more dispersed age-wise. Additionally, teenagers made more use of *MySpace* web 2.0 features (*i.e.*, sharing video and music, and blogging) relative to older people [23].

With regards to how people react to using SNSs, a recent study [27] using psychophysiological measures (skin conductance and facial electromyography) found that social searching (*i.e.*, extracting information from friends' profiles), was more pleasurable than social browsing (*i.e.*, passively reading newsfeeds) [27]. This finding indicates that the goal-directed activity of social searching may activate the appetitive system, which is related to pleasurable experience, relative to the aversive system [28]. On a neuroanatomical level, the appetitive system has been found to be activated in Internet game overusers and addicts [29,30], which may be linked back to a genetic deficiency in the addicts' neurochemical reward system [31]. Therefore, the activation of the appetitive system in social network users who engage in social searching concurs with the activation of that system in people found to suffer from behavioral addictions. In order to establish this link for SNS specifically, further neurobiological research is required.

In reviewing SNS usage patterns, the findings of both consumer research and empirical research indicate that overall, regular SNS use has increased substantially over the last few years. This supports the availability hypothesis that where there is increased access and opportunity to engage in an activity (in this case SNSs), there is an increase in the numbers of people who engage in the activity [32]. Moreover, it indicates that individuals become progressively aware of this available supply and become more sophisticated with regards to their usage skills. These factors are associated with the pragmatics factor of addiction specificity etiology [15]. Pragmatics is one of the four key components of the addiction specificity model and it emphasizes access and habituation variables in the development of specific addictions. Therefore, the pragmatics of SNS usage appears to be a factor related to potential SNS addiction.

In addition to this, the findings of the presented studies indicate that compared to the general population, teenagers and students make most use of SNSs by utilizing the inherent Web 2.0 features. Additionally, there appear to be gender differences in usage, the specifics of which are only vaguely defined and thus require further empirical investigation. In addition, SNSs tend to be used mostly for social purposes of which extracting further information from friends' pages appears particularly pleasurable. This, in turn, may be linked to the activation of the appetitive system, which indicates that engaging in this particular activity may stimulate the neurological pathways known to be related to addiction experience.

3.2. Motivations

Studies suggest that SNS usage in general, and *Facebook* in particular, differs as a function of motivation (*i.e.*, [33]). Drawing on uses and gratification theory, media are used in a goal-directed way for the purpose of gratification and need satisfaction [34] which have similarities with addiction. Therefore, it is essential to understand the motivations that underlie SNS usage. Persons with higher social identity (*i.e.*, solidarity to and conformity with their own social group), higher altruism (related to both, kin and reciprocal altruism) and higher telepresence (*i.e.*, feeling present in the virtual environment) tend to use SNSs because they perceive encouragement for participation from the social

network [35]. Similarly, the results of a survey comprising 170 US university students indicated that social factors were more important motivations for SNS usage than individual factors [36]. More specifically, these participants' interdependent self-construal (*i.e.*, the endorsement of collectivist cultural values), led to SNS usage that in turn resulted in higher levels of satisfaction, relative to independent self-construal, which refers to the adoption of individualist values. The latter were not related to motivations for using SNSs [36].

Another study by Barker [37] presented similar results, and found that collective self-esteem and group identification positively correlated with peer group communication via SNSs. Cheung, Chiu and Lee [38] assessed social presence (*i.e.*, the recognition that other persons share the same virtual realm, the endorsement of group norms, maintaining interpersonal interconnectivity and social enhancement with regards to SNS usage motivations). More specifically, they investigated the We-intention to use *Facebook* (*i.e.*, the decision to continue using a SNS together in the future). The results of their study indicated that We-intention positively correlated with the other variables [38].

Similarly, social reasons appeared as the most important motives for using SNSs in another study [20]. The following motivations were endorsed by the participating university student sample: keeping in touch with friends they do not see often (81%), using them because all their friends had accounts (61%), keeping in touch with relatives and family (48%), and making plans with friends they see often (35%). A further study found that a large majority of students used SNSs for the maintenance of offline relationships, whereas some preferred to use this type of Internet application for communication rather than face-to-face interaction [39].

The particular forms of virtual communication in SNSs include both asynchronous (*i.e.*, personal messages sent within the SNS) and synchronous modes (*i.e.*, embedded chat functions within the SNS) [40]. On behalf of the users, these communication modes require learning differential vocabularies, namely Internet language [41,42]. The idiosyncratic form of communication via SNSs is another factor that may fuel potential SNS addiction because communication has been identified as a component of the addiction specificity etiology framework [15]. Therefore, it can be hypothesized that users who prefer communication via SNSs (as compared to face-to-face communication) are more likely to develop an addiction to using SNSs. However, further empirical research is needed to confirm such a speculation.

Moreover, research suggests that SNSs are used for the formation and maintenance of different forms of social capital [43]. Social capital is broadly defined as *"the sum of the resources, actual or virtual, that accrue to an individual or a group by virtue of possessing a durable network of more or less institutionalized relationships of mutual acquaintance and recognition"* [44]. Putnam [45] differentiates bridging and bonding social capital from one another. Bridging social capital refers to weak connections between people that are based on information-sharing rather than emotional support. These ties are beneficial in that they offer a wide range of opportunities and access to broad knowledge because of the heterogeneity of the respective network's members [46]. Alternatively, bonding social capital indicates strong ties usually between family members and close friends [45].

SNSs are thought to increase the size of potential networks because of the large number of possible weak social ties among members, which is enabled via the structural characteristics of digital technology [47]. Therefore, SNSs do not function as communities in the traditional sense. They do not include membership, shared influence, and an equal power allocation. Instead, they can be

conceptualized as networked individualism, allowing the establishment of numerous self-perpetuating connections that appear advantageous for users [48]. This is supported by research that was carried out on a sample of undergraduate students [43]. More specifically, this study found that maintaining bridging social capital via participation in SNSs appeared to be beneficial for students with regards to potential employment opportunities in addition to sustaining ties with old friends. Overall, the benefits of bridging social capital formed via participation in SNSs appeared to be particularly advantageous for individuals with low-self esteem [49]. However, the ease of establishing and maintaining bridging social capital may become one of the reasons why people with low self-esteem are drawn to using SNSs in a potentially excessive manner. Lower self-esteem, in turn, has been linked to Internet addiction [50,51].

Furthermore, SNS usage has been found to differ between people and cultures. A recent study [52] including samples from the US, Korea and China demonstrated that the usage of different *Facebook* functions was associated with the creation and maintenance of either bridging or bonding social capital. People in the US used the 'Communication' function (*i.e.*, conversation and opinion sharing) in order to bond with their peers. However, Koreans and Chinese used 'Expert Search' (*i.e.*, searching for associated professionals online) and 'Connection' (*i.e.*, maintaining offline relationships) for the formation and sustaining of both bonding and bridging social capital [52]. These findings indicate that due to cultural differences in SNS usage patterns, it appears necessary to investigate and contrast SNS addiction in different cultures in order to discern both similarities and differences.

Additionally, the results of an online survey with a student convenience sample of 387 participants [53] indicated that several factors significantly predicted the intention to use SNSs as well as their actual usage. The identified predictive factors were (i) playfulness (*i.e.*, enjoyment and pleasure), (ii) the critical mass of the users who endorsed the technology, (iii) trust in the site, (iv) perceived ease of use, and (v) perceived usefulness. Moreover, normative pressure (*i.e.*, the expectations of other people with regards to one's behavior) had a negative relationship with SNS usage. These results suggest that it is particularly the enjoyment associated with SNS use in a hedonic context (which has some similarities to addictions), as well as the recognition that a critical mass uses SNSs that motivates people to make use of those SNSs themselves [53].

Another study [54] used a qualitative methodology to investigate why teenagers use SNSs. Interviews were conducted with 16 adolescents aged 13 to 16 years. The results indicated that the sample used SNSs in order to express and actualize their identities either via self-display of personal information (which was true for the younger sample) or via connections (which was true for the older participants). Each of these motivations was found to necessitate a trade-off between potential opportunities for self-expression and risks with regards to compromising privacy on behalf of the teenagers [54].

A study by Barker [37] also suggested there may be differences in motivations for SNS use between men and women. Females used SNSs for communication with peer group members, entertainment and passing time, whereas men used it in an instrumental way for social compensation, learning, and social identity gratifications (*i.e.*, the possibility to identify with group members who share similar characteristics). Seeking friends, social support, information, and entertainment were found to be the most significant motivations for SNS usage in a sample of 589 undergraduate students [55]. In addition to this, endorsement of these motivations was found to differ across cultures. Kim *et al.* [55]

found that Korean college students sought social support from already established relationships via SNSs, whereas American college students looked for entertainment. Similarly, Americans had significantly more online friends than Koreans, suggesting that the development and maintenance of social relationships on SNSs was influenced by cultural artefacts [55]. Furthermore, technology-relevant motivations were related to SNS use. The competence in using computer-mediated communication (*i.e.*, the motivation to, knowledge of, and efficacy in using electronic forms of communication) was found to be significantly associated with spending more time on *Facebook* and checking one's wall significantly more often [33].

Overall, the results of these studies indicate that SNSs are predominantly used for social purposes, mostly related to the maintenance of established offline networks, relative to individual ones. In line with this, people may feel compelled to maintaining their social networks on the Internet which may lead to using SNSs excessively. The maintenance of already established offline networks itself can therefore be seen as an attraction factor, which according to Sussman *et al.* [15] is related to the etiology of specific addictions. Furthermore, viewed from a cultural perspective, it appears that motivations for usage differ between members of Asian and Western countries as well as between genders and age groups. However, in general, the results of the reported studies suggest that the manifold ties pursued online are indicative, for the most part, of bridging rather than bonding social capital. This appears to show that SNSs are primarily used as a tool for staying connected.

Staying connected is beneficial to such individuals because it offers them a variety of potential academic and professional opportunities, as well as access to a large knowledge base. As the users' expectations of connectivity are met through their SNS usage, the potential for developing SNS addiction may increase as a consequence. This is in accordance with the expectation factor that drives the etiology of addiction to a specific behavior [15]. Accordingly, the supposed expectations and benefits of SNS use may go awry particularly for people with low self-esteem. They may feel encouraged to spend excessive amounts of time on SNSs because they perceive it as advantageous. This, in turn, may potentially develop into an addiction to using SNSs. Clearly, future research is necessary in order to establish this link empirically.

Moreover, there appear certain limitations to the studies presented. Many studies included small convenience samples, teenagers or university students as participants, therefore severely limiting the generalizability of findings. Thus, researchers are advised to take this into consideration and amend their sampling frameworks by using more representative samples and thus improve the external validity of the research.

3.3. Personality

A number of personality traits appear to be associated with the extent of SNS use. The findings of some studies (e.g., [33,56]) indicate that people with large offline social networks, who are more extroverted, and who have higher self-esteem, use *Facebook* for social enhancement, supporting the principle of 'the rich get richer'. Correspondingly, the size of people's online social networks correlates positively with life satisfaction and well-being [57], but does neither have an effect on the size of the offline network nor on emotional closeness to people in real life networks [58].

However, people with only a few offline contacts compensate for their introversion, low-self esteem, and low life-satisfaction by using *Facebook* for online popularity, thus corroborating the principle of 'the poor get richer' (*i.e.*, the social compensation hypothesis) [37,43,56,59]. Likewise, people higher in narcissistic personality traits tend to be more active on *Facebook* and other SNSs in order to present themselves favourably online because the virtual environment empowers them to construct their ideal selves [59-62]. The relationship between narcissism and *Facebook* activity may be related to the fact that narcissists have an imbalanced sense of self, fluctuating between grandiosity with regards to explicit agency and low self-esteem concerning implicit communion and vulnerability [63,64]. Narcissistic personality, in turn, has been found to be associated with addiction [65]. This finding will be discussed in more detail in the section on addiction.

Moreover, it appears that people with different personality traits differ in their usage of SNSs [66] and prefer to use distinct functions of *Facebook* [33]. People high in extraversion and openness to experience use SNSs more frequently, with the former being true for mature and the latter for young people [66]. Furthermore, extraverts and people open to experiences are members of significantly more groups on *Facebook,* use socializing functions more [33], and have more *Facebook* friends than introverts [67], which delineates the former's higher sociability in general [68]. Introverts, on the other hand, disclose more personal information on their pages [67]. Additionally, it appears that particularly shy people spend large amounts of time on *Facebook* and have large amounts of friends on this SNS [69]. Therefore, SNSs may appear beneficial for those whose real-life networks are limited because of the possibility of easy access to peers without the demands of real-life proximity and intimacy. This ease of access entails a higher time commitment for this group, which may possibly result in excessive and/or potentially addictive use.

Likewise, men with neurotic traits use SNSs more frequently than women with neurotic traits [66]. Furthermore, neurotics (in general) tend to use *Facebook's* wall function, where they can receive and post comments, whereas people with low neuroticism scores prefer posting photos [33]. This may be due to the neurotic individual's greater control over emotional content with regards to text-based posts rather than visual displays [33]. However, another study [67] found the opposite, namely that people scoring high on neuroticism were more inclined to post their photographs on their page. In general, the findings for neuroticism imply that those scoring high on this trait disclose information because they seek self-assurance online, whereas those scoring low are emotionally secure and thus share information in order to express themselves [67]. High self-disclosure on SNSs, in turn, was found to positively correlate with measures of subjective well-being [57]. It remains questionable whether this implies that low self-disclosure on SNSs may be related to higher risk for potential addiction. By disclosing more personal information on their pages, users put themselves at risk for negative feedback, which has been linked to lower well-being [70]. Therefore, the association between self-disclosure on SNSs and addiction needs to be addressed empirically in future studies.

With regards to agreeableness, it was found that females scoring high on this trait upload significantly more pictures than females scoring low, with the opposite being true for males [67]. In addition to this, people with high conscientiousness were found to have significantly more friends and to upload significantly less pictures than those scoring low on this personality trait [67]. An explanation for this finding may be that conscientious people tend to cultivate their online and offline contacts more without the necessity to share too much personal information publicly.

Int. J. Environ. Res. Public Health **2011**, *8*

Overall, the results of these studies suggest that extraverts use SNSs for social enhancement, whereas introverts use it for social compensation, each of which appears to be related to greater SNS usage. With regards to addiction, both groups could potentially develop addictive tendencies for different reasons, namely social enhancement and social compensation. In addition, the dissimilar findings of studies with regards to the number of friends introverts have online deserve closer scrutiny in future research. The same applies for the results with regards to neuroticism. On the one hand, neurotics use SNSs frequently. On the other hand, studies indicate different usage preferences for people who score high on neuroticism, which calls for further investigation. Furthermore, the structural characteristics of these Internet applications, (*i.e.*, their egocentric construction) appear to allow favourable self-disclosure, which draws narcissists to use it. Finally, agreeableness and conscientiousness appear to be related to the extent of SNS usage. Higher usage associated with narcissistic, neurotic, extravert and introvert personality characteristics may implicate that each of these groups is particularly at risk for developing an addiction to using SNSs.

3.4. Negative Correlates

Some studies have highlighted a number of potential negative correlates of extensive SNS usage. For instance, the results of an online survey of 184 Internet users indicated that people who use SNS more in terms of time spent on usage were perceived to be less involved with their real life communities [71]. This is similar to the finding that people who do not feel secure about their real-life connections to peers and thus have a negative social identity tend to use SNSs more in order to compensate for this [37]. Moreover, it seems that the nature of the feedback from peers that is received on a person's SNS profile determines the effects of SNS usage on wellbeing and self-esteem.

More specifically, Dutch adolescents aged 10 to 19 years who received predominantly negative feedback had low self-esteem which in turn led to low wellbeing [70]. Given that people tend to be disinhibited when they are online [72], giving and receiving negative feedback may be more common on the Internet than in real life. This may entail negative consequences particularly for people with low self-esteem who tend to use SNSs as compensation for real-life social network paucity because they are dependent upon the feedback they receive via these sites [43]. Therefore, potentially, people with lower self-esteem are a population at risk for developing an addiction to using SNSs.

According to a more recent study assessing the relationships between *Facebook* usage and academic performance in a sample of 219 university students [73], *Facebook* users had lower Grade Point Averages and spent less time studying than students who did not use this SNS. Of the 26% of students reporting an impact of their usage on their lives, three-quarters (74%) claimed that it had a negative impact, namely procrastination, distraction, and poor time-management. A potential explanation for this may be that students who used the Internet to study may have been distracted by simultaneous engagement in SNSs, implying that this form of multitasking is detrimental to academic achievement [73].

In addition to this, it appears that the usage of *Facebook* may in some circumstances have negative consequences for romantic relationships. The disclosure of rich private information on one's *Facebook* page including status updates, comments, pictures, and new friends, can result in jealous cyberstalking [74], including interpersonal electronic surveillance (IES; [75]) by one's partner. This

was reported to lead to jealousy [76,77] and, in the most extreme cases, divorce and associated legal action [78].

These few existent studies highlight that in some circumstances, SNS usage can lead to a variety of negative consequences that imply a potential decrease in involvement in real-life communities and worse academic performance, as well as relationship problems. Reducing and jeopardizing academic, social and recreational activities are considered as criteria for substance dependence [18] and may thus be considered as valid criteria for behavioral addictions [79], such as SNS addiction. In light of this, endorsing these criteria appears to put people at risk for developing addiction and the scientific research base outlined in the preceding paragraphs supports the potentially addictive quality of SNSs.

Notwithstanding these findings, due to the lack of longitudinal designs used in the presented studies, no causal inferences can be drawn with regards to whether the excessive use of SNSs is the causal factor for the reported negative consequences. Moreover, potential confounders need to be taken into consideration. For instance, the aspect of university students' multi-tasking when studying appears to be an important factor related to poor academic achievement. Moreover, pre-existent relationship difficulties in the case of romantic partners may potentially be exacerbated by SNS use, whereas the latter does not necessarily have to be the primary driving force behind the ensuing problems. Nevertheless, the findings support the idea that SNSs are used by some people in order to cope with negative life events. Coping, in turn, has been found to be associated with both substance dependence and behavioral addictions [80]. Therefore, it appears valid to claim that there is a link between dysfunctional coping (*i.e.*, escapism and avoidance) and excessive SNS use/addiction. In order to substantiate this conjecture and to more fully investigate the potential negative correlates associated with SNS usage, further research is needed.

3.5. Addiction

Researchers have suggested that the excessive use of new technologies (and especially online social networking) may be particularly addictive to young people [81]. In accordance with the biopsychosocial framework for the etiology of addictions [16] and the syndrome model of addiction [17], it is claimed that those people addicted to using SNSs experience symptoms similar to those experienced by those who suffer from addictions to substances or other behaviors [81]. This has significant implications for clinical practice because unlike other addictions, the goal of SNS addiction treatment cannot be total abstinence from using the Internet *per se* since the latter is an integral element of today's professional and leisure culture. Instead, the ultimate therapy aim is controlled use of the Internet and its respective functions, particularly social networking applications, and relapse prevention using strategies developed within cognitive-behavioral therapies [81].

In addition to this, scholars have hypothesized that young vulnerable people with narcissistic tendencies are particularly prone to engaging with SNSs in an addictive way [65]. To date, only three empirical studies have been conducted and published in peer-reviewed journals that have specifically assessed the addictive potential of SNSs [82-84]. In addition to this, two publicly available Master's theses have analyzed the SNS addiction and will be presented subsequently for the purpose of inclusiveness and the relative lack of data on the topic [85,86]. In the first study [83], 233 undergraduate university students (64% females, mean age = 19 years, *SD* = 2 years) were

surveyed using a prospective design in order to predict high level use intentions and actual high-level usage of SNSs via an extended model of the theory of planned behavior (TPB; [87]). High-level usage was defined as using SNSs at least four times per day. TPB variables included measures of intention for usage, attitude, subjective norm, and perceived behavioral control (PBC). Furthermore, self-identity (adapted from [88]), belongingness [89], as well as past and potential future usage of SNSs were investigated. Finally, addictive tendencies were assessed using eight questions scored on Likert scales (based on [90]).

One week after completion of the first questionnaire, participants were asked to indicate on how many days during the last week they had visited SNSs at least four times a day. The results of this study indicated that past behavior, subjective norm, attitude, and self-identity significantly predicted both behavioral intention as well as actual behavior. Additionally, addictive tendencies with regards to SNS use were significantly predicted by self-identity and belongingness [83]. Therefore, those who identified themselves as SNS users and those who looked for a sense of belongingness on SNSs appeared to be at risk for developing an addiction to SNSs.

In the second study [82], an Australian university student sample of 201 participants (76% female, mean age = 19, *SD* = 2) was drawn upon in order to assess personality factors via the short version of the NEO Personality Inventory (NEO-FFI; [91]), the Self-Esteem Inventory (SEI; [92]), time spent using SNSs, and an Addictive Tendencies Scale (based on [90,93]). The Addictive Tendencies Scale included three items measuring salience, loss of control, and withdrawal. The results of a multiple regression analysis indicated that high extraversion and low conscientiousness scores significantly predicted both addictive tendencies and the time spent using an SNS. The researchers suggested that the relationship between extraversion and addictive tendencies could be explained by the fact that using SNSs satisfies the extraverts' need to socialize [82]. The findings with regards to lack of conscientiousness appear to be in line with previous research on the frequency of general Internet use in that people who score low on conscientiousness tend to use the Internet more frequently than those who score high on this personality trait [94].

In the third study, Karaiskos *et al.* [84] report the case of a 24-year old female who used SNS to such an extent that her behavior significantly interfered with her professional and private life. As a consequence, she was referred to a psychiatric clinic. She used *Facebook* excessively for at least five hours a day and was dismissed from her job because she continuously checked her SNS instead of working. Even during the clinical interview, she used her mobile phone to access *Facebook*. In addition to excessive use that led to significant impairment in a variety of areas in the woman's life, she developed anxiety symptoms as well as insomnia, which suggestively points to the clinical relevance of SNS addiction. Such extreme cases have led to some researchers to conceptualize SNS addiction as Internet spectrum addiction disorder [84]. This indicates that first, SNS addiction can be classified within the larger framework of Internet addictions, and second, that it is a specific Internet addiction, alongside other addictive Internet applications such as Internet gaming addiction [95], Internet gambling addiction [96], and Internet sex addiction [97].

In the fourth study [85], SNS game addiction was assessed via the Internet Addiction Test [98] using 342 Chinese college students aged 18 to 22 years. In this study, SNS game addiction referred specifically to being addicted to the SNS game *Happy Farm*. Students were defined as

addicted to using this SNS game when they endorsed a minimum of five out of eight total items of the IAT. Using this cut-off, 24% of the sample were identified as addicted [85].

Moreover, the author investigated gratifications of SNS game use, loneliness [99], leisure boredom [100], and self esteem [101]. The findings indicated that there was a weak positive correlation between loneliness and SNS game addiction and a moderate positive correlation between leisure boredom and SNS game addiction. Moreover, the gratifications "inclusion" (in a social group) and "achievement" (in game), leisure boredom, and male gender significantly predicted SNS game addiction [85].

In the fifth study [86], SNS addiction was assessed in a sample of 335 Chinese college students aged 19 to 28 years using Young's Internet Addiction Test [98] modified to specifically assess the addiction to a common Chinese SNS, namely *Xiaonei.com*. Users were classified as addicted when they endorsed five or more of the eight addiction items specified in the IAT. Moreover, the author assessed loneliness [99], user gratifications (based on the results of a previous focus group interview), usage attributes and patterns of SNS website use [86].

The results indicated that of the total sample, 34% were classified as addicted. Moreover, loneliness significantly and positively correlated with frequency and session length of using *Xiaonei.com* as well as SNS addiction. Likewise, social activities and relationship building were found to predict SNS addiction [86].

Unfortunately, when viewed from a critical perspective, the quantitative studies reviewed here suffer from a variety of limitations. Initially, the mere assessment of addiction tendencies does not suffice to demarcate real pathology. In addition, the samples were small, specific, and skewed with regards to female gender. This may have led to the very high addiction prevalence rates (up to 34%) reported [86]. Clearly, it needs to be ensured that rather than assessing excessive use and/or preoccupation, addiction specifically needs to be assessed.

Wilson *et al.*'s study [82] suffered from endorsing only three potential addiction criteria which is not sufficient for establishing addiction status clinically. Similarly, significant impairment and negative consequences that discriminate addiction from mere abuse [18] were not assessed in this study at all. Thus, future studies have great potential in addressing the emergent phenomenon of addiction to using social networks on the Internet by means of applying better methodological designs, including more representative samples, and using more reliable and valid addiction scales so that current gaps in empirical knowledge can be filled.

Furthermore, research must address the presence of specific addiction symptoms beyond negative consequences. These might be adapted from the DSM-IV TR criteria for substance dependence [18] and the ICD-10 criteria for a dependence syndrome [102], including (i) tolerance, (ii) withdrawal, (iii) increased use, (iv) loss of control, (v) extended recovery periods, (vi) sacrificing social, occupational and recreational activities, and (vii) continued use despite of negative consequences. These have been found to be adequate criteria for diagnosing behavioral addictions [79] and thus appear sufficient to be applied to SNS addiction. In order to be diagnosed with SNS addiction, at least three (but preferably more) of the above mentioned criteria should be met in the same 12-month period and they must cause significant impairment to the individual [18].

In light of this qualitative case study, it appears that from a clinical perspective, SNS addiction is a mental health problem that may require professional treatment. Unlike the quantitative studies, the

case study emphasizes the significant individual impairment that is experienced by individuals that spans a variety of life domains, including their professional life as well as their psychosomatic condition. Future researchers are therefore advised to not only investigate SNS addiction in a quantitative way, but to further our understanding of this new mental health problem by analyzing cases of individuals who suffer from excessive SNS usage.

3.6. Specificity and Comorbidity

It appears essential to pay adequate attention to (i) the specificity of SNS addiction and (ii) potential comorbidity. Hall *et al.* [103] outline three reasons why it is necessary to address comorbidity between mental disorders, such as addictions. First, a large number of mental disorders feature additional (sub)clinical problems/disorders. Second, comorbid conditions must be addressed in clinical practice in order to improve treatment outcomes. Third, specific prevention programs may be developed which incorporate different dimensions and treatment modalities that particularly target associated mental health problems. From this it follows that assessing the specificity and potential comorbidities of SNS addiction is important. However, to date, research addressing this topic is virtually non-existent. There has been almost no research on the co-occurrence of SNS addiction with other types of addictive behavior, mainly because there have been so few studies examining SNS addiction as highlighted in the previous section. However, based on the small empirical base, there are a number of speculative assumptions that can be made about co-addiction co-morbidity in relation to SNS addiction.

Firstly, for some individuals, their SNS addiction takes up such a large amount of available time that it is highly unlikely that it would co-occur with other behavioral addictions unless the other behavioral addiction(s) can find an outlet via social networking sites (e.g., gambling addiction, gaming addiction). Put simply, there would be little face validity in the same individual being, for example, both a workaholic and a social networking addict, or an exercise addict and a social networking addict, mainly because the amount of daily time available to engage in two behavioral addictions simultaneously would be highly unlikely. Still, it is necessary to pinpoint the respective addictive behaviors because some of these behaviors may in fact co-occur. In one study that included a clinical sample diagnosed with substance dependencies, Malat and colleagues [104] found that 61% pursued at least one and 31% engaged in two or more problematic behaviors, such as overeating, unhealthy relationships and excessive Internet use. Therefore, although a simultaneous addiction to behaviors such as working and using SNS is relatively unlikely, SNS addiction may potentially co-occur with overeating and other excessive sedentary behaviors.

Thus, secondly, it is theoretically possible for a social networking addict to have an additional drug addiction, as it is perfectly feasible to engage in both a behavioral and chemical addiction simultaneously [16]. It may also make sense from a motivational perspective. For instance, if one of the primary reasons social network addicts are engaging in the behavior is because of their low self-esteem, it makes intuitive sense that some chemical addictions may serve the same purpose. Accordingly, studies suggest that the engagement in addictive behaviors is relatively common among persons who suffer from substance dependence. In one study, Black *et al.* [105] found that 38% of problematic computer users in their sample had a substance use disorder in addition to their behavioral

problems/addiction. Apparently, research indicates that some persons who suffer from Internet addiction experience other addictions at the same time.

Of a patient sample including 1,826 individuals treated for substance addictions (mainly cannabis addiction), 4.1% were found to suffer from Internet addiction [106]. Moreover, the findings of further research [107] indicated that Internet addiction and substance use experience in adolescents share common family factors, namely higher parent-adolescent conflict, habitual alcohol use of siblings, perceived parents' positive attitude to adolescent substance use, and lower family functioning. Moreover, Lam *et al.* [108] assessed Internet addiction and associated factors in a sample of 1,392 adolescents aged 13–18 years. In terms of potential comorbidity, they found that drinking behavior was a risk factor for being diagnosed with Internet addiction using the Internet Addiction Test [109]. This implies that potentially, alcohol abuse/dependence can be associated with SNS addiction. Support for this comes from Kuntsche *et al.* [110]. They found that in Swiss adolescents, the expectancy of social approval was associated with problem drinking. Since SNSs are inherently social platforms that are used by people for social purposes, it appears reasonable to deduce that there may indeed be people who suffer from comorbid addictions, namely SNS addiction and alcohol dependence.

Thirdly, it appears that there may be a relationship between SNS addiction specificity and personality traits. Ko *et al.* [111] found that Internet addiction (IA) was predicted by high novelty seeking (NS), high harm avoidance (HA), and low reward dependence (RD) in adolescents. Those adolescents who were addicted to the Internet and who had experience of substance use scored significantly higher on NS and lower on HA than the IA group. Therefore, it appears that HA particularly impacts Internet addiction specificity because high HA discriminates Internet addicts from individuals who are not only addicted to the Internet, but who use substances. Therefore, it seems plausible to hypothesize that persons with low harm avoidance are in danger of developing comorbid addictions to SNSs and substances. Accordingly, research needs to address this difference specifically for those who are addicted to using SNSs in order to demarcate this potential disorder from comorbid conditions.

In addition to this, it seems reasonable to specifically address the respective activities people can engage in on their SNS. There have already been a number of researchers who have begun to examine the possible relationship between social networking and gambling [112-116], and social networking and gaming [113,116,117]. All of these writings have noted how the social networking medium can be used for gambling and/or gaming. For instance, online poker applications and online poker groups on social networking sites are among the most popular [115], and others have noted the press reports surrounding addiction to social networking games such as *Farmville* [117]. Although there have been no empirical studies to date examining addiction to gambling or gaming via social networking, there is no reason to suspect that those playing in the social networking medium are any less likely than those playing other online or offline media to become addicted to gambling and/or gaming.

Synoptically, addressing the specificity of SNS addiction and comorbidities with other addictions is necessary for (i) comprehending this disorder as distinct mental health problem while (ii) paying respect to associated conditions, which will (iii) aid treatment and (iv) prevention efforts. From the reported studies, it appears that the individual's upbringing and psychosocial context are influential factors with regards to potential comorbidity between Internet addiction and substance dependence, which is supported by scientific models of addictions and their etiology [16,17]. Moreover, alcohol

and cannabis dependence were outlined as potential co-occurring problems. Nonetheless, apart from this, the presented studies do not specifically address the discrete relationships between particular substance dependencies and individual addictive behaviors, such as addiction to using SNSs. Therefore, future empirical research is needed in order to shed more light upon SNS addiction specificity and comorbidity.

4. Discussion and Conclusions

The aim of this literature review was to present an overview of the emergent empirical research relating to usage of and addiction to social networks on the Internet. Initially, SNSs were defined as virtual communities offering their members the possibility to make use of their inherent Web 2.0 features, namely networking and sharing media content. The history of SNSs dates back to the late 1990s, suggesting that they are not as new as they may appear in the first place. With the emergence of SNSs such as *Facebook*, overall SNS usage has accelerated in such a way that they are considered a global consumer phenomenon. Today, more than 500 million users are active participants in the *Facebook* community alone and studies suggest that between 55% and 82% of teenagers and young adults use SNSs on a regular basis. Extracting information from peers' SNS pages is an activity that is experienced as especially enjoyable and it has been linked with the activation of the appetitive system, which in turn is related to addiction experience.

In terms of sociodemographics, the studies presented indicate that overall, SNS usage patterns differ. Females appear to use SNS in order to communicate with members of their peer group, whereas males appear to use them for the purposes of social compensation, learning, and social identity gratifications [37]. Furthermore, men tend to disclose more personal information on SNS sites relative to women [25,118]. Also, more women were found to use *MySpace* specifically relative to men [26]. Moreover, usage patterns were found to differ between genders as a function of personality. Unlike women with neurotic traits, men with neurotic traits were found to be more frequent SNS users [66]. In addition to this, it was found that males were more likely to be addicted to SNS games specifically relative to females [85]. This is in line with the finding that males in general are a population at risk for developing an addiction to playing online games [95].

The only study that assessed age differences in usage [23] indicated that the latter in fact varies as a function of age. Specifically, "silver surfers" (*i.e.*, those over the age of 60 years) have a smaller circle of online friends that differs in age relative to younger SNS users. Based on the current empirical knowledge that has predominantly assessed young teenage and student samples, it appears unclear whether older people use SNSs excessively and whether they potentially become addicted to using them. Therefore, future research must aim at filling this gap in knowledge.

Next, the motivations for using SNSs were reviewed on the basis of needs and gratifications theory. In general, research suggests that SNSs are used for social purposes. Overall, the maintenance of connections to offline network members was emphasized rather than the establishment of new ties. With regards to this, SNS users sustain bridging social capital through a variety of heterogeneous connections to other SNS users. This appeared to be beneficial for them with regards to sharing knowledge and potential future possibilities related to employment and related areas. In effect, the

knowledge that is available to individuals via their social network can be thought of as "collective intelligence" [119].

Collective intelligence extends the mere idea of shared knowledge because it is not restricted to knowledge shared by all members of a particular community. Instead, it denotes the aggregation of each individual member's knowledge that can be accessed by other members of the respective community. In this regard, the pursuit of weak ties on SNSs is of great benefit and thus coincides with the satisfaction of the members' needs. At the same time, it is experienced as gratifying. Therefore, rather than seeking emotional support, individuals make use of SNSs in order to communicate and stay in touch not only with family and friends, but also with more distant acquaintances, therefore sustaining weak ties with potentially advantageous environments. The benefits of large online social networks may potentially lead people to excessively engage in using them, which, in turn, may purport addictive behaviors.

As regards personality psychology, certain personality traits were found to be associated with higher usage frequency that may be associated with potential abuse and/or addiction. Of those, extraversion and introversion stand out because each of these is related to more habitual participation in social networks on the Internet. However, the motivations of extraverts and introverts differ in that extraverts enhance their social networks, whereas introverts compensate for the lack of real life social networks. Presumably, the motivations for higher SNS usage of people who are agreeable and conscientious may be related to those shared by extraverts, indicating a need for staying connected and socializing with their communities. Nevertheless, of those, high extraversion was found to be related to potential addiction to using SNS, in accordance with low conscientiousness [82].

The dissimilar motivations for usage found for members scoring high on the respective personality trait can inform future research into potential addiction to SNSs. Hypothetically, people who compensate for scarce ties with their real life communities may be at greater risk to develop addiction. In effect, in one study, addictive SNS usage was predicted by looking for a sense of belongingness in this community [83], which supports this conjecture. Presumably, the same may hold true for people who score high on neuroticism and narcissism, assuming that members of both groups tend to have low self-esteem. This supposition is informed by research indicating that people use the Internet excessively in order to cope with everyday stressors [120,121]. This may serve as a preliminary explanation for the findings regarding the negative correlates that were found to be associated with more frequent SNS usage.

Overall, the engagement in particular activities on SNSs, such as social searching, and the personality traits that were found to be associated with greater extents of SNS usage may serve as an anchor point for future studies in terms of defining populations who are at risk for developing addiction to using social networks on the Internet. Furthermore, it is recommended that researchers assess factors that are specific to SNS addiction, including the pragmatics, attraction, communication and expectations of SNS use because these may predict the etiology of SNS addiction as based on the addiction specificity etiology framework [15]. Due to the scarcity of research in this domain with a specific focus on SNS addiction specificity and comorbidity, further empirical research is necessary. Moreover, researchers are encouraged to pay close attention to the different motivations of introverts and extraverts because each of those appears to be related to higher usage frequency. What is more, investigating the relationship of potential addiction with narcissism seems to be a fruitful area for

empirical research. In addition to this, motivations for usage as well as a wider variety of negative correlates related to excessive SNS use need to be addressed.

In addition to the above mentioned implications and suggestions for future research, specific attention needs to be paid to selecting larger samples which are representative of a broader population in order to increase the respective study's external validity. The generalizability of results is essential in order to demarcate populations at risk for developing addiction to SNSs. Similarly, it appears necessary to conduct further psychophysiological studies in order to assess the phenomenon from a biological perspective. Furthermore, clear-cut and validated addiction criteria need to be assessed. It is insufficient to limit studies into addiction to assessing just a few criteria. The demarcation of pathology from high frequency and problematic usage necessitates adopting frameworks that have been established by the international classification manuals [18,102]. Moreover, in light of clinical evidence and practice, it appears essential to pay attention to the significant impairment that SNS addicts experience in a variety of life domains as a consequence of their abusive and/or addictive behaviors.

Similarly, the results of data based on self-reports are not sufficient for diagnosis because research suggests that they may be inaccurate [122]. Conceivably, self-reports may be supplemented with structured clinical interviews [123] and further case study evidence as well as supplementary reports from the users' significant others. In conclusion, social networks on the Internet are iridescent Web 2.0 phenomena that offer the potential to become part of, and make use of, collective intelligence. However, the latent mental health consequences of excessive and addictive use are yet to be explored using the most rigorous scientific methods.

References

1. Cohen, E. *Five Clues that You Are Addicted to Facebook*; CNN Health: Atlanta, GA, USA, 2009; Available online: http://articles.cnn.com/2009-04-23/health/ep.facebook.addict_1_facebook-page-facebook-world-social-networking?_s=PM:HEALTH (accessed on 18 August 2011).
2. Webley, K. *It's Time to Confront Your Facebook Addiction*; Time Inc.: New York, NY, USA, 2011; Available online: http://newsfeed.time.com/2010/07/08/its-time-to-confront-your-facebook-addiction/ (accessed on 18 August 2011).
3. Hafner, K. *To Deal with Obsession, Some Defriend Facebook*; The New York Times Company: New York, NY, USA, 2009; Available online: http://www.nytimes.com/2009/12/21/technology/internet/21facebook.html (accessed on 18 August 2011).
4. Revoir, P. *Facebook to Blame for* "*Friendship Addiction*"; Associated Newspapers Ltd.: London, UK, 2008; Available online: http://www.dailymail.co.uk/sciencetech/article-1079633/Facebook-blame-friendship-addiction-women.html (accessed on 18 August 2011).
5. The Nielsen Company. *Global Audience Spends Two Hours More a Month on Social Networks than Last Year*; The Nielsen Company: New York, NY, USA, 2010; Available online: http://blog.nielsen.com/nielsenwire/global/global-audience-spends-two-hours-more-a-month-on-social-networks-than-last-year/ (accessed 18 August 2011).
6. Griffiths, M. Internet addiction—Time to be taken seriously? *Addict. Res.* **2000**, *8*, 413-418.
7. Young, K. Internet addiction: Evaluation and treatment. *Student Brit. Med. J.* **1999**, *7*, 351-352.

8. Young, K. *Facebook Addiction Disorder?*; The Center for Online Addiction: Bradford, PA, USA, 2009; Available online: http://www.netaddiction.com/index.php?option=com_blog&view=comments&pid=5&Itemid=0 (accessed on 29 November 2010).
9. Boyd, D.M.; Ellison, N.B. Social network sites: Definition, history, and scholarship. *J. Comput. Mediat. Comm.* **2008**, *13*, 210-230.
10. Jenkins, H. *Convergence Culture. Where Old and New Media Collide*; New York University Press: New York, NY, USA, 2006.
11. Milgram, S. The small world problem. *Psychol. Today* **1967**, *2*, 60-67.
12. The Nielsen Company. *Global Faces and Networked Places*; The Nielsen Company: New York, NY, USA, 2009; Available online: http://blog.nielsen.com/nielsenwire/wp-content/uploads/2009/03/nielsen_globalfaces_mar09.pdf (accessed on 18 August 2011).
13. Rheingold, H. *The Virtual Community: Homesteading on the Electronic Frontier*; MIT: Cambridge, MA, USA, 1993.
14. Li, L. Exploration of adolescents' Internet addiction. *Psychol. Dev. Educ.* **2010**, *26*; Available online: http://en.cnki.com.cn/Article_en/CJFDTOTAL-XLFZ201005019.htm (accessed on 16 Feburary 2011).
15. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for specificity of the addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
16. Griffiths, M.D. A "components" model of addiction within a biopsychosocial framework. *J. Subst. Use* **2005**, *10*, 191-197.
17. Shaffer, H.J.; LaPlante, D.A.; LaBrie, R.A.; Kidman, R.C.; Donato, A.N.; Stanton, M.V. Toward a syndrome model of addiction: Multiple expressions, common etiology. *Harvard Rev. Psychiat.* **2004**, *12*, 367-374.
18. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders—Text Revision, Fourth Edition*; American Psychiatric Association: Washington, DC, USA, 2000.
19. Lenhart, A. *Social Networking Websites and Teens: An Overview*; Pew Research Center: Washington, DC, USA, 2007; Available online: http://www.pewinternet.org/~/media//Files/Reports/2007/PIP_SNS_Data_Memo_Jan_2007.pdf.pdf (accessed on 27 November 2010).
20. Subrahmanyam, K.; Reich, S.M.; Waechter, N.; Espinoza, G. Online and offline social networks: Use of social networking sites by emerging adults. *J. Appl. Dev. Psychol.* **2008**, *29*, 420-433.
21. Pempek, T.A.; Yermolayeva, Y.A.; Calvert, S.L. College students' social networking experiences on Facebook. *J. Appl. Dev. Psychol.* **2009**, *30*, 227-238.
22. Raacke, J.; Bonds-Raacke, J. MySpace and facebook: Applying the uses and gratifications theory to exploring friend-networking sites. *CyberPsychol. Behav.* **2008**, *11*, 169-174.
23. Pfeil, U.; Arjan, R.; Zaphiris, P. Age differences in online social networking—A study of user profiles and the social capital divide among teenagers and older users in MySpace. *Comput. Hum. Behav.* **2009**, *25*, 643-654.
24. Fogel, J.; Nehmad, E. Internet social network communities: Risk taking, trust, and privacy concerns. *Comput. Hum. Behav.* **2009**, *25*, 153-160.

25. Jelicic, H.; Bobek, D.L.; Phelps, E.; Lerner, R.M.; Lerner, J.V. Using positive youth development to predict contribution and risk behaviors in early adolescence: Findings from the first two waves of the 4-H Study of Positve Youth Development. *Int. J. Behav. Dev.* **2007**, *31*, 263-273.
26. Wilkinson, D.; Thelwall, M. Social network site changes over time: The case of MySpace. *J. Am. Soc. Inf. Sci. Tech.* **2010**, *61*, 2311-2323.
27. Wise, K.; Alhabash, S.; Park, H. Emotional responses during social information seeking on Facebook. *Cyberpsychol. Behav. Soc. Network.* **2010**, *13*, 555-562.
28. Lang, A.; Potter, R.F.; Bolls, P.D. Where psychophysiology meets the media: Taking the effects out of mass communication research. In *Media Effects: Advances in Theory and Research*; Bryant, J., Oliver, M.B., Eds.; Routledge Taylor and Francis Group: New York, NY, USA, 2009; pp. 185-206.
29. Park, H.S.; Kim, S.H.; Bang, S.A.; Yoon, E.J.; Cho, S.S.; Kim, S.E. Altered regional cerebral glucose metabolism in Internet game overusers: A F-18-fluorodeoxyglucose Positron Emission Tomography study. *CNS Spectr.* **2010**, *15*, 159-166.
30. Ko, C.H.; Liu, G.C.; Hsiao, S.M.; Yen, J.Y.; Yang, M.J.; Lin, W.C.; Yen, C.F.; Chen, C.S. Brain activities associated with gaming urge of online gaming addiction. *J. Psychiat. Res.* **2009**, *43*, 739-747.
31. Comings, D.E.; Blum, K. Reward deficiency syndrome: Genetic aspects of behavioral disorders. In *Cognition, Emotion and Autonomic Responses: The Integrative Role of the Prefrontal Cortex and Limbic Structures*; Uylings, H.B.M., VanEden, C.G., DeBruin, J.P.C., Feenstra, M.G.P., Pennartz, C.M.A., Eds.; Elsevier Science: Amsterdam, The Netherlands, 2000; Vol. 126, pp. 325-341.
32. Griffiths, M. Internet gambling: Issues, concerns, and recommendations. *CyberPsychol. Behav.* **2003**, *6*, 557-568.
33. Ross, C.; Orr, E.S.; Sisic, M.; Arseneault, J.M.; Simmering, M.G.; Orr, R.R. Personality and motivations associated with Facebook use. *Comput. Hum. Behav.* **2009**, *25*, 578-586.
34. Katz, E.; Blumler, J.; Gurevitch, M. Uses of mass communications by the individual. In *Mass Communication Research: Major Issues and Future Directions*; Davidson, W., Yu, F., Eds.; Praeger: New York, NY, USA, 1974; pp. 11-35.
35. Kwon, O.; Wen, Y. An empirical study of the factors affecting social network service use. *Comput. Hum. Behav.* **2010**, *26*, 254-263.
36. Kim, J.H.; Kim, M.S.; Nam, Y. An analysis of self-construals, motivations, facebook use, and user satisfaction. *Int. J. Hum-Comput. Int.* **2010**, *26*, 1077-1099.
37. Barker, V. Older adolescents' motivations for social network site use: The influence of gender, group identity, and collective self-esteem. *CyberPsychol. Behav.* **2009**, *12*, 209-213.
38. Cheung, C.M.K.; Chiu, P.-Y.; Lee, M.K.O. Online social networks: Why do students use facebook? *Comput. Hum. Behav.* **2010**, *27*, 1337-1343.
39. Kujath, C.L. Facebook and MySpace: Complement or substitute for face-to-face interaction? *Cyberpsychol. Behav. Soc. Network.* **2011**, *14*, 75-78.
40. Walther, J.B. Computer-mediated communication: Impersonal, interpersonal, and hyperpersonal interaction. *Commun. Res.* **1996**, *23*, 3-43.

41. Crystal, D. The scope of Internet linguistics. In *Proceedings of American Association for the Advancement of Science Conference*; American Association for the Advancement of Science Conference, Washington, DC, USA, 17–21 February 2005; American Association for the Advancement of Science: Washington, DC, USA; Available online: http://www.davidcrystal.com/DC_articles/Internet2.pdf (accessed on 18 August 2011).
42. Thurlow, C. The Internet and language. In *Concise Encyclopeia of Sociolinguistics*; Mesthrie, R., Asher, R., Eds.; Pergamon: London, UK, 2001; pp. 287-289.
43. Ellison, N.B.; Steinfield, C.; Lampe, C. The benefits of Facebook "friends": Social capital and college students' use of online social network sites. *J. Comput-Mediat. Comm.* **2007**, *12*; Avaiable online: http://jcmc.indiana.edu/vol12/issue4/ellison.html (accessed on 18 August 2011).
44. Bourdieu, P.; Wacquant, L. *An Invitation to Reflexive Sociology*; University of Chicago Press: Chicago, IL, USA, 1992.
45. Putnam, R.D. *Bowling Alone*; Simon & Schuster: New York, NY, USA, 2000.
46. Wellman, B.; Gulia, M. The network basis of social support: A network is more than the sum of its ties. In *Networks in the Global Village*; Wellman, B., Ed.; Westview: Boulder, CO, USA, 1999.
47. Donath, J.; Boyd, D. Public displays of connection. *BT Technol. J.* **2004**, *22*, 71-82.
48. Reich, S.M. Adolescents' sense of community on MySpace and facebook: A mixed-methods approach. *J. Community Psychol.* **2010**, *38*, 688-705.
49. Steinfield, C.; Ellison, N.B.; Lampe, C. Social capital, self-esteem, and use of online social network sites: A longitudinal analysis. *J. Appl. Dev. Psychol.* **2008,** *29*, 434-445.
50. Armstrong, L.; Phillips, J.G.; Saling, L.L. Potential determinants of heavier internet usage. *Int. J. Hum-Comput. St.* **2000**, *53*, 537-550.
51. Ghassemzadeh, L.; Shahraray, M.; Moradi, A. Prevalence of Internet addiction and comparison of Internet addicts and non-addicts in Iranian high schools. *CyberPsychol. Behav.* **2008**, *11*, 731-733.
52. Ji, Y.G.; Hwangbo, H.; Yi, J.S.; Rau, P.L.P.; Fang, X.W.; Ling, C. The influence of cultural differences on the use of social network services and the formation of social capital. *Int. J. Hum-Comput. Int.* **2010**, *26*, 1100-1121.
53. Sledgianowski, D.; Kulviwat, S. Using social network sites: The effects of playfulness, critical mass and trust in a hedonic context. *J. Comput. Inform. Syst.* **2009**, *49*, 74-83.
54. Livingstone, S. Taking risky opportunities in youthful content creation: Teenagers' use of social networking sites for intimacy, privacy and self-expression. *New Media Soc.* **2008**, *10*, 393-411.
55. Kim, Y.; Sohn, D.; Choi, S.M. Cultural difference in motivations for using social network sites: A comparative study of American and Korean college students. *Comput. Hum. Behav.* **2011**, *27*, 365-372.
56. Zywica, J.; Danowski, J. The faces of Facebookers: Investigating social enhancement and social compensation hypotheses: Predicting Facebook and offline popularity from sociability and self-esteem, and mapping the meanings of popularity with semantic networks. *J. Comput-Mediat. Comm.* **2008**, *14*, 1-34.
57. Lee, G.; Lee, J.; Kwon, S. Use of social-networking sites and subjective well-being: A study in South Korea. *Cyberpsychol. Behav. Soc. Network.* **2011**, *14*, 151-155.

58. Pollet, T.V.; Roberts, S.G.B.; Dunbar, R.I.M. Use of social network sites and instant messaging does not lead to increased offline social network size, or to emotionally closer relationships with offline network members. *Cyberpsychol. Behav. Soc. Network.* **2011**, *14*, 253-258.
59. Mehdizadeh, S. Self-presentation 2.0: Narcissism and self-esteem on facebook. *Cyberpsychol. Behav. Soc. Network.* **2010**, *13*, 357-364.
60. Buffardi, E.L.; Campbell, W.K. Narcissism and social networking web sites. *Pers. Soc. Psychol. B.* **2008**, *34*, 1303-1314.
61. Zhao, S.Y.; Grasmuck, S.; Martin, J. Identity construction on Facebook: Digital empowerment in anchored relationships. *Comput. Hum. Behav.* **2008**, *24*, 1816-1836.
62. Manago, A.M.; Graham, M.B.; Greenfield, P.M.; Salimkhan, G. Self-presentation and gender on MySpace. *J. Appl. Dev. Psychol.* **2008**, *29*, 446-458.
63. Campbell, W.K.; Bosson, J.K.; Goheen, T.W.; Lakey, C.E.; Kernis, M.H. Do narcissists dislike themselves "deep down inside"? *Psychol. Sci.* **2007**, *18*, 227-229.
64. Cain, N.M.; Pincus, A.L.; Ansell, E.B. Narcissism at the crossroads: Phenotypic description of pathological narcissism across clinical theory, social/personality psychology, and psychiatric diagnosis. *Clin. Psychol. Rev.* **2008**, *28*, 638-656.
65. La Barbera, D.; La Paglia, F.; Valsavoia, R. Social network and addiction. *Cyberpsychol. Behav.* **2009**, *12*, 628-629.
66. Correa, T.; Hinsley, A.W.; de Zuniga, H.G. Who interacts on the Web?: The intersection of users' personality and social media use. *Comput. Hum. Behav.* **2010**, *26*, 247-253.
67. Amichai-Hamburger, Y.; Vinitzky, G. Social network use and personality. *Comput. Hum. Behav.* **2010**, *26*, 1289-1295.
68. Costa, P.T.; McCrae, R.R. *Revised NEO Personality Inventory (NEO-PI-R) and the NEO Five-Factor Inventory (NEO-FFI): Professional Manual*; Psychological Assessment Resources: Odessa, FL, USA, 1992.
69. Orr, E.S.; Ross, C.; Simmering, M.G.; Arseneault, J.M.; Orr, R.R. The influence of shyness on the use of Facebook in an undergraduate sample. *CyberPsychol. Behav.* **2009**, *12*, 337-340.
70. Valkenburg, P.M.; Peter, J.; Schouten, A.P. Friend networking sites and their relationship to adolescents' well-being and social self-esteem. *CyberPsychol. Behav.* **2006**, *9*, 584-590.
71. Nyland, R.; Marvez, R.; Beck, J. MySpace: Social networking or social isolation? In *Proceedings of the Midwinter Conference of the Association for Education in Journalism and Mass Communication*, Midwinter Conference of the Association for Education in Journalism and Mass Communication, Reno, NV, USA, 23–24 February 2007.
72. Suler, J. The online disinhibition effect. *CyberPsychol. Behav.* **2004**, *7*, 321-326.
73. Kirschner, P.A.; Karpinski, A.C. Facebook and academic performance. *Comput. Hum. Behav.* **2010**, *26*, 1237-1245.
74. Phillips, M. *MySpace or Yours? Social Networking Sites Surveillance in Romantic Relationships*; Western States Communication Association: Mesa, AZ, USA, 2009.
75. Tokunaga, R.S. Social networking site or social surveillance site? Understanding the use of interpersonal electronic surveillance in romantic relationships. *Comput. Hum. Behav.* **2011**, *27*, 705-713

76. Muise, A.; Christofides, E.; Desmarais, S. More information than you ever wanted: Does facebook bring out the green-eyed monster of jealousy? *CyberPsychol. Behav.* **2009**, *12*, 441-444.
77. Persch, J.A. *Jealous Much? MySpace, Facebook Can Spark It*; The Msnbc Digital Network: New York, NY, USA, 2007; Available online: http://www.msnbc.msn.com/id/20431006/ (accessed on 18 August 2011).
78. Luscombe, B. Social norms: Facebook and divorce. *Time* **2009**, *173*, 93-94.
79. Grüsser, S.M.; Thalemann, C.N. *Verhaltenssucht—Diagnostik, Therapie, Forschung*; Hans Huber: Bern, Germany, 2006.
80. Kuntsche, E.; Stewart, S.H.; Cooper, M.L. How stable is the motive-alcohol use link? A cross-national validation of the drinking motives questionnaire revised among adolescents from Switzerland, Canada, and the United States. *J. Stud. Alcohol Drugs* **2008**, *69*, 388-396.
81. Echeburua, E.; de Corral, P. Addiction to new technologies and to online social networking in young people: A new challenge. *Adicciones* **2010**, *22*, 91-95.
82. Wilson, K.; Fornasier, S.; White, K.M. Psychological predictors of young adults' use of social networking sites. *Cyberpsychol. Behav. Soc. Network.* **2010**, *13*, 173-177.
83. Pelling, E.L.; White, K.M. The theory of planned behavior applied to young people's use of social networkting web sites. *CyberPsychol. Behav.* **2009**, *12*, 755-759.
84. Karaiskos, D.; Tzavellas, E.; Balta, G.; Paparrigopoulos, T. Social network addiction: A new clinical disorder? *Eur. Psychiat.* **2010**, *25*, 855.
85. Zhou, S.X. Gratifications, loneliness, leisure boredom and self-esteem as predictors of SNS-game addiction and usage pattern among Chinese college students. M.S. Thesis, Chinese University of Hong Kong: Hong Kong, China, 2010.
86. Wan, C. Gratifications & loneliness as predictors of campus-SNS websites addiction & usage pattern among Chinese college students. M.S. Thesis, Chinese University of Hong Kong: Hong Kong, China, 2009.
87. Ajzen, I. The theory of planned behavior. *Organ. Behav. Hum. Dec.* **1991**, *50*, 179-211.
88. Terry, D.H.M.; White, K. The theory of planned behavior: Self-identity, social identity and group norms. *Brit. J. Soc. Psychol.* **1999**, *38*, 225-244.
89. Baumeister, R.; Leary, M. The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychol. Bull.* **2005,** *117*, 497-529.
90. Ehrenberg, A.; Juckes, S.; White, K.M.; Walsh, S.P. Personality and self-esteem as predictors of young people's technology use. *CyberPsychol. Behav.* **2008**, *11*, 739-741.
91. Costa, P.T.; McCrae, R.R. *NEO PI-R Professional Manual*; Psychological Assessment Resources: Odessa, TX, USA, 1992.
92. Coopersmith, S. *Self-esteem Inventories*; Consulting Psychologists Press: Palo Alto, CA, USA, 1981.
93. Walsh, S.P.; White, K.M.; Young, R.M. Young and connected: Psychological influences of mobile phone use amongst Australian youth. In *Mobile Media 2007*, Proceedings of an International Conference on Social and Cultural Aspects of Mobile Phones, Media, and Wireless Technologies, Sydney, Australia, 2–4 July 2007; Goggin, G., Hjorth, L., Eds.; University of Sydney: Sydney, Australia, 2007; pp. 125-134.

94. Landers, R.N.; Lounsbury, J.W. An investigation of Big Five and narrow personality traits in relation to Internet usage. *Comput. Hum. Behav.* **2004**, *22*, 283-293.
95. Kuss, D.J.; Griffiths, M.D. Internet gaming addiction: A systematic review of empirical research. *Int. J. Ment. Health Addict.* **2011**, *in press*.
96. Kuss, D.J.; Griffiths, M.D. Internet gambling behavior. In *Encyclopedia of Cyber Behavior*; IGI Global: Hershey, PA, USA, 2011, *in press*.
97. Kuss, D.J.; Griffiths, M.D. Internet sex addiction: A review of empirical research. *Addict. Res. Theory* **2011**, *in press*.
98. Young, K. Internet addiction: The emergence of a new clinical disorder. *CyberPsychol. Behav.* **1996**, *3*, 237-244.
99. Russell, D.; Peplau, L.A.; Cutrona, C.E. The revised UCLA Loneliness Scale: Concurrent and discriminant validity evidence. *J. Pers. Soc. Psychol.* **1980**, *39*, 472-480.
100. Iso-Ahola, S.E.; Weissinger, E. Receptions of boredom in leisure: Conceptualization, reliability and validity of the leisure boredom scale. *J. Leisure Res.* **1990**, *22*, 1-17.
101. Rosenberg, M.; Schooler, C.; Schoenbach, C. Self-esteem and adolescent problems: Modeling reciprocal effects. *Am. Sociol. Rev.* **1989**, *54*, 1004-1018.
102. World Health Organization (WHO). *ICD 10: The ICD-10 Classification of Mental and Behavioral Disorders: Clinical Descriptions and Diagnostic Guidelines*; WHO: Geneva, Switzerland, 1992.
103. Hall, W.; Degenhardt, L.; Teesson, M. Understanding comorbidity between substance use, anxiety and affective disorders: Broadening the research base. *Addict. Behav.* **2009**, *34*, 795-799.
104. Malat, J.; Collins, J.; Dhayanandhan, B.; Carullo, F.; Turner, N.E. Addictive behaviors in comorbid addiction and mental illness: Preliminary results from a self-report questionnaire. *J. Addict. Med.* **2010**, *4*, 38-46.
105. Black, D.W.; Belsare, G.; Schlosser, S. Clinical features, psychiatric comorbidity, and health-related quality of life in persons reporting compulsive computer use behavior. *J. Clin. Psychiat.* **1999**, *60*, 839-844.
106. Müller, K.W.; Dickenhorst, U.; Medenwaldt, J.; Wölfling, K.; Koch, A. Internet addiction as comorbid disorder in patients with a substance-related disorder: Results from a survey in different inpatient clinics. *Eur. Psychiat.* **2011**, *26,* 1912.
107. Yen, J.Y.; Yen, C.F.; Chen, C.C.; Chen, S.H.; Ko, C.H. Family factors of Internet addiction and substance use experience in Taiwanese adolescents. *CyberPsychol. Behav.* **2007**, *10*, 323-329.
108. Lam, L.T.; Peng, Z.W.; Mai, J.C.; Jing, J. Factors associated with Internet addiction among adolescents. *CyberPsychol. Behav.* **2009**, *12*, 551-555.
109. Young, K. *Caught in the Net*. Wiley: New York, NY, USA, 1998.
110. Kuntsche, E.; Knibbe, R.; Gmel, G.; Engels, R. Replication and validation of the drinking motive questionnaire revised (DMQ-R, Cooper, 1994) among adolescents in Switzerland. *Eur. Addict. Res.* **2006**, *12*, 161-168.
111. Ko, C.H.; Yen, J.Y.; Chen, C.C.; Chen, S.H.; Wu, K.; Yen, C.F. Tridimensional personality of adolescents with Internet addiction and substance use experience. *Can. J. Psychiat.* **2006**, *51*, 887-894.

112. Downs, C. The Facebook phenomenon: Social networking and gambling. In *Proceeding of the Gambling and Social Responsibility Forum Conference*, Manchester, UK, 2–3 September 2008; Manchester Metropolitan University: Manchester, UK, 2008.
113. Griffiths, M.D.; King, D.L.; Delfabbro, P.H. Adolescent gambling-like experiences: Are they a cause for concern? *Educ. Health* **2009**, *27*, 27-30.
114. Ipsos MORI. *British Survey of Children, the National Lottery and Gambling 2008–2009. Report of A Quantitative Survey*; National Lottery Commission: Salford, UK, 2009.
115. Griffiths, M.D.; Parke, J. Adolescent gambling on the Internet: A review. *Int. J. Adol. Med. Health* **2010**, *22*, 58-75.
116. King, D.; Delfabbro, P.; Griffiths, M. The convergence of gambling and digital media: Implications for gambling in young people. *J. Gambl. Stud.* **2010**, *26*, 175-187.
117. Griffiths, M.D. Gaming in social networking sites: A growing concern? *World Online Gambl. Law Rep.* **2010**, *9*, 12-13.
118. Fogel, J.; Nehmad, E. Internet social network communities: Risk taking, trust, and privacy concerns. *Comput. Hum. Behav.* **2009**, *25*, 153-160.
119. Lévy, P. *Collective Intelligence: Mankind's Emerging World in Cyberspace*; Perseus: Cambridge, MA, USA, 1997.
120. Batthyány, D.; Müller, K.W.; Benker, F.; Wölfling, K. Computer game playing: Clinical characteristics of dependence and abuse among adolescents. *Wiener Klinsche Wochenschrift* **2009**, *121*, 502-509.
121. Wölfling, K.; Grüsser, S.M.; Thalemann, R. Video and computer game addiction. *Int. J. Psychol.* **2008**, *43*, 769-769.
122. Bhandari, A.; Wagner, T.H. *Self-report Utilization: Improving Measurement and Accuracy*; US National Institutes of Health: San Diego, CA, USA, 2004.
123. Beard, K.W. Internet addiction: A review of current assessment techniques and potential assessment questions. *CyberPsychol. Behav.* **2005**, *8*, 7-14.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Pentz, M.A.; Spruijt-Metz, D.; Chou, C.P.; Riggs, N.R. High Calorie, Low Nutrient Food/Beverage Intake and Video Gaming in Children as Potential Signals for Addictive Behavior. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4406-4424; doi:10.3390/ijerph8124406.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

High Calorie, Low Nutrient Food/Beverage Intake and Video Gaming in Children as Potential Signals for Addictive Behavior

Mary Ann Pentz *, Donna Spruijt-Metz, Chih Ping Chou and Nathaniel R. Riggs

Department of Preventive Medicine, Institute for Prevention Research, University of Southern California, 2001 N. Soto Street, Los Angeles, CA 90033, USA; E-Mails: dmetz@usc.edu (D.S.-M.); cchou@usc.edu (C.P.C.); nriggs@usc.edu (N.R.R.)

* Author to whom correspondence should be addressed; E-Mail: pentz@usc.edu; Tel.: +1-323-442-8227; Fax: +1-323-442-7264.

Received: 28 September 2011; in revised form: 22 November 2011 / Accepted: 22 November 2011 / Published: 29 November 2011

Abstract: Little is known about the co-occurrence of health risk behaviors in childhood that may signal later addictive behavior. Using a survey, this study evaluated high calorie, low nutrient HCLN intake and video gaming behaviors in 964 fourth grade children over 18 months, with stress, sensation-seeking, inhibitory control, grades, perceived safety of environment, and demographic variables as predictors. SEM and growth curve analyses supported a co-occurrence model with some support for addiction specificity. Male gender, free/reduced lunch, low perceived safety and low inhibitory control independently predicted both gaming and HCLN intake. Ethnicity and low stress predicted HCLN. The findings raise questions about whether living in some impoverished neighborhoods may contribute to social isolation characterized by staying indoors, and HCLN intake and video gaming as compensatory behaviors. Future prevention programs could include skills training for inhibitory control, combined with changes in the built environment that increase safety, e.g., implementing Safe Routes to School Programs.

Keywords: eating; video gaming; children; addictive behavior

Int. J. Environ. Res. Public Health **2011**, *8*

1. Introduction

As discussed in the first paper of this special issue of the *International Journal of Environmental Research and Public Health*, health risk behaviors which are potentially addictive follow a particular course, from initial appetite for the behavior, preoccupation with the behavior, loss of control over the behavior, negative consequences of continued behavioral practice, and lack of ability to stop the behavior [1]. DSM IV captures this progression in its diagnostic criteria for addictive behavior, and classifies actual addiction as extreme repetitive practice of a behavior to the extent that normal daily living functions are disrupted and the behavior produces harm to the individual [2]. DSM IV also considers co-morbidities associated with addiction. Whether separate addictions are co-occurring, or even co-predictive, is another question and one that is being addressed in the various papers in this issue.

Historically, addictions have been examined as addictions to substance use, *i.e.*, tobacco, alcohol, and/or other drug use [3]. However, there is growing evidence that other health risk behaviors may also exhibit addiction "propensity." In a review of 83 studies, Sussman *et al.* [4] concluded that eight behaviors in addition to substance use could be potentially addictive: binge eating, gambling, internet use, love, sex, exercise, work, and shopping. Video gaming has also gained recent attention as a potentially addictive behavior [5-7]. At least two of these potentially addictive behaviors, frequent or excessive high calorie, low nutrient (HCLN) food/beverage intake (which could relate to binge eating) and video gaming (a sedentary activity) have particular relevance to obesity, one of the foremost and escalating health problems in the world today [8-11].

1.1. Addictive High Calorie, Low Nutrient Food Intake and Video Gaming as Risk Factors for Obesity

The significant, global rise in obesity rates poses multiple and costly problems for health and society, including increased risk for cancer, heart disease, and diabetes, among other diseases [8]. Excessive HCLN intake, *i.e.*, high fat, high sugar snack foods and beverages, and video gaming (as part of sedentary behavior) have been shown to be significant risk factors for obesity [9-12]. Identifying the developmental course of HCLN intake and video gaming *before* these behaviors become addictive, and sufficiently early in life, could have important implications then for preventing both obesity and substance use.

1.2. Obesity Risk in Childhood

Obesity risk escalates during the childhood years, commensurate with stages of development that are associated with adiposity rebound [13]. One of these stages is childhood [14]. HCLN intake has been shown to be a predictor of obesity at this age and later in life [14]. Sedentary behavior is another predictor of obesity in childhood [10,13]. Video gaming is considered one of several types of sedentary behavior that is usually evaluated together with television viewing, internet use, and inactivity [11,12,15]. There is relatively little research on the potential addictive progression of video gaming as a separate sedentary behavior in childhood. One of the few studies, conducted on students in grades 3,4,7 and 8, showed that increased frequency of gaming, low social competence, and high

levels of impulsivity were associated with progression to pathological gaming over a two year period [5]. However, whether progression differed by grade level or age was not reported.

No studies have been reported on addictive progression of HCLN intake in children, and none on the potential co-occurrence of video gaming and HCLN intake as addictive behaviors in this age group. Additionally, while there is some research on predictors of early sedentary behavior and HCLN intake frequency in children, including parental modeling, family rules, and feeding patterns, there is no research on predictors of *growth* in these behaviors in this age group that might signal addiction propensity (see [9,13]). Identifying predictors of addiction propensity for these behaviors in children, as well as addictive progression of these behaviors, could have significant implications for designing early addiction prevention programs.

1.3. Predictors of Video Gaming and High Calorie, Low Nutrient Food Intake in Children

With virtually no studies available on predictors of propensity for addictive video gaming or HCLN intake in children, identifying potential predictors depends on findings from addiction studies on other age groups, most prominently, adolescents, and on other addictive behaviors that may share common risk factors with video gaming and high calorie, low nutrient food intake, most prominently, substance use. These include, but are not limited to: *early or frequent substance use relative to peers, high sensation-seeking, low impulse control, high stress, poor coping sills, poor school achievement, male gender, white race/ethnicity, and low socioeconomic status.*

An extensive body of research on adolescents has shown that early and frequent substance use, high sensation-seeking, and low impulse control consistently predict progression of substance use [16-22]. Increasingly, low impulse control has been interpreted as a deficit in one aspect of a larger set of cognitive-emotional functions referred to as executive cognitive function (ECF, [22-24]), which is linked with brain neurocircuitry [25-27]. Animal and human studies on adults have shown that physical obesity and substance use share common risk pathways through at least two areas of the brain: the pre-frontal cortex, particularly as relates to affective decision-making involving arousal and craving; and the nucleus acumens, particularly as relates to delay of reward or reinforcement of a behavior [28-33]. Inability to manage arousal and delay immediate reinforcement is observed as problems in ECF, more specifically as an inhibitory control deficit [34,35].

Other predictors have also shown relationships to adolescent substance use, particularly with progression from experimental to more regular, and potentially, more addictive use. Among these are high stress and low ability to cope with anxiety, and poor school achievement [28,36,37]. Demographic variables of gender, race/ethnicity, and socioeconomic status have also been shown to relate to substance use, although the direction of relationships varies somewhat by type of substance used [38,39].

Several of the risk factors found to predict adolescent substance use have also been found to predict obesity risk in children, if not specifically HCLN intake or video gaming. For example, obesity risk status, based on anthropometric measures of body mass index or waist circumference, is positively related to Hispanic and/or African-American race/ethnicity, gender (female, especially after puberty; male Hispanic), low socioeconomic status, inhibitory control deficit, poor grades, stress, and low coping ability [40-44]. Whether these variables also predict video gaming and high calorie, low

nutrient food intake in children is not yet clear, although our previous study of latent classes of obesity risk indicated that in high obesity risk classes, male and Hispanic children were more likely than female and white children to engage in both HCLN and sedentary behavior (measured as total screen time) [40]. The Gentile *et al*. study [5] found that in the mixed sample of children and adolescents, progression to pathological gaming was associated with male gender, as well as later increases in anxiety/stress, depression, and social phobia—a potential indicator of social isolation—and decreased school achievement.

1.4. Objectives of the Present Study

In keeping with the intent of this special issue on co-occurring and co-morbid addictive behaviors, the present study tested four hypotheses pertaining to video gaming and high calorie, low nutrient food intake as potentially addictive behaviors in children. The first hypothesis was that video gaming and high calorie, low nutrient food intake would grow over time. The second hypothesis was that both video gaming and HCLN would co-occur within individuals. The third hypothesis was that both outcomes would have a common set of risk factors, based on variables derived from previous research on adolescent substance use and child obesity risk. The common risk factors were hypothesized to be inhibitory control problems, male gender, Hispanic race/ethnicity, low socioeconomic status, poor grades, high stress, and poor coping. The fourth hypothesis was that there would be some risk factors that differentiate the two behaviors, signaling potential addiction specificity. This hypothesis was exploratory, since there is relatively little research in children on whether specificity of risk factors would take the form of a difference in strength of risk or an opposite relationship of risk.

2. Methods

2.1. Background

The present study used data from the baseline, six month follow-up, and 18-month follow-up waves of measurement in a randomized controlled trial for prevention of substance use and obesity in children, Pathways for Health, hereafter referred to as Pathways. The objective of Pathways is to translate two evidence-based programs for violence and substance use prevention, the Midwestern Prevention Project, or STAR [45], and PATHS [46] to a substance use and obesity prevention program for children, targeting schools that might represent higher risk for obesity by virtue of their Hispanic/Latino and low socioeconomic status representation. A total of 28 elementary schools in Southern California, including Title 12 schools (receiving federal aid) and schools with higher proportions of Hispanic/Latino students, from two Southern California school districts were matched in pairs on school-level demographic characteristics of achievement, size, ethnicity (% Hispanic/Latino), and socioeconomic status (% on free/reduced lunch) using the SAS RANUNI function [47] and then randomly assigned from within each pair to a school and parent-based program or control. Matching and randomization were conducted within each school district.

Int. J. Environ. Res. Public Health **2011**, *8*

2.2. Participants

At baseline, participants were 1,005 fourth grade (mean age 9.27 years) students from all 85 classrooms and 28 schools who had full active parent and self-consent for participation in the study. Of those 1,005, 96% (964) had complete data for study variables at baseline and constituted the current sample. Table 1 illustrates the sample characteristics for all study variables at baseline. Thirty-one percent were Caucasian, 27% were Hispanic/Latino and an additional, 8% Asian, 3% African American, 31% were either Hispanic multi-racial or "other." Fifty percent were male and 25% reported receiving a free lunch at school.

2.3. Measures

Participants completed a survey consisting of 145-items. The survey was administered aloud by a trained data collector, with a second data collector available to answer individual student questions about comprehension. Common to many school-based studies (e.g., [15]) data collection was constrained to one class period of approximately 45 minutes. Due to constraints of time, and possible constraints of comprehension and attention of fourth grade children, longer measures that had been previously developed and validated on adolescents were abbreviated in length, adapted for fourth grade reading comprehension, and re-validated. There is support in the psychometric literature for using abbreviated scales [48] and the practical reality of school-based prevention research is that assessment tools must be administered within the restrictions of time for school-based assessment. All procedures were approved by the University of Southern California Institutional Review Board.

2.3.1. High Calorie Low Nutrient Food/Beverage (HCLN) Intake

HCLN intake was assessed with five items taken from a validated open-source food frequency questionnaire [49]. The choice to select a subset of items from this questionnaire was based on constraints of survey length, as well as factor loadings on one factor representing HCLN. The five items included: How often do you drink soda—not diet (one can or glass); eat French fries or fried potatoes; eat corn chips, potato chips, popcorn, or crackers; eat doughnuts, pastries, cake, cookies (not low-fat); eat candy (chocolate, hard candy, candy bars) were selected by project investigators who have used these items in previous studies [41,42] and were compared to school teacher reports of food/beverage intake of their students as well as results of the California Healthy Kids Survey [50]. Abbreviated versions of food frequency questionnaires have demonstrated validity for fourth grade youth [48] and these specific items have been used with younger populations [41]. Response choices were 1 (Less than once a week), 2 (Once a week), 3 (2–3 times a week), 4 (4–6 times a week), 5 (Once a day), and 6 (2 or more of these a day). Internal consistency for the five items was adequate ($\alpha = 0.80$).

2.3.2. Video Gaming

Video gaming items were selected from the School-Based Nutrition Monitoring Student Questionnaire (NMSQ; 12). The two items were "On a regular school day, how many hours per day do you usually spend playing video games that you sit down to play like PlayStation, Xbox, GameBoy, or

arcade games?" as well as "video games that make you move or breathe hard like Nintendo Wii?" Response choices ranged from 1 ("I don't play videogames") to 7 ("6 or more hours per day"). The mean of the two items was computed and the internal consistency for the two items was adequate (α = 0.74).

2.3.3. Inhibitory Control Problems

Items from the Inhibit clinical sub-scale of the Behavioral Rating Inventory of Executive Function, Self-Report [34] were included to assess inhibitory control (e.g., "I do things without thinking first"). Item response choices ranged from 1 = Never, 2 = Sometimes, 3 = Often. Previous pilot studies, including a study of 107 fourth grade students, have demonstrated acceptable internal consistency for the full Inhibit scale (α = 0.78) [24,42]. For the current study, an abbreviated scale was constructed using the six highest loading index items from our pilot data. The abbreviated scale demonstrated predictive validity when compared to the full BRIEF-SR scale (α = 0.74).

2.3.4. Stress and Coping

Eight index items were selected from the Perceived Stress Scale (PSS) [51] based on our pilot analyses. The PSS contains items that tap reactivity to stressors (e.g., in the past week "I felt nervous or stressed"), as well as aspects of an individual's capacity to cope with stressors (e.g., "I handled problems that bothered me"). A principal components analysis with promax rotation was conducted resulting in a two-factor (stress and poor coping) solution. The largest loading items were then selected to represent these factors. Internal consistencies were 0.59 and 0.67 for stress and low coping, respectively.

2.3.5. Sensation Seeking

Three items were adapted from the Brief Sensation Seeking Scale for children and adolescents (BSS4 and SS2; e.g., "I like to do things that are a little scary;" 50). Based on a pilot study of 107 fourth grade children [24], language and scaling were simplified for comprehension (e.g., "frightening" was replaced with "scary;" a five point response choice was replaced with a three point response choice; from not at all = 1, very often = 5 to never = 1, often = 3). Three items were dropped due to low loadings in factor analyses, leaving a three-item scale. Internal consistency was α = 0.42, comparable to reports on the BSS4 (α = 0.44; [50,52]).

2.3.6. Perceived Neighborhood Safety

Children's perceptions of neighborhood safety was assessed utilizing a single item from the Youth Risk Behavior Survey (YRBS) asking children "In the last month, have you ever not gone to school because you felt you would be unsafe at school or on your way to or from school?" [53,54]. Item response choices were reversed to represent 0 = no, 1 = yes.

Int. J. Environ. Res. Public Health **2011**, *8*

2.3.7. Covariates

Gender, ethnicity (African-American or Hispanic/Latino *vs*. Other), self-reported school grades, and socio-economic status (free lunch as proxy) were included as potential risk factors based on previous studies have shown some differences in sedentary activities and obesity risk by gender, ethnicity, and socioeconomic status [44,55].

2.4. Analysis Plan

All analyses were conducted using the individual as the unit of analysis and proceeded through a two-step process. Means and standard errors were computed in step one to describe the sample. In step two, growth curve analyses modeled relationships between independent variables and intercept and growth in HCLN intake and video gaming over an 18 month period (two school years, three waves of data).

Growth Curve Analysis

Group differences in two parameters, the intercept and slope, were estimated for a growth curve model (GCM). The intercept represents the starting status of an individual's use trajectory, from the first wave of data collection, in longitudinal observation. The linear slope represents the unidirectional trend of the change in high calorie snack food and video gaming across time. A GCM can be expressed as:

$$y_{ij} = a_j + t_{ij}b_j + e_{ij}$$

where y_{ij} represents the outcome measure for individual j at time i; t_{ij} is the time of measurement (e.g., $t_{ij} = i - 1$); while a_j and b_j stand for intercept and slope, respectively, and e_{ij} is normally distributed with mean 0 and variance $\sigma^2 e$. It is a_j and b_j that characterize the growth profile of an individual.

Intercept and growth profiles were simultaneously estimated for HCLN intake and video gaming using Mplus 6.1 software with full information maximum likelihood imputation [56]. Mplus has the capacity to conduct analysis of complex survey data and yields standard errors and a chi-square test of model fit. Model fit indices for exploratory hypothesis testing included Chi-Square, Root Mean Square Error of Approximation (RMSEA), and Comparative Fit Index (CFI). Relationships among the two behaviors were modeled within each wave to evaluate a co-occurrence model, as well as across behaviors to evaluate a co-prediction model.

3. Results

3.1. Descriptive Characteristics

Demographic and behavioral characteristics of the sample at fourth grade baseline are shown in Table 1, expressed as means (for scaled variables), and percentages (for categorical variables). Mean values were used for growth curve analyses. Percentages are shown for descriptive purposes only, representing a relatively high level of risk or problem behavior where relevant (e.g., children who reported that they were "always" stressed for each of the three stress variables). The mean score for low inhibitory control indicates that on average, children report inhibitory control problems either

"never" or "sometime," with about 5% indicating "always" having inhibitory control problems for each of the 6 inhibitory items. The average for academic grades was between A's and B's, with 2.28% reporting that they received D's or lower, representing poor school performance. On average children reported to be stressed between "never" to "sometimes" with almost 2% stating that they were "always" stressed for each of the three items. Children reported, on average, to "sometimes" be able to cope with stress as well as enjoy seeking sensation, with about 2% stating that they were "never" able to cope with stress and 2% stating that they "always" participated in sensation seeking activities. Almost 9% of children perceived their neighborhood to be unsafe enough so as to not go to school at least once in the last month. Children reported playing video games approximately 2.5 hours per day. Almost two-thirds of youth reported playing video games more than 20.5 hours per week. The cut-off of 20.5 hours per week for video gaming was based on Gentile *et al.*'s [5] analysis of ≥20.5 hours/week as representing a high level of video gaming, which far exceeds the 2 hours or more of daily television viewing that is typically considered as high risk sedentary behavior [15]. On average, children reported consuming each of five HCLN items between "once a week" and "2–3 times per week." The cut-off used to illustrate high HCLN intake, ≥25 times per week, was arbitrary, based on a sum of frequency of daily consumption of different types of foods and beverages that would represent HCLN intake more than three times per day (≥25 times/week).

Table 1. Behavioral and demographic characteristics of sample.

Variable	X (SE)	% (SE)
Inhibitory Control Problems	1.29 (0.01)	
Low Inhibitory Control		5.08 (0.01)
Grades	1.73 (0.02)	
Low Achievement		2.28 (0.00)
Stress	1.75 (0.02)	
High Stress		1.66 (0.00)
Coping	2.15 (0.02)	
Low Coping		1.76 (0.00)
Sensation Seeking	1.92 (0.01)	
High Sensation Seeking		2.28 (0.00)
White		30.50 (0.01)
Hispanic		26.97 (0.01)
African American		2.90 (0.01)
Asian		8.20 (0.01)
Mixed/Bi-Racial/Other		31.43 (0.01)
Free Lunch		23.34 (0.01)
Unsafe		8.60 (0.01)
Male		49.59 (0.02)
Video Gaming hours/day	2.45 (0.05)	
≥20.5 hours/week		62.96 (0.01)
HCLN † Intake	2.37 (0.03)	
≥25 Times Per Week		8.51 (0.01)

† HCLN = High Calorie, Low Nutrient food/beverage intake. N = 964 fourth grade students with complete data.

Int. J. Environ. Res. Public Health **2011**, *8*

Current nutritional guidelines, which could have been used to establish cut-offs, are based on % of caloric intake per day, which was not measured in this study (*cf.* [57,58]; http://www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm). A total of 8.51% of the sample reported HCLN intake ≥25 times/week.

Table 2 presents correlations among study variables. As illustrated, inhibitory control problems and sensation seeking were positively correlated with video gaming and HCLN intake. Grades were negatively correlated with video gaming and HCLN intake. Stress was positively correlated with HCLN intake, and coping was not correlated with either video gaming or HCLN intake.

Table 2. Bivariate correlations among study variables.

		1	2	3	4	5	6	7	8	9	10
1.	Inhibitory Control Problems										
2.	Grades	−0.15***									
3.	Stress	0.33**	−0.07*								
4.	Coping	−0.13***	0.09**	−0.08*							
5.	Sensation Seeking	0.33***	0.00	0.13***							
6.	Hisp/AA	−0.04	−0.18***	−0.03	−0.02	−0.07*					
7.	Free Lunch	0.00	−0.16***	0.01	−0.03	−0.02	0.27**				
8.	Unsafe	0.11***	−0.12***	0.13***	−0.01	0.11***	0.07*	0.05			
9.	Male	0.11***	−0.11***	−0.05	0.03	0.15***	0.05	0.01	0.02		
10.	Video Gaming	0.15***	0.13***	0.04	−0.03	0.15***	0.06*	0.06	0.14***	0.32***	
11.	HCLN Intake	0.13***	0.14***	0.11***	−0.05	0.11***	0.14***	0.18***	0.11***	0.17***	0.38***

*** = $p < 0.001$, ** = $p < 0.01$, * = $p < 0.05$.

HCLN = High Calorie Low Nutrient Food/Beverage Consumption.

3.2. Estimates of Relationships of Predictors (Including Demographic Covariates) to HCLN Intake and Video Gaming

Fit estimates for the growth curve model were adequate ($X^2(325) = 685.68$, $p < 0.001$; CFI = 0.950; TLI = 0.950; RMSEA = 0.034). Additionally, the variances for each of our intercept and growth parameters was significant (intercept and slope of video gaming, $p < 0.01$; intercept of HCLN, $p < 0.01$; slope of HCLN, $p < 0.05$). The independent relationships of each predictor to the intercept and slope of each outcome are shown in Table 3. Each outcome was tested in a separate model to determine whether the same set of predictors should be entered in subsequent growth curve analyses with both outcomes modeled simultaneously. As is shown in Table 3, the pattern of predictor/outcome relationships was similar for each outcome. Stress, grades, and coping showed relatively weak relationships to each outcome, but were marginally significant. All predictors were retained for subsequent analyses.

Int. J. Environ. Res. Public Health **2011**, *8*

Table 3. Predictors of growth in HCLN intake and video gaming.

Predictors	HCLN Intake		Video Gaming	
	Intercept	Slope	Intercept	Slope
	β (S.E.)	*β (S.E.)*	*β (S.E.)*	*β (S.E.)*
Low Grades	−0.08 (0.04)†	0.00 (0.07)	−0.06 (0.04)†	−0.10 (0.05)
Male	0.18 (0.04)***	−0.26(0.05)***	0.42 (0.03)***	0.03 (0.08)
Hispanic/AA	0.11 (0.04)**	0.16 (0.05)*	0.04 (0.04)	0.17 (0.09)†
Free Lunch	0.16 (0.04)***	0.07 (0.06)	0.08 (0.04)*	0.06 (0.09)
Unsafe Environment	0.09 (0.04)*	0.06 (0.07)	0.14 (0.04)***	0.02 (0.09)
High Stress	0.08 (0.04)†	−0.18 (0.05)*	−0.01 (0.04)	0.16 (0.09)†
Low Coping Skills	0.05 (0.04)	−0.06 (0.05)	0.07 (0.04)†	−0.10 (0.08)
Inhibitory Problems	0.11 (0.07)**	−0.08 (0.08)	0.13 (0.04)**	−0.28 (0.10)**

† = $p < 0.10$, * = $p < 0.05$, ** = $p < 0.01$; N = 964; HCNL = High Calorie, Low (Poor) Nutrient food and beverage intake.

3.3. Co-Occurrence and Growth in HCLN Intake and Video Gaming

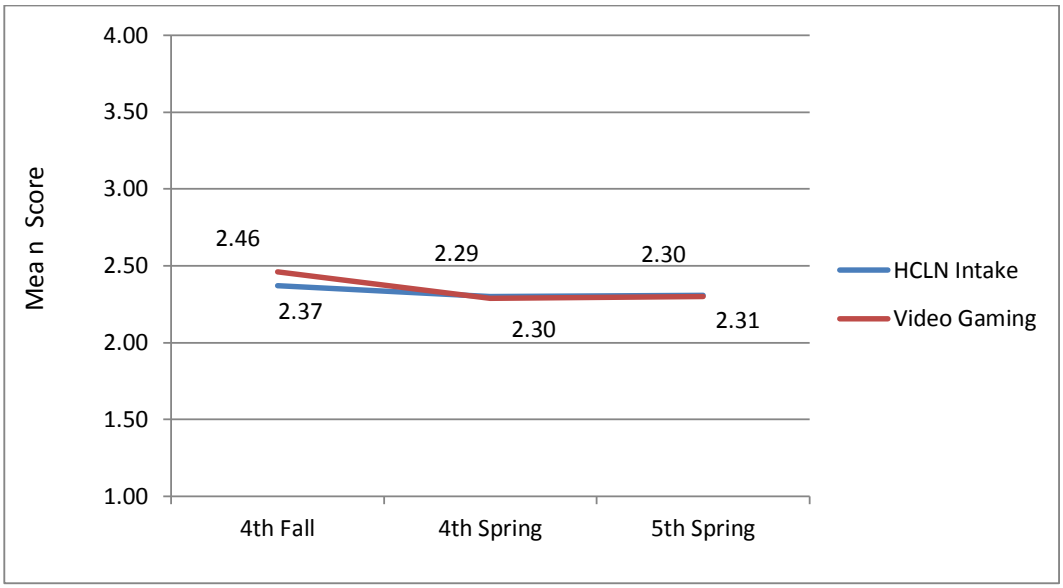
Figure 1 illustrates means for the dependent variables at each study wave, with similar patterns of growth for both HCLN and gaming. Figure 1 illustrates that study Hypothesis 1 was not supported: there was no significant growth in either HCLN intake or video gaming across study waves. Although we did not find growth in mean levels of video gaming and HCLN intake over the three waves of data, we did, as stated above, find significant variance in each of our intercept and, perhaps more importantly, growth parameters. Thus, there is the potential our independent variables to predict this variance. Figure 2 illustrates significant positive associations between video gaming and HCLN intercepts which supports Hypothesis 2 that the two health behaviors would co-occur.

3.4. Common and Behavior Specific Predictors

As illustrated by Figure 2, inhibitory control problems, perceived neighborhood danger or lack of safety, being male, and free/reduced lunch status were each significantly and positively associated with both HCLN intake and video gaming intercepts. Being male was also positively associated with HCLN slope. Being either Hispanic or African-American was significantly associated with HCLN intake intercept and slope. High stress was negatively associated with HCLN intake slope. Inhibitory control problems were significantly negatively associated with video gaming slope. Neither school grades nor poor coping skills were significantly associated with growth parameters. Therefore, Hypotheses 3 and 4 were supported in that there were both common and specific predictors to substance use and HCLN.

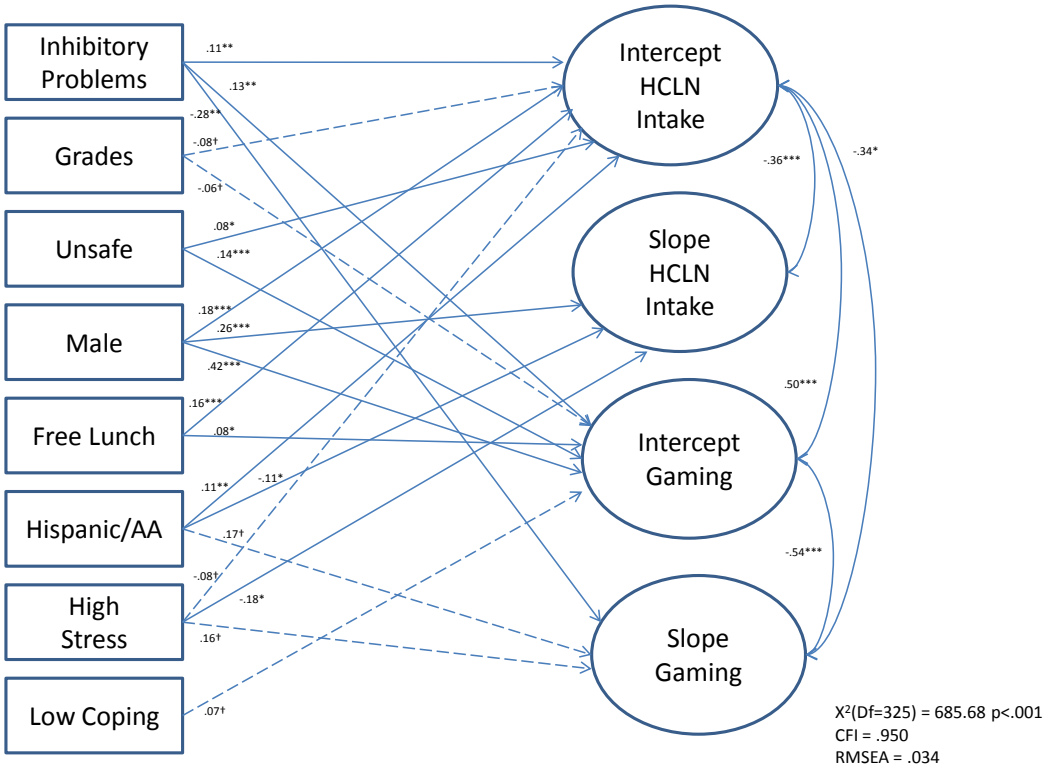
Int. J. Environ. Res. Public Health **2011**, *8*

Figure 1. Co-occurrence and growth in HCLN and video gaming.



N = 964. Adjusted mean scores.

Figure 2. Growth curve model of predictors of HCLN intake and video gaming.



--- Paths are marginally significant.

Int. J. Environ. Res. Public Health **2011**, *8*

4. Discussion and Conclusions

4.1. Summary

The present study tested four hypotheses, three of which were supported. The first hypothesis was that video gaming and HCLN intake grow over time in children. Rapid or extreme growth was assumed to represent potential for addictive behavior. Results of this study showed that high levels of video gaming and HCLN intake were apparent as early as fourth grade, a period of child development associated with adiposity rebound and increased risk for obesity, and that the two behaviors were highly correlated. However, from the starting point of high levels, there was little further growth in video gaming or HCLN intake from fourth through fifth grade, although there was significant variance in the growth parameters for each behavior. There are several plausible explanations for lack of growth. One is that initially high levels may have produced a ceiling effect. A second is that the study examined growth over a relatively short period of time representing two grades and an 18 month period. A third is that growth was examined only during the elementary school years, representing a developmental period over which children may have relatively little individual choice in behavior. Future research could examine whether growth in both behaviors increases significantly once children move into middle school and the early adolescent years that are associated with increased parental autonomy, peer pressure, and individual choice. If so, the pattern would support the use of a piece-wise growth curve model of analysis that can estimate the effects of change in school environment as well as change in developmental stage (from childhood to adolescence).

The second hypothesis was that video gaming and HCLN intake would co-occur. Results of this study supported a co-occurrence model of video gaming and HCLN intake. Both the intercepts and the slopes of the behaviors were highly correlated. In addition, a higher level (intercept) of HCLN intake at baseline was associated with lower growth in video gaming over time (slope). This finding would appear to be counterintuitive but three possibilities could explain this finding. One possibility is that snacking might make video gaming difficult if both hands are occupied in operating video game controls. A second possibility is that since the growth in both behaviors was almost negligent, the finding may be an artifact of a ceiling effect produced by the high intercept values, as noted above. A third possibility is that high levels of HCLN intake may be occurring during periods of alternative sedentary activities such as television viewing or computer homework activities, or around school hours, during which video gaming would not be likely. The present study did not evaluate this possibility, although the correlations of video gaming with other sedentary activities were relatively high (TV watching hours as screen time, $r = 0.50$, $p < 0.001$; computer hours, $r = 0.46$, $p < 0.001$), and are consistent with findings from other research that has shown a negative correlational relationship between video gaming and length of time spent on exercise [11,12].

The third hypothesis was that there was a common set of predictors of both video gaming and HCLN intake. Based on previous research, these were inhibitory control problems, male gender, low socioeconomic status, poor grades, Hispanic race/ethnicity, high stress, and poor coping [5,38-42]. Four common risk factors were found, thus supporting the third hypothesis. The strongest predictors of both behaviors were inhibitory control problems, male gender, low socioeconomic status as measured by receiving free or reduced lunch at school, and an exploratory factor, perceived lack of safety of the

neighborhood environment, which has received relatively little attention in the literature on addictive behaviors in children. Overall, the results are consistent with findings from adolescent substance use studies that have shown associations between inhibitory control problems, male gender, low socioeconomic status and substance use (e.g., [17,18]), as well as a previous study on children [24]. Inhibitory control deficit (similar to impulsivity) was significantly related to high intercept levels of both video gaming *and* HCLN intake. Boys exhibited higher levels of both video gaming and HCLN intake than girls, and slightly more growth in video gaming. Receiving free or reduced lunch, was positively related to intercepts and growth in both video gaming and HCLN intake. Additionally, perceived lack of safety in the environment representing from home to school was significantly related to high levels of both video gaming and HCLN intake. This may be the first study to relate safety to these co-occurring behaviors. Previous research on adults has focused on the relationships of lack of perceived safety of the neighborhood environment to low levels of walking as a physical activity [59].

The results of the present study raise the possibility that perceived lack of safety may keep children indoors at home, whether this is a personal decision or due to parent rules and concerns about safety. With few opportunities to engage in physical activity within the home, combined with potential boredom over being restricted in activity, children may turn to greater HCLN intake and video gaming as means to cope with confinement. In conjunction with the findings on socioeconomic status, results of this study raise the question of whether children who live in some types of impoverished, unsafe neighborhoods might constitute a maj or risk group for developing addictive gaming and HCLN intake behaviors, and subsequently, health problems related to these behaviors, including obesity and Type II diabetes [60,61].

The fourth exploratory hypothesis was that some risk factors differentiated video gaming and HCLN intake either in terms of strength or directionality which could signal potential addiction specificity. There were three. Hispanic or African-American status was positively related to intercept and growth in HCLN intake, but not related to gaming. High stress was negatively related to HCLN intake, but showed a non-significant positive relationship to growth in gaming. The direction of relationship of stress to HCLN intake is counter to findings on effects of stress and poor coping on binge eating in adults, as well as stressful, emotional eating as reported by adolescents [32,33,41]. One possible explanation for the contrary finding is that children may consume HCLN products because they may be readily available in the home rather than as a response to stress. The low prevalence of children in this study who reported high levels of stress would support this explanation. Finally, although the relationship was not significant, low coping was positively related to the gaming intercept, but not related to HCLN intake.

4.2. Unexpected Findings

School achievement was not significantly related to either video gaming or HCLN intake, although there was a non-significant trend of lower grades associated with both higher video gaming and HCLN intake intercepts (*cf.* [5,38]). The Gentile *et al*. [5] study found a significant relationship of poor achievement to video gaming, however, achievement was examined as an outcome rather than as a predictor and the focus was pathological gaming rather than growth in gaming behavior.

Int. J. Environ. Res. Public Health **2011**, *8*

Another unexpected finding was the lack of relationship of sensation-seeking to either video gaming or HCLN intake in growth curve analyses. Although the initial correlations of sensation-seeking with these behaviors were significant, they were small ($r = 0.16$ with gaming, $r = 0.11$ with high calorie, low nutrient food intake, p's < 0.05), and sensation-seeking was subsequently eliminated from further analyses because it did not contribute to model fit. It also showed poor internal consistency ($\alpha = 0.42$), although comparable to that found for the BSS4 ($\alpha = 0.44$; 50). One possible explanation is that much of previous research that has measured sensation-seeking and shown relationships of sensation-seeking to substance use and other health risk behaviors is based on adolescent populations (e.g., [17-19]). Arousal and impulsivity, which are associated with increased risk-taking and sensation seeking, appear to be linked with changes in brain circuitry during adolescence [62]. Furthermore, research suggests an increased neurobiological vulnerability to addictive behavior during adolescence [25,36]. These neurobiological changes may not have occurred yet in children. Thus, even if a child exhibited a high level of sensation-seeking, it may not yet operate as a neurobiological trigger to addictive behavior.

4.3. Limitations

There are several study limitations which should be considered in drawing conclusions about video gaming and HCLN intake as potentially addictive behaviors in childhood. One is reliance on self-report measures, several of which were abbreviated for use with children. However, the study used measures that have been standardized in other studies and abbreviated to accommodate to school class time restrictions, with comparable reliability [41,48,49,52]. Another is that the study period, although longitudinal with three waves of measurement, may not yet be sufficient to find significant growth in behavior. However, the focus on children for purposes of early prediction, combined with the finding of relatively high intercepts at baseline in fourth grade, should have important implications for both identifying and preventing addictive behavior propensity. An additional limitation is that other potential risk factors for video gaming and HCLN intake were not included in this study, primarily because there were no corresponding measures for both behaviors. Primary among these are parent influences [9]. While modeling of HCLN intake by parents is included in the Pathways trial, there are no corresponding variables available for video gaming. Thus these risk factors could not be evaluated in a co-occurrence or co-prediction model.

4.4. Implications of the Findings and Future Directions

Several findings have particular importance for designing programs to prevent addictive behavior as early as in childhood. One is that video gaming and HCLN intake appear to co-occur in children and exhibit several common risk factors which are also associated with substance use behavior. This finding argues strongly for the development of universal prevention programs that are aimed at preventing multiple health risk behaviors early in childhood [9]. Second is the strong predictive relationship of low inhibitory control and low perceived safety to both HCLN intake and gaming. These findings suggest that a multiple health risk behavior prevention program should probably take a multi-level, ecological approach that incorporates individual skills training to improve executive cognitive function (ECF) [35], as well as physical exercise to replace sedentary activity or

promotion of active rather than passive video gaming in the absence of other physical activity opportunities [11,12], and strategies to improve the safety of the built environment surrounding the child in order to facilitate walking and other types of outdoor exercise, for example, introduction of a Safe Routes to Schools program. There is already growing evidence to suggest that ECF training has multiple benefits for children [63], that increased physical activity has a positive effect on ECF [64] and negative effect on substance use [65], and that increasing perceived safety of the environment promotes more walking [59]. Whether increased walking can replace sedentary screen time, whether this involves gaming, television viewing, or internet or mobile use, is not yet known [66,67]. Finally, some factors that have been found to predict substance use in adolescents did not predict HCLN intake or gaming in children (low grades, sensation seeking, low coping), and others (race/ethnicity and high stress) had a differential effect on HCLN but not gaming. The variation in risk factors suggests that future programs that do include multiple health risk behaviors might tailor applications of skills training to different groups and different situational contexts. For example, addressing prosocial alternatives to sensation-seeking might be applied to substance use risk situations but not food choice situations; and addressing healthy food choices might be tailored to the context of different parent modeling behaviors or different food products that are available in some homes but not others.

Acknowledgements

This paper was supported by grants from the National Institute of Child Health and Human Development (#HD-052107) and National Institute on Drug Abuse (#DA-027226) and is registered at ClinicalTrials.gov (#NCT00787709).

References

1. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
2. *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR*, 4th ed.; American Psychiatric Association: Arlington, VA, USA, 2000; pp. 1-943.
3. Potenza, M.N. Should addictive disorders include non-substance-related conditions*? Addiction* **2006**, *101(Suppl 1)*, 142-151.
4. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority. *Eval. Health Prof.* **2011**, *34*, 3-56.
5. Gentile, D.A.; Choo, H.; Liau, A.; Sim, T.; Li, D.; Fung, D.; Khoo, A. Pathological video game use among youths: A two-year longitudinal study. *Pediatrics* **2011**, *127*, e319-e329.
6. Petry, N.M. Substance abuse, pathological gambling, and impulsiveness. *Drug Alcohol Depend.* **2001**, *63*, 29-38.
7. Grüsser, S.M.; Thalemann, R.; Griffiths, M.D. Excessive computer game playing: Evidence for addiction and aggression? *Cyberpsychol. Behav.* **2007**, *10*, 290-292.
8. Ogden, C.L.; Carroll, M.D.; Curtin, L.R.; McDowell, M.A.; Tabak, C.J.; Flegal, K.M. Prevalence of overweight and obesity in the United States, 1999–2004. *J. Am. Med. Assoc.* **2006**, *295*, 1549-1555.

9. Pentz, M.A. Understanding and preventing risks for adolescent obesity. In *Adolescent Health: Understanding and Preventing Risk*; Crosby, R., Santelli, J., Eds.; John Wiley & Sons: Hoboken, NJ, USA, 2009; pp. 147-164.
10. Pearson, N.; Biddle, S.J. Sedentary behavior and dietary intake in children, adolescents, and adults: A systematic review. *Am. J. Prev. Med.* **2011**, *41*,178-188.
11. Ballard, M.E. Video game play, behavior, and dietary health. In *Handbook of Behavior, Food, and Nutrition*; Preedy, V.R., Watson, R.R., Martin, C.R., Eds.; Springer: New York, NY, USA, 2011; pp. 2169-2181.
12. Ballard, M.E.; Gray, M.A.; Reilly, J.; Noggle, M. Correlates of video game screen time among males: Body mass, physical activity, and other media use. *Eat. Behav.* **2009**, *10*, 161-167.
13. Institute of Medicine. *Overview of the IOM's Childhood Obesity Prevention Study*; 2004. Available online: www.iom.edu/report.asp?id=22596 (accessed on 14 April 2008).
14. Dorosty, A.R.; Emmett, S.R.D.; Cowin, I.S.; Reilly, J.J. Factors associated with early adiposity rebound. ALSPAC Study Team. *Pediatrics* **2000**, *105*, 1115-1118.
15. Hoelscher, D.M.; Day, R.S.; Kelder, S.H.; Ward, J.L. Reproducibility and validity of the secondary level School-Based Nutrition Monitoring (SBNM) student questionnaire. *J. Am. Diet. Assoc.* **2003**, *103*, 186-194.
16. Bardo, M.T.; Pentz, M.A. Translational research. In *APA Handbook of Research Methods in Psychology*; Cooper, H., Ed.; American Psychiatric Association: Washington, DC, USA, 2011.
17. Stephenson, M.T.; Hoyle, R.H.; Palmgreen, P.; Slater, M.D. Brief measures of sensation seeking for screening and large-scale surveys. *Drug Alcohol Depend.* **2003**, *72*, 279-286.
18. Crawford, A.M.; Pentz, M.A.; Chou, C.P.; Li, C.; Dwyer, J.H. Parallel developmental trajectories of sensation seeking and regular substance use in adolescents. *Psychol. Addict. Behav.* **2003**, *17*, 179-192
19. Kelley, A.E.; Schochet, T.; Landry, C.F. Risk taking and novelty seeking in adolescence: Introduction to part I. *Ann. NY Acad. Sci.* **2004**, *1021*, 27-32.
20. Belin, D.; Mar, A.C.; Dalley, J.W.; Robbins, T.W.; Everitt, B.J. High impulsivity predicts the switch to compulsive cocaine-taking. *Science* **2008**, *320*, 1352-1355.
21. Verdejo-García, A.; Lawrence, A.J.; Clark, L. Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neurosci. Biobehav. Rev.* **2008**, *32*, 777-810.
22. Verdejo-Garcia, A.; Bechara, A.; Recknor, E.C.; Pérez-Garcia, M. Executive dysfunction in substance dependent individuals during drug use and abstinence: An examination of the behavioral, cognitive and emotional correlates of addiction. *J. Int. Neuropsychol. Soc.* **2006**, *12*, 405-415.
23. Bechara, A. Risky business: Emotion, decision-making, and addiction. *J. Gambl. Stud.* **2003**, *19*, 23-51.
24. Riggs, N.R.; Spruijt-Metz, D.; Chou, C.P.; Pentz, M.A. Relationships between executive cognitive function and lifetime substance use, and obesity-related behaviors in fourth grade youth. *Child Neuropsychol.* **2011**, *7*, 1-11
25. Chambers, R.A.; Taylor, J.R.; Potenza, M.N. Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *Am. J. Psychiatry* **2003**, *160*, 1041-1052.

26. Fishbein, D.; Tarter, R. Infusing neuroscience into the study and prevention of drug misuse and co-occurring aggressive behavior. *Subst. Use Misuse* **2009**, *44*, 1204-1235.
27. Riggs, N.R.; Greenberg, M.T. Neurocognition as a moderator and mediator of adolescent substance misuse prevention. *Am. J. Drug Alcohol Abuse* **2009**, *35*, 209-213.
28. Cerqueira, J.J.; Mailliet, F.; Almeida, O.F.X.; Jay, T.M.; Sousa, N. The prefrontal cortex as a key target of the maladaptive response to stress. *J. Neurosci.* **2007**, *27*, 2781-2787.
29. Bickel, W.K.; Marsch, L.A. Toward a behavioral economic understanding of drug dependence: Delay discounting processes. *Addiction* **2001**, *96*, 73-86.
30. Montague, P.R.; Berns, G.S. Neural economics and the biological substrates of valuation. *Neuron* **2002**, *36*, 265-284.
31. Posner, M.I.; Rothbart, M.K. Toward a physical basis of attention and self regulation. *Phys. Life Rev.* **2009**, *6*, 103-120.
32. Volkow, N.D.; Wise, R.A. How can drug addiction help us understand obesity? *Nat. Neurosci.* **2005**, *8*, 555-560.
33. Marsh, R.; Horga, G.; Wang, Z.; Wang, P.; Klahr, K.W.; Berner, L.A.; Walsh, B.T.; Peterson, B.S. An fMRI study of self-regulatory control and conflict resolution in adolescents with bulimia nervosa. *Am. J. Psychiatry* **2011**, doi: 10.1176/appi.ajp.2011.11010094.
34. Guy, S.C.; Isquith, P.K.; Gioia, G.A. *BRIEF (The Behavior Rating Inventory of Executive-Function-Self-Report Version BRIEF-SR)*, Behavior Rating Inventory of Executive Function-Self-Report Version; Psychological Assessment Resources, Inc.: Odessa, FL, USA, 2004.
35. Riggs, N.R.; Kobayakawa-Sakuma, K.L.; Pentz, M.A. Preventing risk for obesity by promoting self-regulation and decision-making skills: Pilot results from the Pathways to Health Program. *Eval. Rev.* **2007**, *31*, 287-310.
36. Romeo, R.D.; McEwen, B.S. Stress and the adolescent brain. *Ann. NY Acad. Sci.* **2006**, *1094*, 202-214.
37. Jasuja, G.K.; Chou, C.P.; Riggs, N.R.; Pentz, M.A. Early cigarette use and psychological distress as predictors of obesity risk in adulthood. *Nicotine Tob. Res.* **2008**, *10*, 325-335.
38. Johnston, L.D.; O'Malley, P.M.; Bachman, J.G.; Schulenberg, J.E. *Monitoring the Future National Survey Results on Drug Use, 1975–2010. Volume I: Secondary School Students*; Institute for Social Research, University of Michigan: Ann Arbor, MI, USA, 2011; p. 734.
39. Sussman, S.; Pentz, M.A.; Spruijt-Metz, D.; Miller, T. Abuse of "study drugs": Prevalence, consequences, and implications for therapeutic prescription and policy. *Subst. Abuse Treat. Prev. Policy* **2006**, *1*, doi: 10.1186/1747-597X-1-15.
40. Huh, H.; Riggs, N.R.; Spruijt-Metz, D.; Chou, C.P.; Huang, Z.; Pentz, M. Identifying patterns of eating and physical activity in children: A latent class analysis of obesity risk. *Obesity* **2010**, *19*, 652-658.
41. Nguyen-Michel, S.T.; Unger, J.B.; Metz, D.S. Dietary correlates of emotional eating in adolescence. *Appetite* **2007**, *49*, 494-499.
42. Riggs, N.R.; Spruijt-Metz, D.; Sakuma, K.L.; Chou, C.P.; Pentz, M.A. Executive cognitive function and food intake in children. *J. Nutr. Educ. Behav.* **2010**, *42*, 398-403.

43. Rollins, B.Y.; Riggs, N.R.; Spruijt-Metz, D.; McClain, A.D.; Chou, C.P.; Pentz, M.A. Psychometrics of the Eating in Emotional Situations Questionnaire (EESQ) among low-income Latino elementary-school children. *Eat. Behav.* **2011**, *12*, 156-159.
44. Singh, G.K.; Slahpush, M.; Kogan, M.D. Rising social inequalities in U.S. childhood obesity, 2003–2007. *Ann. Epidemiol.* **2010**, *20*, 40-52.
45. Pentz, M.A.; Johnson, C.A.; Dwyer, J.H.; MacKinnon, W.B.; Flay, B.R. A comprehensive community approach to adolescent drug abuse prevention: Effects on cardiovascular disease risk behaviors. *Ann. Med.* **1989**, *21*, 219-222.
46. Greenberg, M.T.; Kusché, C.A. *Promoting Social and Emotional Development in Deaf Children: The PATHS Project*; University of Washington Press: Seattle, WA, USA, 1993.
47. SAS Institute Inc. *SAS Language: Reference, Version 6*, 1st ed.; SAS Institute Inc.: Cary, NC, USA, 1990.
48. Field, A.E.; Peterson, K.E.; Gortmaker, S.L.; Cheung, L.; Rockett, H.; Fox, M.K.; Colditz, G.A. Reproducibility and validity of a food frequency questionnaire among fourth to seventh grade inner-city school children: Implications of age and day-to-day variation in dietary intake. *Public Health Nutr.* **1999**, *2*, 293-300.
49. Willett, W.C.; Sampson, L.; Stampfer, M.J.; Rosner, B.; Bain, C.; Witschi, J.; Hennekens, C.H.; Speizer, F.E. Reproducibility and validity of a semiquantitative food frequency questionnaire. *Am. J. Epidemiol.* **1985**, *122*, 51-65.
50. *California Healthy Kids Survey: Main Report*; WestEd Health and Human Development Program for the California Department of Education: San Francisco, CA, USA, 2009.
51. Cohen, S.; Kamarck, T.; Mermelstein, R.A. Global measure of perceived stress. *J. Health Soc. Behav.* **1983**, *24*, 385-396.
52. Malmberg, M.; Overbeek, G.; Monshouwer, K.; Lammers, J.; Vollebergh, W.A.; Engels, R.C. Substance use risk profiles and associations with early substance use in adolescence. *J. Behav. Med.* **2010**, *33*, 474-485.
53. Centers for Disease Control (CDC). *Youth Risk Behavior Survey*; CDC: Atlanta, GA, USA, 2005. Available online: http://www.cdc.gov/HealthyYouth/yrbs/pdfs/2005highschoolquestionnaire.pdf (accessed on 22 November 2011).
54. Eaton, D.K.; Kann, L.; Kinchen, S.; Ross, J.; Hawkins, J.; Harris, W.A.; Lowry, R.; McManus, T.; Chyen, D.; Shanklin, S.; *et al.* Youth risk behavior surveillance—United States, 2005. *J. School Health* **2006**, *76*, 353-372.
55. Paeratakul, S.; Lovejoy, J.C.; Ryan, D.H.; Bray, G.A. The relation of gender, race and socioeconomic status to obesity and obesity comorbidities in a sample of US adults. *Int. J. Obes. Relat. Metab. Disord.* **2002**, *26*, 1205-1210.
56. Muthén, L.K.; Muthén, B.O. *Mplus Users Guide*, 6th ed.; Muthén & Muthén: Los Angeles, CA, USA, 1998.
57. Golley, R.K.; Hendrie, G.A.; McNaughton, S.A. Scores on the dietary guideline index for children and adolescents are associated with nutrient intake and socio-economic position but not adiposity. *J. Nutr.* **2011**, *141*, 1340-1347.
58. Reedy, J.; Krebs-Smith, S.M. Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. *J. Am. Diet Assoc.* **2010**, *110*, 1477-1484.

59. Durand, C.P.; Andalib, M.; Dunton, G.F.; Wolch, J.; Pentz, M.A. A systematic review of built environment factors related to physical activity and obesity risk: Implications for smart growth urban planning. *Obes. Rev.* **2011**, *12*, e173-e182.
60. Midei, A.J.; Matthews, K.A. Interpersonal violence in childhood as a risk factor for obesity: A systematic review of the literature and proposed pathways. *Obes. Rev.* **2011**, *12*, e159-e172.
61. Pentz, M.A. Defining Neighborhoods of Opportunity by Green Spaces, School Achievement, and Perceived Neighborhood Safety: Illustrations from Two Drug Abuse and Obesity Prevention Trials. Presented at *National Hispanic Science Network on Drug Abuse*, New Orleans, LA, USA; 30 September–2 October 2010.
62. Galvan, A.; Hare, T.; Voss, H.; Glover, G.; Casey, B.J. Risk-taking and the adolescent brain: Who is at risk? *Dev. Sci.* **2007**, *10*, 8-14.
63. Diamond, A.; Lee, K. Interventions shown to aid executive function development in children 4 to 12 years old. *Science* **2011**, *333*, 959-964.
64. Davis, C.L.; Tomporowski, P.D.; Boyle, C.A.; Waller, J.L.; Miller, P.H.; Naglieri, J.A.; Gregoski, M. Effects of aerobic exercise on overweight children's cognitive functioning: A randomized controlled trial. *Res. Q. Exerc. Sport* **2007**, *78*, 510-519.
65. Terry-McElrath, Y.M.; O'Malley, P.M.; Johnston, L.D. Exercise and substance use among American youth, 1991–2009. *Am. J. Prev. Med.* **2011**, *40*, 530-540.
66. Steffen, L.M.; Dai, S.; Fulton, J.E.; Labarthe, D.R. Overweight in children and adolescents associated with TV viewing and parental weight: Project HeartBeat! *Am. J. Prev. Med.* **2009**, *37(Suppl 1)*, S50-S55.
67. Robinson, T.N.; Wilde, M.L.; Navracruz, L.C.; Haydel, K.F.; Varady, A. Effects of reducing children's television and video game use on aggressive behavior: A randomized controlled trial. *Arch. Pediatr. Adolesc. Med.* **2001**, *155*, 17-23.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Ream, G.L.; Elliott, L.C.; Dunlap, E. Patterns of and Motivations for Concurrent Use of Video Games and Substances. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3999-4012; doi:10.3390/ijerph8103999.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Patterns of and Motivations for Concurrent Use of Video Games and Substances

Geoffrey L. Ream [1,*], Luther C. Elliott [2] and Eloise Dunlap [2]

[1] School of Social Work, Adelphi University, 1 South Avenue, Garden City, NY 11530, USA
[2] Institute for Special Populations Research, National Development and Research Institutes, 8th Floor, 71 West 23rd Street, New York, NY 10010, USA; E-Mails: luther@nyu.edu (L.C.E.); dunlap@ndri.org (E.D.)

* Author to whom correspondence should be addressed; E-Mail: ream@adelphi.edu; Tel.: +1-516-877-4432; Fax: +1-516-877-4392.

Received: 24 August 2011; in revised form: 6 October 2011 / Accepted: 12 October 2011 / Published: 19 October 2011

Abstract: "Behavioral addictions" share biological mechanisms with substance dependence, and "drug interactions" have been observed between certain substances and self-reinforcing behaviors. This study examines correlates of patterns of and motivations for playing video games while using or feeling the effects of a substance (concurrent use). Data were drawn from a nationally-representative survey of adult Americans who "regularly" or "occasionally" played video games and had played for at least one hour in the past seven days (n = 3,380). Only recent concurrent users' data were included in analyses (n = 1,196). Independent variables included demographics, substance use frequency and problems, game genre of concurrent use (identified by looking titles up in an industry database), and general game playing variables including problem video game play (PVP), consumer involvement, enjoyment, duration, and frequency of play. Exploratory factor analysis identified the following dimensions underlying patterns of and motivations for concurrent use: pass time or regulate negative emotion, enhance an already enjoyable or positive experience, and use of video games and substances to remediate each other's undesirable effects. Multivariate regression analyses indicated PVP and hours/day of video game play were associated with most patterns/motivations, as were caffeine, tobacco, alcohol, marijuana, and painkiller use problems. This suggests that concurrent use with some regular situational pattern or effect-seeking motivation is part of the addictive

process underlying both PVP and substance dependence. Various demographic, game playing, game genre of concurrent use, and substance use variables were associated with specific motivations/patterns, indicating that all are important in understanding concurrent use.

Keywords: video games; caffeine; tobacco; alcohol; marijuana; addiction; dependence

1. Introduction

Although the potential of substance use to add to the experience of self-reinforcing behaviors is recognizable by anyone who smokes tobacco while playing cards or drinks wine at the theater, there is little systematic research on it. Research should address this issue because, at least in the case of behaviors with addictive potential, concurrent substance use might not be entirely benign. A recent review found problem gambling to share nosological, clinical, and biological features with substance use disorders [1], and laboratory studies find alcohol exacerbates problem gambling [2-4]. Several studies, in fact, have found "behavioral addictions" [5] and substance use problems to share common biological pathways, including the endogenous cannabinoid [6], dopamine [7], and hypocretin [8] systems underlying reward and arousal.

The present study is concerned with video game play, which has demonstrated effects on the brain over time similar to substance addiction [9-11]. Video game "addiction" is not universally accepted [12-14], and a lack of consensus on how to measure it makes estimating prevalence difficult [15,16]. However, research has generally affirmed the existence of clinically significant problem video game play, with prevalence estimates of problem gaming behaviors hovering between 4.9% and about 9% among video gamers internationally [15,17-21]. Smaller numbers have been found to fit stricter criteria for dependence, e.g., 2–5% of children and youth overall in a recent review [15], and 3% of male and 0.3% of female adolescents in a German national study [22]. Problem video gaming behavior is a growing source of scholarly and clinical concern [23,24], with an American Medical Association report calling for more research on it [25]. Findings from treatment of problem video gaming behavior further underscore its biological dimensions [15]: Video games are associated with development of attention problems in children [26,27], although this relationship is complex and involves other factors [28], and dopaminergic medications indicated for ADHD and substance dependence have been shown to remediate problem video gaming behavior [29,30].

Problem video gaming behavior is also associated with measures of "addiction" to various substances, even caffeine [18,31-33]. This leads to the concern that playing video games while using or feeling the effects of substances—called "concurrent use" in this study—may create a complementary effect similar to the one found with gambling [2-4]. To the extent that players experience this "drug interaction" intentionally through concurrent use, it is logical to ask about patterns of and motivations for this behavior [24]. These considerations are potentially important to addiction specificity [34], *i.e.*, differential development of specific patterns of addictive behaviors based on attraction to the behavior, shared experiences with other participants in the behavior, expectations for outcomes of the behavior, and other factors. For example, concurrent use with any

conscious, regular situational pattern or effect-seeking motivation is probably associated with greater degrees of problem use of both video games and substances than merely coincidental concurrent use. Also, specific substances of concurrent use, demographic groups of concurrent users, or social situations of concurrent use may be associated with some concurrent use patterns/motivations but not others. Video game genres [35-38] involved in concurrent use may be differentially compatible with specific patterns/motivations of concurrent use.

This study, accordingly, explored patterns/motivations of concurrent use in the context of general patterns of game playing and substance use behavior. In addition to simple frequency and duration of concurrent use, we considered preferred context of concurrent use (alone or with certain friends) and several effect-seeking motivations, including self-medication of loneliness or depression, using substances to enhance game experience, using substances to cope with game-related frustration, using video games to pass time while feeling effects of substances, and using video games to cope with substance withdrawal. Our analyses first distinguished common factors underlying patterns of and motivations for concurrent use. Then, we explored potential correlates of both frequency variables and these factors among demographics, general game playing behavior, genre of concurrent use, and substance of concurrent use, including substance use problems.

2. Methods

2.1. Participants and Recruitment

Participants were a subset of a nationally representative KnowledgePanel® maintained by the commercial online research survey provider Knowledge Networks (KN). KN selects panel members via random-digit dial and address-based sampling, provides computers and internet access if needed, establishes informed consent, and collects demographics. KN randomly recruits panel members via e-mail for client surveys (e.g., the present study) which they incentivize with "points" toward cash and other rewards. KN offers the option of screening the randomly selected panel members and only allowing them to participate if they meet client-specified criteria. For this survey, 15,642 e-mails were sent to panel members ages 18 and over, and 9,215 (59%) completed the screening instrument. The screener asked whether participants "regularly," "occasionally," or "never" participated in 11 different hobby activities in the past year, including video games. Participants who responded "regularly" or "occasionally" about video games were then asked how many hours they played in the past 7 days. Participants who reported one or more hours (n = 3,380; 37%), were allowed to take the survey. The screening and survey were conducted in either English or Spanish. Median completion time was 10 minutes, the maximum feasible given budgetary and methodological constraints. The protocol for this study was reviewed and approved by all investigators' Institutional Review Boards.

2.2. Measures

Demographics. Age, race/ethnicity, gender, education, income, metropolitan statistical area (MSA) resident status, and employment status were taken from Knowledge Networks' basic demographic survey.

Video game days used, hours/day used, and enjoyment. Participants were asked to list up to five video game titles they had "spent a lot of time playing in the past 12 months." For each title, they were asked how many days of the past 30 they had played it, how many hours they played on days they played it, and how much they enjoyed it. Enjoyment was a single 7-point Likert scale in which 1 = "it was the worst game I've ever played," 4 = "about the same as most games," and 7 = "it was my single all-time favorite." These variables were averaged within each participant to reflect the average game that person played.

Consumer involvement. Participants were asked about dimensions of enthusiasm for video games with no necessary addictive connotation – attraction, centrality/importance, and self-expression [39-41] using a 3-item Likert-scale measure adapted from leisure and marketing studies, Cronbach's $\alpha = 0.72$.

Problem video game playing (PVP). A 5-item version of the original 9-item Likert scale [42] was used to measure increased time spent playing (tolerance), difficulty controlling time spent playing, restlessness/irritability when can't play (withdrawal), play to relieve negative emotions (self-medication), and disregarding negative consequences of play, Cronbach's $\alpha = 0.76$.

Video game genres of concurrent use. Of 7,203 titles from 3,380 participants, 6,056 from 2,885 participants could be clearly distinguished as valid titles of single games or game series with identical genre descriptors (e.g., professional football simulations updated annually to include current years' players). Valid titles were those that could be found in GameFaqs [43], an exhaustive database of user-generated content maintained and edited by an industry group. Invalid entries included overly-broad categories of games, names of game platforms, or qualitative responses. Participants listed a total of 1,335 different valid titles, ranging in frequency from 1 to 340. Each title's genre was coded as the major category under which it was listed in GameFaqs, with some categories broken into theoretically significant subcategories, as follows: Action-adventure, massively multiplayer online role-playing games (MMORPG's), other role-playing games (RPG's), first-person shooter (FPS), other shooter, gambling, real-time Strategy (RTS), other strategy, board/card games, sports general, other sports, puzzle, rhythm, driving, platformer, and a catch-all category of other genres including titles that were valid but belonged to genre categories with 10 or fewer titles or 30 or fewer players (e.g., fighting).

Participants were also asked whether they had played each game that they listed while using or feeling the effects of substances. For each of 16 dummy variables for genre of concurrent use, participants were coded 1 if they reported playing a game from that genre and concurrent use with it, and 0 of they either reported playing a game from that genre with no concurrent use or did not report playing that genre.

Substance use frequency and use problems. Measures were adapted from the National Survey of Drug Use and Health (NSDUH, [44]). Participants chose substances they had used in the past 30 days from a list. For each substance used in the past 30 days, participants were asked on how many of the past 30 days they had used and presented with a series of abuse/dependence symptoms. In order to keep the survey within the median length of 10 minutes, measures were shortened by selecting those with the highest correlation to an underlying dimension of abuse/dependence from factor analysis of data from a related study (a computer assisted personal interview survey supported by the same grant as the present study; data analyses are underway) which used the full measures. Measures for this

study included five dichotomous items for caffeine (tolerance, difficulty controlling use, desire to quit/cut down, withdrawal, disregarding negative emotional/physical health consequences), four Likert-scale items for tobacco (withdrawal, craving, worry over running out, tolerance), and seven dichotomous items each for alcohol, marijuana, painkillers, and sedatives (tolerance, difficulty controlling use, desire to quit/cut down, withdrawal, disregarding negative emotional/physical health consequences, neglecting positive activities, and spending a lot of time obtaining or using). Reliability was found to be adequate according to comparative fit indices from confirmatory factor analyses for each substance: caffeine: 0.962, tobacco: 0.990, alcohol: 0.997, marijuana: 0.998, painkillers: 0.979, sedatives: 0.994.

Concurrent use patterns and motivations. These items assessed endorsement of various patterns of and motivations for concurrent use of video games and substances identified in previous research and pilot qualitative data. Participants who reported recent concurrent use were asked how many days of the past 30 and the average number of hours on each of those days they spent concurrently using, and to respond to each of the following on a 5-point Likert scale from 1 = "Not at all true" to 5 = "Extremely true": "When you're alone, you like to play video games and use substances," "When you get together with certain friends, you often use substances and play video games," "You use video games and substances together to help cope with loneliness or depression," "Certain substances really enhance your experience of certain video games," "When video games become frustrating, you use substances to calm down," "You play video games to pass the time while feeling the effects of a substance," "You play video games to get through withdrawal, being hung over, or coming down from a substance".

2.3. Approach to Analyses

Income was categorized into increments that were increasingly larger further up the scale until "$175,000 or more." Employment was collapsed into categories of (1) working, either for wages or self-employed, or (2) non-working for any reason, e.g., disability, retirement, layoff, *etc*. Because these variables were from KN's demographic database, no data are missing on them.

Table 1 reports the most parsimoniously interpretable set of results from several exploratory factor analyses conducted in STATA 12 of the Likert-scale pattern/motivation variables. Similar structure emerged from procedures involving all combinations of Kaiser normalization on or off, maximum likelihood or principal factors estimation, and promax or (oblique) Bentler's invariant pattern simplicity rotation. Most other estimation and rotation methods produced either a single factor or so many cross-loaded items that simple structure was not achieved. Once the factor structure was settled upon, confirmatory factor analysis was run in MPlus 6.0 using maximum likelihood estimation with robust standard errors.

The analysis presented in Table 2 is a single multivariate model estimated in MPlus 6.0 with the frequency indicators and motivation factors as dependent variables and all demographic, game playing behavior, game genre, and substance use/problem variables as independent variables. We specified the model so that dependent variables were allowed to correlate. In order to include all cases in this single omnibus analysis, zeroes were imputed for substance use variables for non-users of the substance in question. Like the factor analysis reported in Table 1, the analysis reported in Table 2 is also the most

parsimoniously interpretable of several alternative specifications, which variously involved latent indicators for some or all of the composite variables. All alternatively specified models had adequate fit [45] and similar patterns of significant coefficients for independent variables, indicating that our results are robust.

All analyses employed post-stratification weights provided by Knowledge Networks so that estimates more accurately reflect what would have been obtained from a true random sample of English- and Spanish-speaking American adult video game players [46]. Only weighted point estimates and hypothesis tests are presented in the results section.

Table 1. Exploratory factor analysis derivation of factor score indicators for patterns of and motivations for concurrent use.

		Pass time or regulate negative emotion	**Enhance positive experience**	**Remediate each other's [a] undesirable effects**	**Uniqueness**
Factor loadings:	While alone	**0.78**	0.12	−0.23	0.45
	Cope with loneliness/depression	**0.70**	−0.26	0.26	0.43
	Pass time while feeling effects of substance	**0.51**	0.20	0.09	0.49
	With certain friends	−0.01	**0.72**	0.09	0.44
	Substances enhance experience of games	0.09	**0.55**	0.18	0.51
	Substance to cope with game-related frustration	0.04	0.04	**0.60**	0.58
	Play games to cope with substance withdrawal	−0.08	0.13	**0.63**	0.59
Correl-ation matrix:	Pass time or regulate negative emotion	1	0.63	0.63	
	Enhance positive experience	0.63	1	0.38	
	Remediate each other's undesirable effects [a]	0.63	0.38	1	
	Proportion of variance explained	0.68	0.53	0.52	

Exploratory factor analysis using maximum likelihood estimation produced three factors with positive Eigenvalues; results shown are after oblique Bentler's invariant pattern simplicity rotation with Kaiser normalization applied to 3-factor solution. [a] Refers to use of video games to remediate the undesirable effects of substances, and vice versa.

3. Results

Cases were valid for analysis if they reported at least one game title for which a genre could be discerned and recent use of at least one game with a substance (or *vice versa*), resulting in a total sample size of 1,196 concurrent users. Their mean age was 40.6 (SD = 15.1) and mean educational level (operationalized as an ordinal variable) corresponded to "some college, no degree," and mean income level (also ordinal) corresponded to $35,000–$39,999. The majority of concurrent users were white (76%), male (64%), living in an MSA (82%) and engaged in work for regular wages (53%). The average participant reported on 2.5 games out of the 5 the survey allowed, played their average game 11.5 days out of the past 30 with 2.9 hours per day played. They reported enjoying their average game "more than most games".

Mean consumer involvement was 2.3 (SD = 0.9), and mean problem video game play was 1.7 (SD = 0.7). Caffeine use in the past 30 days was reported by 78% (n = 940), tobacco by 45% (n = 540), alcohol by 43% (n = 512), marijuana by 11% (n = 136), painkillers by 11% (n = 131), and sedatives by 4% (n = 51). The modal response to all substance dependence measures was 1 on a scale of 1–5 for tobacco and 0 symptoms for all other substances. Means for Likert-scaled concurrent use situations and complementary use motivations variables were close to 1 on a scale of 1–5, and 34% of participants responded "not at all true" to all of them, indicating that about two-thirds of concurrent users had any conscious situational pattern or effect-seeking motivation for their concurrent use.

Table 1 reports the results of an exploratory factor analysis (EFA) of the pattern/motivation variables. Use while alone, use to cope with loneliness/depression, and use to pass time while feeling the effects of a substance loaded > 0.5 on a factor of concurrent use to pass time or regulate negative emotion. Use with certain friends and belief that certain substances enhance the experience of certain games loaded > 0.5 on a factor of concurrent use to enhance an already enjoyable or positive experience. Use of substances to cope with game-related frustration and use of video games to cope with hangover or other substance withdrawal loaded > 0.5 on a factor of concurrent use to remediate each other's undesirable effects (*i.e.*, video games to remediate the undesirable effects of substances and *vice-versa*). A confirmatory factor analysis of this 3-factor solution (using maximum likelihood estimation with robust standard errors) produced CFI = 0.962, standardized factor loadings between 0.624 and 0.764, and correlations among factors between 0.643 and 0.771. Factor scores from the EFA were saved and used as dependent variables in the following analysis.

Table 2 reports the results of a single multivariate analysis with days of concurrent use, hours/day of concurrent use, and the three pattern/motivation factors as dependent variables and demographics, general game playing variables, genres of concurrent use, and substance use frequency and problem use indicators as independent variables. Among demographic factors, younger age was associated only with concurrent use to enhance an already enjoyable or positive experience, lower education was associated only with longer concurrent use sessions, and non-working status was associated only with more days of concurrent use and use to pass time or regulate negative emotion.

Among game playing variables, days of video game play was only associated with days of concurrent use. Consumer involvement and game enjoyment did not have significant direct effects on any pattern/motivation. Hours/day of video game play and PVP were, however, associated with hours/day of concurrent use and all three pattern/motivation factors. Given that all five general game playing variables were correlated, with weighted Pearson r's between 0.13 and 0.59, all p's < 0.0001, it is suggestive that hours/day of video game play and PVP stood out as significant after statistical control. It is also noteworthy that, after controlling for all of these game playing factors, certain genres of concurrent use emerged as particularly compatible with certain patterns/motivations. The only genres not associated with either frequency variable or any pattern/motivation factor were MMORPG's, gambling, RTS, sports-general. The only dependent variable with no genre uniquely associated with it was concurrent use of video games and substances to remediate each other's undesirable effects.

Int. J. Environ. Res. Public Health **2011**, *8*

Table 2. Standardized coefficients from multivariate model predicting days of concurrent use, hours/day of concurrent use, and pattern/motivation factors from demographics, game playing variables, and genres/substances of concurrent use.

		Days of concurrent use	Hours/day concurrent use	Pass time or regulate negative emotion	Enhance positive experience	Remediate each other's undesirable effects
Age		0.04	0.07	0.03	−0.09 **	−0.03
Education		0.04	−0.11 **	−0.01	−0.04	0.02
Race: Black		−0.06 *	0.15 *	0.06 +	0.00	0.04
Race: Asian		−0.08*	−0.04	−0.01	0.05	0.04
Race: Native		−0.04	0.03	−0.04	−0.06 *	−0.02
Race: Other		−0.05 **	−0.01	0.00	−0.01	−0.01
Non-working		0.07 *	0.00	0.07 *	0.04	0.04
Days played average game		0.34 ***	0.06	0.03	−0.01	0.00
Hours/day played avg. game		0.01	0.23 ***	0.17 **	0.17 ***	0.17 *
Problem video game play		0.08 +	0.12 *	0.24 ***	0.13 **	0.26 ***
Genre: Action-adventure		0.06 *	0.08 *	0.04	0.08 *	0.01
Genre: Other RPG (not MMO)		0.08 **	0.03	0.08 *	0.10 **	0.00
Genre: First-person shooter (FPS)		0.11 ***	−0.01	0.10 **	0.10 *	0.06
Genre: Other shooter		0.06 *	0.07	−0.01	−0.01	−0.03
Genre: Other strategy (not RTS)		0.10 ***	0.01	−0.01	0.02	−0.04
Genre: Board/card		0.15 ***	0.03	0.10 ***	0.07 **	0.02
Genre: Other sports		0.07 **	0.00	0.04	0.11 **	0.01
Genre: Puzzle		0.15 ***	0.00	0.00	−0.01	−0.04
Genre: Rhythm		0.06 *	0.03	0.04	0.03	0.02
Genre: Platformer		0.05 *	0.02	0.08 *	0.02	0.03
Genre: Other		0.10 ***	0.03	0.03	−0.02	0.03
Caffeine days used		0.17 ***	0.08 +	−0.08 *	−0.07 *	−0.09 *
Caffeine use problems		−0.03	−0.08 +	0.12 ***	0.08 *	0.05
Tobacco use problems		0.05	0.16 +	0.19 **	0.05	0.23 **
Alcohol days used		0.07 *	−0.04	0.05	0.08 **	−0.02
Alcohol use problems		−0.11 ***	0.00	0.08 *	0.09 *	0.05
Marijuana days used		0.16 ***	−0.02	0.07	0.10 +	−0.06
Marijuana use problems		−0.08 *	0.06	0.15 *	0.21 **	0.15 *
Painkiller days used		−0.01	−0.08 *	−0.14 ***	−0.08**	−0.11 ***
Painkiller use problems		0.01	0.00	0.24 ***	0.12*	0.34 ***
Correlations among dependent variables:	Days concurrent use	1	0.10 **	0.16 ***	0.03	0.03
	Hours/day concurrent use	0.10 **	1	0.06	0.04	0.01
	Pass time/regulate emotion	0.16 ***	0.06	1	0.68 ***	0.70 ***
	Enhance pos. experience	0.03	0.04	0.68 ***	1	0.46 ***
	Remediate undesirable eff.	0.03	0.01	0.70 ***	0.46 ***	1
Variance explained (R^2)		0.38 ***	0.22 ***	0.45 ***	0.42 ***	0.38 ***
Overall model fit:		CFI > 0.999, RMSEA < 0.001, SRMR < 0.001				

Coefficients omitted from presentation in the table (they are included in the analysis) because $p > 0.05$ for all dependent variables include income; gender; race: Latino; MSA non-residence; enjoyment of average game; consumer involvement; the following genres: MMORPG, Gambling, RTS, Sports-general, Driving; days of tobacco use; days of sedative use; and sedative use problems. Reference category for race is white. + $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

With respect to substance use, almost all associations specified between indicators of substance use problems and pattern/motivation factors were significant. Negative coefficients for substance use frequency variables should be interpreted in light of bivariate analyses (not shown) in which substance use frequency was generally unrelated to concurrent use patterns/motivations; their significance in the multivariate context may be because of these indicators' collinearity with the use problems variables. The only substance of concurrent use not uniquely associated with any concurrent use frequency variable or pattern/motivation factor was sedatives.

4. Discussion

This study found that concurrent use with some situational pattern or effect-seeking motivation behind it is, like PVP [21], not universal among those who engage in the prerequisite behavior, but still observable in an appreciable fraction: 42% of the entire valid sample of video gamers were concurrent users, and 66% of concurrent users at least partially endorsed one or more patterns/motivations. The reliable correlation of most patterns/motivations with PVP and substance use problems suggests that concurrent use is part of a shared underlying addictive process [47], as other research on concurrent use of substances with self-reinforcing behaviors [3-6,48,49] suggests.

Moreover, demographics, genre of concurrent use, and substance of concurrent use variables were all uniquely and differentially related to concurrent use frequency and patterns/motivations. Concurrent use to enhance an already enjoyable or positive experience – either being with certain friends, or use of certain substances to enhance the experience of certain games – was more strongly endorsed by younger participants. An example of this factor's operation in context might be evident in ethnographic findings on parties among young marijuana users, where video games were commonly part of the entertainment [50]. Others of our results indicate differences among concurrent users in how concurrent use fits into the context of their lives, including that respondents with lower degrees of education reported longer hours/day of concurrent use, and non-working respondents reported greater degrees of use to pass time or regulate negative emotion.

Both intuitive and counterintuitive findings emerged for genre of concurrent use, which may further inquiry into connections between specific game features and problem video gaming behaviors [35]. In spite of the problem use potential of MMORPG's [37,38], concurrent users of MMORPG's were no more likely than others to endorse particular concurrent use patterns or motives. Other (non-MMO) RPG's, in contrast, were associated with more days of concurrent use, greater endorsement of use to pass time or regulate negative emotion, and higher degrees of concurrent use to enhance an already-positive or enjoyable experience. This may have to do with differences in game play—one possible explanation is that it is easier to use a substance with non-MMO RPG's because players can take non-MMO RPG's at their own pace and do not have to remain engaged with other players in real time. First-person shooters (FPS) were also distinct from other shooters, significantly related to the same factors as RPG's while other shooters were not. This may be because many FPS games are designed for team or competitive play among players gathered in person, and concurrent users use substances at these gatherings. Alternatively, perhaps the intensity of seeing the game from the character's perspective—e.g., bullets flying right at the player's face—occasionally causes a concurrent user to want something to calm them down. Board/card games were also associated with

both concurrent use to pass time or regulate negative emotion and concurrent use to enhance an already-enjoyable or positive experience while gambling games were not, perhaps because even fictional money is enough to alter the experience of a board/card game. Finally, the category of other sports games, which included mostly multiplayer team sports simulations, was associated with concurrent use to enhance a positive experience, while the mostly motion-control sports-general games were not. This could be because of a special association between team sports simulation games and gatherings involving substance use, or perhaps because use of some substances makes the coordinated full-body motion required to play motion control games more difficult.

Substance use problems were not positively related to simple frequency or duration of concurrent use. They were, however, reliably related to the pattern/motivation factors, with some exceptions: For example, caffeine is understandably not helpful for calming down from video game related frustration ("rage quit" in gamers' own parlance), nor does its withdrawal syndrome usually cause enough impairment to distract from normal activities. These and other non-significant findings among the general pattern of significant associations between substance use problems and pattern/motivation factors are potentially relevant to the concern of addiction specificity [34], in that every behavior/substance combination may have its own unique complementarity of effects, context of use, expectations among users as to what they will get out of it, and other unique considerations which ultimately affect which patterns of addictive behaviors will develop. Our measures were not, however, set up to directly operationalize addiction specificity itself, e.g., clinically significant substance use problems without PVP and *vice-versa*. It will be up to future work to explore those associations. Ideally, questions about media and other behavioral addictions would be added to existing nationally representative panel studies addressing substance use and health like NSDUH [44] or Monitoring the Future [51].

One way in which this study uniquely contributes to the literature is that, rather than focusing on specific game genres, game features, playing behaviors, or substances, it allowed for an open field of possible correlates. It also departed from this area's frequent focus on youth problem behavior [15] by including a nationally representative sample of adults. It is limited, however, in that our survey's 10-minute median length meant that established measures [42,44] had to be abridged. Hence, our references to "substance use problems" rather than actual abuse/dependence diagnoses. Also, as an internet survey, it was vulnerable to same risk of invalid response endemic to any survey method that does not have interviewers engaged in-person with participants. One symptom of this was that 15% of our respondents did not provide even one valid video game title. The online nature of the survey in and of itself, however, probably cannot be argued to be a limitation, as experiments have shown point estimates to be consistent across methods of obtaining random, representative survey samples [52]. Finally, our cross-sectional data can only contemporaneous association; they cannot address development of motivations for and patterns of behavior over time.

Although this study's results did not coalesce into a simple story, we believe that this is actually a more authentic representation of the phenomenon under study than could have been achieved by focusing on specific demographics, substances, gaming behaviors, and game genres, or by oversimplifying the question into "are video games addictive?" Our results represent a complex issue in its complexity, suggesting that social situation, playing behaviors, genre and substance of concurrent

use, and motivations for concurrent are all potentially relevant to the effect of concurrent use on individuals' lives.

Acknowledgments

This research was supported by grant R01-DA027761, "Video Games' Role in Developing Substance Use", from the National Institute of Drug Abuse.

Conflict of Interest

None of the authors has any conflict of interest that might affect the integrity of the results.

References

1. Wareham, J.D.; Potenza, M.N. Pathological gambling and substance use disorders. *Am. J. Drug Alcohol Abuse* **2010**, *36*, 242-247.
2. Ellery, M.; Stewart, S.H.; Loba, P. Alcohol's effects on video lottery terminal (vlt) play among probable pathological and non-pathological gamblers. *J. Gambl. Stud.* **2005**, *21*, 299-324.
3. Cronce, J.M.; Corbin, W.R. Effects of alcohol and initial gambling outcomes on within-session gambling behavior. *Exp. Clin. Psychopharmacol.* **2010**, *18*, 145-157.
4. Dickerson, M.; Baron, E. Contemporary issues and future directions for research into pathological gambling. *Addiction* **2000**, *95*, 1145-1159.
5. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority? *Eval. Health Prof.* **2011**, *34*, 3-56.
6. López-Moreno, J.A.; González-Cuevas, G.; Moreno, G.; Navarro, M. The pharmacology of the endocannabinoid system: Functional and structural interactions with other neurotransmitter systems and their repercussions in behavioral addiction. *Add. Biol.* **2008**, *13*, 160-187.
7. Robbins, T.W.; Cador, M.; Taylor, J.R.; Everitt, B.J. Limbic-striatal interactions in reward-related processes. *Neurosci. Biobehav. Rev.* **1989**, *13*, 155-162.
8. De Lecea, L.; Jones, B.E.; Boutrel, B.; Borgland, S.L.; Nishino, S.; Bubser, M.; DiLeone, R. Addiction and arousal: Alternative roles of hypothalamic peptides. *J. Neurosci.* **2006**, *26*, 10372-10375.
9. Han, D.H.; Bolo, N.; Daniels, M.A.; Arenella, L.; Lyood, I.K.; Renshawe, P.F. Brain activity and desire for internet video game play. *Compr. Psychiatr.* **2011**, *52*, 88-95.
10. Han, D.H.; Kim, Y.S.; Lee, Y.S.; Min, K.J.; Renshaw, P.F. Changes in cue-induced, prefrontal cortex activity with video-game play. *Cyberpsychol. Behav. Soc. Netw.* **2010**, *13*, 655-661.
11. Weinstein, A.M. Computer and video game addiction—A comparison between game users and non-game users. *Am. J. Drug Alcohol Abuse* **2010**, *36*, 268-276.
12. Wood, R.T.A. Problems with the concept of video game "addiction": Some case study examples. *Int. J. Mental Health Addict.* **2008**, *6*, 169-178.
13. Steenhuysen, J. *Addiction Experts Say Video Games Not An Addiction*. 2007. Available online: http://www.reuters.com/article/idUSN2425415820070624 (accessed on 19 January 2011).

14. American Psychiatric Association. *Substance Use and Addictive Disorders*; American Psychiatric Association: Arlington, VA, USA, 2010. Available online: http://www.dsm5.org/Proposed Revision/Pages/SubstanceUseandAddictiveDisorders.aspx (accessed on 13 July 2011).
15. Kuss, D.; Griffiths, M. Internet gaming addiction: A systematic review of empirical research. *Int. J. Mental Health Addict.* **2011**, doi: 10.1007/s11469-011-9318-5.
16. Petry, N.M. Commentary on Van Rooij *et al.* (2011): 'Gaming addiction'—A psychiatric disorder or not? *Addiction* **2011**, *106*, 213-214.
17. Grüsser, S.M.; Thalemann, R.; Griffiths, M.D. Excessive computer game playing: Evidence for addiction and aggression? *Cyberpsychol. Behav.* **2007**, *10*, 290-292.
18. Porter, G.; Starcevic, V.; Berle, D.; Fenech, P. Recognizing problem video game use. *Aust. N. Z. J. Psychiatr.* **2010**, *44*, 120-128.
19. Gentile, D.A.; Choo, H.; Liau, A.; Sim, T.; Li, D.; Fung, D.; Khoo, A. Pathological video game use among youths: A two-year longitudinal study. *Pediatrics* **2011**, doi: 10.1542/peds.2010-1353.
20. Gentile, D.A. Pathological video-game use among youth ages 8 to 18: A national study. *Psychol. Sci.* **2009**, *20*, 594-602.
21. Desai, R.A.; Krishnan-Sarin, S.; Cavallo, D.; Potenza, M.N. Video-gaming among high school students: Health correlates, gender differences, and problematic gaming. *Pediatrics* **2010**, doi: 10.1542/peds.2009-2706.
22. Rehbein, F.; Psych, G.; Kleimann, M.; Mediasci, G.; Moßle, T. Prevalence and risk factors of video game dependency in adolescence: Results of a german nationwide survey. *Cyberpsychol. Behav. Soc. Netw.* **2010**, *13*, 269-277.
23. Young, K.S. Understanding online gaming addiction and treatment issues for adolescents. *Am. J. Fam. Ther.* **2009**, *37*, 355-372.
24. Griffiths, M.; Meredith, A. Videogame addiction and its treatment. *J. Contemp. Psychother.* **2009**, *39*, 247-253.
25. Khan, M.K.; Kantof, E.P. *Emotional and Behavioral Effects, Including Addictive Potential, of Video Games*; American Medical Association: Chicago, IL, USA, 2007. Available online: www.ama-assn.org/ama1/pub/upload/mm/467/csaph12a07.doc (accessed on 19 January 2011).
26. Swing, E.L.; Gentile, D.A.; Anderson, C.A.; Walsh, D.A. Television and video game exposure and the development of attention problems. *Pediatrics* **2010**, *126*, 214-221.
27. Rowan, C. Unplug—Don't drug: A critical look at the influence of technology on child behavior with an alternative way of responding other than evaluation and drugging. *Ethic. Hum. Psychol. Psychiatr.* **2010**, *12*, 60-68.
28. Ferguson, C.J. The influence of television and video game use on attention and school problems: A multivariate analysis with other risk factors controlled. *J. Psychiatr. Res.* **2011**, *45*, 808-813.
29. Han, D.H.; Hwang, J.W.; Renshaw, P.F. Bupropion sustained release treatment decreases craving for video games and cue-induced brain activity in patients with internet video game addiction. *Exp. Clin. Psychopharmacol.* **2010**, *18*, 297-304.
30. Han, D.H.; Lee, Y.S.; Na, C.; Ahn, J.Y.; Chung, U.S.; Daniels, M.A.; Haws, C.A.; Renshaw, P.F. The effect of methylphenidate on internet video game play in children with attention-deficit/hyperactivity disorder. *Compr. Psychiatr.* **2009**, *50*, 251-256.

31. Greenberg, J.L.; Lewis, S.E.; Dodd, D.K. Overlapping addictions and self-esteem among college men and women. *Addict. Behav.* **1999**, *24*, 565-571.
32. Rozin, P.; Stoess, C. Is there a general tendency to become addicted? *Addict. Behav.* **1993**, *18*, 81-87.
33. Padilla-Walker, L.M.; Nelson, L.J.; Carroll, J.S.; Jensen, A.C. More than a just a game: Video game and internet use during emerging adulthood. *J. Youth Adolesc.* **2010**, *39*, 103-113.
34. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
35. King, D.; Delfabbro, P.; Griffiths, M. Video game structural characteristics: A new psychological taxonomy. *Int. J. Mental Health Addict.* **2010**, *8*, 90-106.
36. Lee, M.S.; Ko, Y.H.; Song, H.S.; Kwon, K.H.; Lee, H.S.; Nam, M.; Jung, I.K. Characteristics of internet use in relation to game genre in Korean adolescents. *Cyberpsychol. Behav.* **2007**, *10*, 278-285.
37. Smyth, J.M. Beyond self-selection in video game play: An experimental examination of the consequences of massively multiplayer online role-playing game play. *Cyberpsychol. Behav.* **2007**, *10*, 717-727.
38. Stetina, B.U.; Kothgassner, O.D.; Lehenbauer, M.; Kryspin-Exner, I. Beyond the fascination of online-games: Probing addictive behavior and depression in the world of online-gaming. *Comput. Hum. Behav.* **2011**, *27*, 473-479.
39. Laurent, G.; Kapferer, J.-N. Measuring consumer involvement profiles. *J. Market. Res.* **1985**, *22*, 41-53.
40. Wiley, C.G.E.; Shaw, S.M.; Havitz, M.E. Men's and women's involvement in sports: An examination of the gendered aspects of leisure involvement. *Leisure Sci.* **2000**, *22*, 19-31.
41. Gursoy, D.; Gavcar, E. International leisure tourists' involvement profile. *Ann. Tourism Res.* **2003**, *30*, 906-926.
42. Tejeiro Salguero, R.A.; Bersabé Morán, R.M. Measuring problem video game playing in adolescents. *Addiction* **2002**, *97*, 1601-1606.
43. CBS Interactive Inc. *Video Game Cheats, Reviews, Faqs, Message Boards, and More—Gamefaqs*; CBS Interactive Inc.: San Francisco, CA, USA, 2011. Available online: http://www.gamefaqs.com/ (accessed on 15 February 2011).
44. Substance Abuse and Mental Health Statistics (SAMHSA). *Methodology Reports for the National Household Survey on Drug abuse & the National Survey on Drug Use & Health*; SAMHSA: Rockville, MD, USA, 2009. Available online: http://oas.samhsa.gov/nsduh/methods.cfm (accessed on 25 November 2010).
45. Kenny, D.A. *Measuring Model Fit*; 2011. Available online: http://www.davidakenny.net/cm/fit.htm (accessed on 12 September 2011).
46. DiSogra, C. *Overview of Knowledgepanel® Statistical Weighting Protocol*; Knowledge Networks: New York, NY, USA, 2009. Available online: http://www.knowledgenetworks.com/ganp/docs/kn-weighting-synopsis.pdf (accessed on 25 April 2011).
47. Thalemann, R.; Wölfling, K.; Grüsser, S.M. Specific cue reactivity on computer game-related cues in excessive gamers. *Behav. Neurosci.* **2007**, *121*, 614-618.

48. Ernst, M.; Grant, S.J.; London, E.D.; Contoreggi, C.S.; Kimes, A.S.; Spurgeon, L. Decision making in adolescents with behavior disorders and adults with substance abuse. *Am. J. Psychiatr.* **2003**, *160*, 33-42.
49. Schutter, D.J.L.G.; van Bokhoven, I.; Vanderschuren, L.J.M.J.; Lochman, J.E.; Matthys, W. Risky decision making in substance dependent adolescents with a disruptive behavior disorder. *J. Abnorm. Child Psychol.* **2011**, *39*, 333-339.
50. Dunlap, E.; Johnson, B.D.; Sifaneck, S.J.; Benoit, E. Sessions, cyphers, and parties: Settings for informal social controls of blunt smoking. *J. Ethn. Subst. Abuse* **2005**, *4*, 43-80.
51. Monitoring the Future (MTF). *Monitoring the Future: A Continuing Study of American Youth*; MTF: Ann Arbor, MI, USA, 2011; Available online: http://monitoringthefuture.org/ (accessed on 11 October 2011).
52. Zukin, C. *Game Change: The Challenge of Finding and Interviewing a Random Sample in 2010*; Knowledge Networks Seminars, New York, NY, USA, 28 October 2010.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Ream, G.L.; Elliott, L.C.; Dunlap, E. Playing Video Games While Using or Feeling the Effects of Substances: Associations with Substance Use Problems. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3979-3998; doi:10.3390/ijerph8103979.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Article

Playing Video Games While Using or Feeling the Effects of Substances: Associations with Substance Use Problems

Geoffrey L. Ream [1,*], Luther C. Elliott [2] and Eloise Dunlap [2]

[1] School of Social Work, Adelphi University, 1 South Avenue, Garden City, NY 11530, USA
[2] Institute for Special Populations Research, National Development and Research Institutes, 8th Floor, 71 West 23rd Street, New York, NY 10010, USA; E-Mails: luther@nyu.edu (L.C.E.); dunlap@ndri.org (E.D.)

* Author to whom correspondence should be addressed; E-Mail: ream@adelphi.edu; Tel.: +1-516-877-4432; Fax: +1-516-877-4392.

Received: 19 August 2011; in revised form: 13 October 2011 / Accepted: 13 October 2011 / Published: 18 October 2011

Abstract: This study tested the hypothesis that playing video games while using or feeling the effects of a substance—referred to herein as "concurrent use"—is related to substance use problems after controlling for substance use frequency, video gaming as an enthusiastic hobby, and demographic factors. Data were drawn from a nationally representative online survey of adult video gamers conducted by Knowledge Networks, valid n = 2,885. Problem video game playing behavior was operationalized using Tejeiro Salguero and Bersabé Morán's 2002 problem video game play (PVP) measure, and measures for substance use problems were taken from the National Survey of Drug Use and Health (NSDUH). Separate structural equation modeling analyses were conducted for users of caffeine, tobacco, alcohol, and marijuana. In all four models, concurrent use was directly associated with substance use problems, but not with PVP. Video gaming as an enthusiastic hobby was associated with substance use problems via two indirect paths: through PVP for all substances, and through concurrent use for caffeine, tobacco, and alcohol only. Results illustrate the potential for "drug interaction" between self-reinforcing behaviors and addictive substances, with implications for the development of problem use.

Keywords: video games; caffeine; tobacco; alcohol; marijuana; addiction; dependence

Int. J. Environ. Res. Public Health **2011**, *8*

1. Introduction

Between self-reinforcing behaviors and substances, there may exist a "drug interaction" which exacerbates addictive patterns. Addictive substances stimulate the brain's natural reward pathways [1], and behavioral addictions involve the endogenous cannabinoid and related brain systems [2]. Dopamine plays a role in the self-reinforcing nature of substance use [3-5], and elevated dopamine plays a "gain-amplifying role" [6] in responses to "rewarding" Pavlovian stimuli [7]. As would be expected based on these shared biological mechanisms, survey studies find relationships between substance use problems and self-reinforcing behaviors like eating, gambling, internet use, sex, exercise, "workaholism," shopping, television viewing, and video games [8-13].

Such a "drug interaction" between self-reinforcing behaviors and addictive substances has been discovered in survey and laboratory findings that alcohol exacerbates problem gambling [14-18]. Part of the explanation offered for this is that alcohol contributes to disinhibition and risky decision making [19]. If this "drug interaction" effect is not, however, unique to the specific context of the alcohol/gambling combination but part of a larger pattern in which behavioral and substance addictions contribute to each other, then it should be evident in other substance/behavior combinations. It should also hold that engaging in self-reinforcing behavior while using or feeling the effects of a substance is uniquely associated with not only behavioral addiction, but substance use problems as well. In the present study, we focus on video game play. Video gaming is both similar to and distinct from gambling [20] in its self-reinforcing features [21] and degrees of prevalence and social acceptability, which all contribute to its unique characteristics as an addictive behavior [13].

The premise that video game playing is a potentially addictive behavior has empirical support [22]. Although the idea of video game addiction is not universally accepted [23], particularly as a discrete diagnostic category [24], and is not apparently likely to be included in the forthcoming DSM-V [25], problem video gaming behavior remains an area of scholarly and clinical [26,27] concern. A report by the American Medical Association's Council on Science and Public Health called for more research on it [28]. A growing body of research on problem video gaming behaviors has emerged from within work on problem gambling [29] and adapted its measures [27], which originally came from Diagnostic and Statistical Manual (DSM) substance abuse/dependence criteria [30,31]. Prevalence estimates of significant problem video gaming behavior hover between 4.9% and 9% among video gamers internationally [11,32-35], with smaller numbers fitting stricter criteria for dependence [22,36].

Video game addiction has identifiable biological dimensions, which are similar to those for other behavioral addictions as well as substance dependence. An electroencephalography study found differences in processing of game-related cues between "casual" and "excessive" video game players suggestive of an addiction-like process among the "excessive" players [37]. Video game play stimulates dopamine [38] through the "reward" structures programmed into video games [39,40]. Dopaminergic medications indicated for ADHD and substance dependence help remediate problem video gaming behavior [41,42], and video games are associated with development of attention problems in children [43,44].

In addition to shared biological mechanisms, problem video gaming behavior and substance use have several risk correlates in common. Examples include mood disorders [33,45,46], impulsivity [33,47], attention deficit hyperactivity disorder symptoms [42,48-52], low social

competence [53,54], low academic performance [55-58], and (in the case of violent video games) violence [59,60]. Although many of these studies on negative effects of problem video gaming behavior focus on children and youth [24], there are similar findings on adults [22], which implies that it would not be accurate to frame video game addiction as a youth problem behavior. Additional studies on adults would help distinguish problem video gaming behavior as an issue with addiction, rather than something parsimoniously explained by youth problem behavior theory [61].

Given shared biological mechanisms and risk correlates of problem video gaming behavior and substance use problems, as well as the general trend for behavioral and substance addictions to co-occur [13], this study investigated unique potential of playing video games while using or feeling the effects of a substance—referred to herein as "concurrent use"—to contribute to problem video gaming behavior and substance use problems. Its hypothesis was that concurrent use would explain variance in problem video gaming behavior and substance use problems that would not be explained by substance use frequency, video gaming as an enthusiastic hobby (video game play frequency, enjoyment, and consumer involvement), or demographics, including gender [30,34,62,63], race [64], age, and socioeconomic indicators. Analyses tested this hypothesis with respect to caffeine, tobacco, alcohol, and marijuana.

2. Methods

2.1. Participants and Recruitment

Participants were a subset of a nationally representative KnowledgePanel® maintained by Knowledge Networks, a commercial online survey service provider. Knowledge Networks selects panel members via random-digit dial and address-based sampling methods, provides panel members with computers and internet access if needed, establishes informed consent, and collects basic demographic information. Once in the panel, members are randomly recruited via e-mail for client surveys, including this study. For each survey, participants receive "points" toward cash and other incentives offered by Knowledge Networks. For some client surveys, panel members are presented with screening questions and only allowed to participate if they meet specific criteria. For this survey, 15,642 e-mails were sent to panel members age 18 and over, and 9,215 (59%) completed the screening instrument. The valid sample was 42% female, 69% white, 11% Black, 13% Latino, 4% Asian, 2% Native (includes American Indian, Alaska Native, Native Hawaiian, and Pacific Islander), and 2% multiracial or other. More than half (58%) were currently either employed for wages or self-employed. The average participant was 40.4 years old (SD = 15.7), with annual income between $35,000 and $39,999, and had some college education but no degree.

The screener asked whether participants "regularly," "occasionally," or "never" participated in 11 different hobby activities in the past year, including video games. Participants who responded "regularly" or "occasionally" about video games were then asked how many hours of video and/or computer games they played in the past seven days. Participants who reported one or more hours, n = 3,380 (37%), were allowed to take the survey. Screening and the survey itself were conducted in English and Spanish. The Spanish version was professionally translated by Knowledge Networks using multiple translators and back-translation. It was also reviewed by the first author, who is fluent

in written Spanish. Completion of the measure took an average of 10 minutes, which was the maximum median length feasible given constraints of the method and budget. The protocol for this study was reviewed and approved by all investigators' Institutional Review Boards.

2.2. Measures

Substance use frequency. These measures were adapted from the National Survey of Drug Use and Health. (NSDUH, [65]). Participants chose, from a list, those substances they had used in the past 30 days. For each they chose, they were asked on how many of the past 30 days they had used it.

Substance use problems. NSDUH-based [65] abuse and dependence symptom items were presented for each substance used in the past 30 days. A set of questions was adapted for caffeine by using the full set of abuse/dependence items and leaving out those inapplicable to it (e.g., spending a lot of time obtaining/using/recovering from it, neglecting work and social life in order to use it). Because the full-length measures could not realistically be included in a survey constrained to a median length of 10 minutes, subsets were selected based on factor analyses of another dataset collected in this project (not yet published) which did include full-length measures—seven dichotomous items for caffeine, 11 Likert-scale items for tobacco, and 14 dichotomous items for each of alcohol and marijuana. For sets of dichotomous items, tetrachoric correlation matrices were factor analyzed. The goal of item selection was not to reproduce DSM-IV diagnoses but to measure problem substance use as a matter of degrees as authentically as possible within time constraints. Items were chosen that were highly correlated with the first/only factor, but not redundant—when collinearity resulted from everyone who reported one symptom also having reported another symptom, the less-frequently-reported symptom was left out. The final 5-item measure for caffeine included symptoms of tolerance, difficulty controlling use, desire to quit or cut down, withdrawal, and disregarding negative emotional or physical health consequences of use, CFI from confirmatory factor analysis = 0.962. Final measures for alcohol (CFI = 0.997) and marijuana (CFI = 0.998) included these plus neglecting positive activities and spending a lot of time obtaining or using, a total of 7 items each. The final measure for tobacco included four Likert-scale items for symptoms presently experienced including withdrawal, craving, worry over running out, and tolerance, CFI = 0.990.

Concurrent use with video games. For each substance that participants reported having used in the past 30 days, they were asked, "During the past 30 days, have you played video games while using (substance in question) or feeling its effects?"

Video game use and enjoyment. Participants were asked to list, via text entry, up to five video game titles they had "spent a lot of time playing in the past 12 months." They were asked a series of questions about each title, including on how many days of the past 30 they had played it and how much they enjoyed it. Enjoyment was a single 7-point Likert scale in which 1 indicated "it was the worst game I've ever played," four indicated "about the same as most games," and 7 indicated "it was my single all-time favorite." Because all dependent variables in the present analyses were person-level, within-person means were calculated to reflect the properties of the average game that person played. Entries for use and enjoyment variables were only considered valid for 2,885 participants who entered at least one valid title, and titles were considered valid if they could be uniquely identified in

GameFaqs [66], a large and comprehensive database of user-generated content maintained and edited by an industry group.

Consumer involvement in video games. Another indicator of video game playing as an enthusiastic hobby [27,67-69] as distinct from addiction [70] was a measure of consumer involvement adapted from leisure and marketing studies [71-73]. It addresses attraction, centrality/importance, and self-expression, Cronbach's $\alpha = 0.70$.

Problem video game playing (PVP). Because there is still debate about the definition of video game abuse/dependence [23,74,75], and symptoms of any disorder may constitute a problem in living even if they do not meet a clinical threshold, this study operationalized problem video gaming behavior with a continuous Likert scale measure (PVP, [31]). Like our measures for substance use problems, this scale also had to be abridged to fit into the 10-minute median time limit for this survey. Items were selected based on factor analysis of the same data we used to derive the substance use measures. For that study, the original 9-item Likert scale PVP measure had been slightly edited by splitting the longest item ("I have tried to control, cut back, or stop playing, or I usually play with the video games over a longer period than I intended") into two separate items and deleting "with the" to produce a measure with a total of 10 items. The five highest loading items on the first/only factor across all four estimation procedures available in STATA 11.0 (principal factor, principal-component factor, iterated principal factor, and maximum likelihood factor) addressed increasing time spent playing (tolerance), difficulty controlling time spent playing, restlessness/irritability when can't play (withdrawal), play to relieve negative affect (self-medication), and engaging in problem behavior in order to play games, CFI = 0.959, $\alpha = 0.74$. Response choices ranged from "not at all true" to "extremely true," so that participants who scored anywhere above the lowest possible score at least slightly positively identified with at least one item.

Demographics. Age, race/ethnicity, gender, education, income, and employment status were taken from the Knowledge Networks' basic demographic survey. No data were missing on these variables. Income was categorized into 19 increments beginning with "less than $5,000" that are increasingly larger further up the scale until the final category, "$175,000 or more." Education was an ordinal variable with 14 possible categories ranging from "no formal education" to "professional or doctoral degree." Employment, for purposes of these analyses, was collapsed into categories of (1) working, either for wages or self-employed, or (2) non-working for any reason, e.g., disability, retirement, layoff, *etc*.

2.3. Approach to Analyses

All analyses employed post-stratification weights provided by Knowledge Networks so that estimates approximate what would have been obtained from a true random sample of English- and Spanish-speaking American adult video game players [76]. Calculated based on current data from the U.S. Census Current Population Survey, Knowledge Networks' weights adjust for survey non-response and client surveys' own sample designs, such as our screening procedure. We only present weighted estimates in the results section.

Bivariate relationships among study variables were computed using scale values for consumer involvement, PVP, and tobacco dependence, and count variables for the number of abuse/dependence

symptoms for caffeine, alcohol, and marijuana. For these tables, significance flags were adjusted relative to convention in light of multiple tests, so that * indicates $p < 0.01$ rather than $p < 0.05$, ** indicates $p < 0.001$ rather than $p < 0.01$, *etc*.

For main hypothesis tests, structural equation models were run in MPlus 6.0, separately by substance. In each model, PVP and substance use problems were continuous latent variables measured by their observed components and allowed to correlate. MPlus is capable of creating a continuous latent variable from binary observed indicators, and this was done for the five indicators of caffeine use problems and the seven indicators of each of marijuana and alcohol use problems. PVP and substance use problems were regressed over the concurrent use binary variable, the observed indicator of substance use frequency, and a latent indicator of video gaming as an enthusiastic hobby. In a separate statement, concurrent use was regressed over substance use frequency and the latent variable for video gaming as an enthusiastic hobby. Substance use frequency and the latent variable for video gaming as an enthusiastic hobby were allowed to correlate. The latent variable of video gaming as an enthusiastic hobby was measured by the observed indicators of game enjoyment, hours played, and the scale score for consumer involvement. In the last statement specifying the model, all of these variables were regressed over demographic variables, so that the structural models described below refer to effects after demographic controls. The binary variable for concurrent use was accommodated in a mediating role through theta parameterization, and post-stratification weights were accommodated using means and variance adjusted weighted least squares (WLSMV) estimation.

Models were determined to have acceptable fit based on RMSEA < 0.05, even though the lowest CFI was 0.89 and the highest was 0.94, which are just below the usual strict standard of 0.95 [77,78]. Alternative specifications were attempted that included only either the observed indicator for consumer involvement or video game play frequency in place of the "enthusiastic hobby" factor, and left "problem behavior to play games" out of PVP. These yielded CFI $\geq$ 0.95 for all four models and significant paths between concurrent use and PVP for caffeine, tobacco, and marijuana users. However, this specification would have limited the conceptual scope of the study. Observed components of the enthusiastic hobby factor were selected for conceptual completeness according to the logic of a formative indicator [79]; they were not expected to be redundant. Including all components of the enthusiastic hobby indicator was necessary in order to fully distinguish problem video gaming behavior from mere engagement [27]. Without them, we could not be sure the significant paths between concurrent use and PVP were not type I error due to underspecification. Another choice made for conceptual completeness was to leave the PVP measure intact despite the low-loading item and not reduce it even further relative to the original. Given that all RMSEAs were still < 0.05 [77,78] and some diminishment of CFI is forgivable when including variables that are not expected to be correlated but still need to be in the model for conceptual reasons [80], we determined the models described in Figures 1–4 to be the most authentic representation of the findings among the possibilities.

3. Results

Table 1 provides descriptive statistics on video game and substance use variables, as well as differences by categorical demographic factors.

Table 1. Overall means/proportions for game playing and substance use variables, and differences by demographic factors.

		Overall mean*(sd)* or proportion	Gender		Race						Working	
			Male	Female	White	Black	Latino	Asian	Native	Other	Yes	No
Video Games	Days played average game	9.8(*8.5*)	9.5	10.2+	9.9(ref)	9.3	9.3	12.2 *	9.2	7.9	8.6	11.4 ***
weighted	Enjoyment of average game	5.2(*1.1*)	5.19	5.30 *	5.21(ref)	5.44 ***	5.24	5.14	5.30	5.12	5.22	5.26
n(users) =	Consumer involvement	2.1(*0.8*)	2.23	2.03 ***	2.12(ref)	2.38 ***	2.02 +	2.18	2.44 *	2.20	2.10	2.20 *
2,869.5	Problem video game play	1.6(*0.6*)	1.58	1.53 +	1.53(ref)	1.64 *	1.54	1.73 *	1.78 *	1.64	1.50	1.64 ***
Caffeine	Any use of caffeine	64%	63%	67% +	70%(ref)	44% ***	51% ***	63%	72%	66%	66%	62%
weighted	Days/past 30 used caffeine [a]	24.4(*8.7*)	24.4	24.4	25.6(ref)	19.6 ***	20.8 ***	22.0 **	21.5 *	23.2	24.6	24.1
n(users) =	Caffeine use problems [a]	1.08(*1.40*)	1.01	1.18 *	1.07(ref)	1.02	1.08	1.32	1.41	1.01	1.06	1.12
1,849.3	Concurrent use with caffeine [a]	41%	45%	35% ***	44%(ref)	34% +	27% ***	19% **	35%	47%	39%	43%
Tobacco	Any use of tobacco	26%	27%	25%	27%(ref)	26%	19% *	19%	44% +	27%	23%	30% **
weighted	Days/past 30 used tobacco [a]	24.0(*10.6*)	22.0	27.2 ***	24.4(ref)	24.6	22.6	26.4	19.8 +	19.0+	22.4	25.8 ***
n(users) =	Tobacco use problems [a]	2.55(*1.20*)	2.37	2.83 ***	2.58(ref)	2.51	2.32	3.19 +	2.44	2.08	2.36	2.76 ***
748.7	Concurrent use with tobacco [a]	61%	62%	59%	61%(ref)	66%	49%	28% *	76%	89%+	52%	70% ***
Alcohol	Any use of alcohol	34%	40%	25% ***	35%(ref)	33%	31%	15% **	38%	41%	37%	29% ***
weighted	Days/past 30 used alcohol [a]	11.0(*8.8*)	11.3	10.3	11.8(ref)	9.0 *	8.4	8.0	8.2 **	12.6	10.4	12.0 *
n(users) =	Alcohol use problems [a]	1.33(*1.70*)	1.34	1.33	1.28(ref)	1.44	1.50	0.74	2.86 **	0.97	1.21	1.55*
964.6	Concurrent use with alcohol [a]	38%	42%	28% **	40%(ref)	42%	21%	14%	46%	38%	34%	44%*
Marijuana	Any use of marijuana	5.6%	6.4%	4.7%	5.7%(ref)	7.4%	5.5% **	0%	3.5%	7.5%	5.0%	6.6%
weighted	Days/past 30 used marijuana [a]	19.1(*11.3*)	19.7	18.1	19.1(ref)	17.3	21.1	[b]	[b]	16.9	19.5	18.8
n(users) =	Marijuana use problems [a]	2.28(*2.10*)	2.50	1.86	2.07(ref)	2.80	2.91	[b]	[b]	2.34	1.98	2.60
162.0	Concurrent use with marijuana [a]	80%	84%	72%	80%(ref)	76%	77%	[b]	[b]	100%	78%	81%

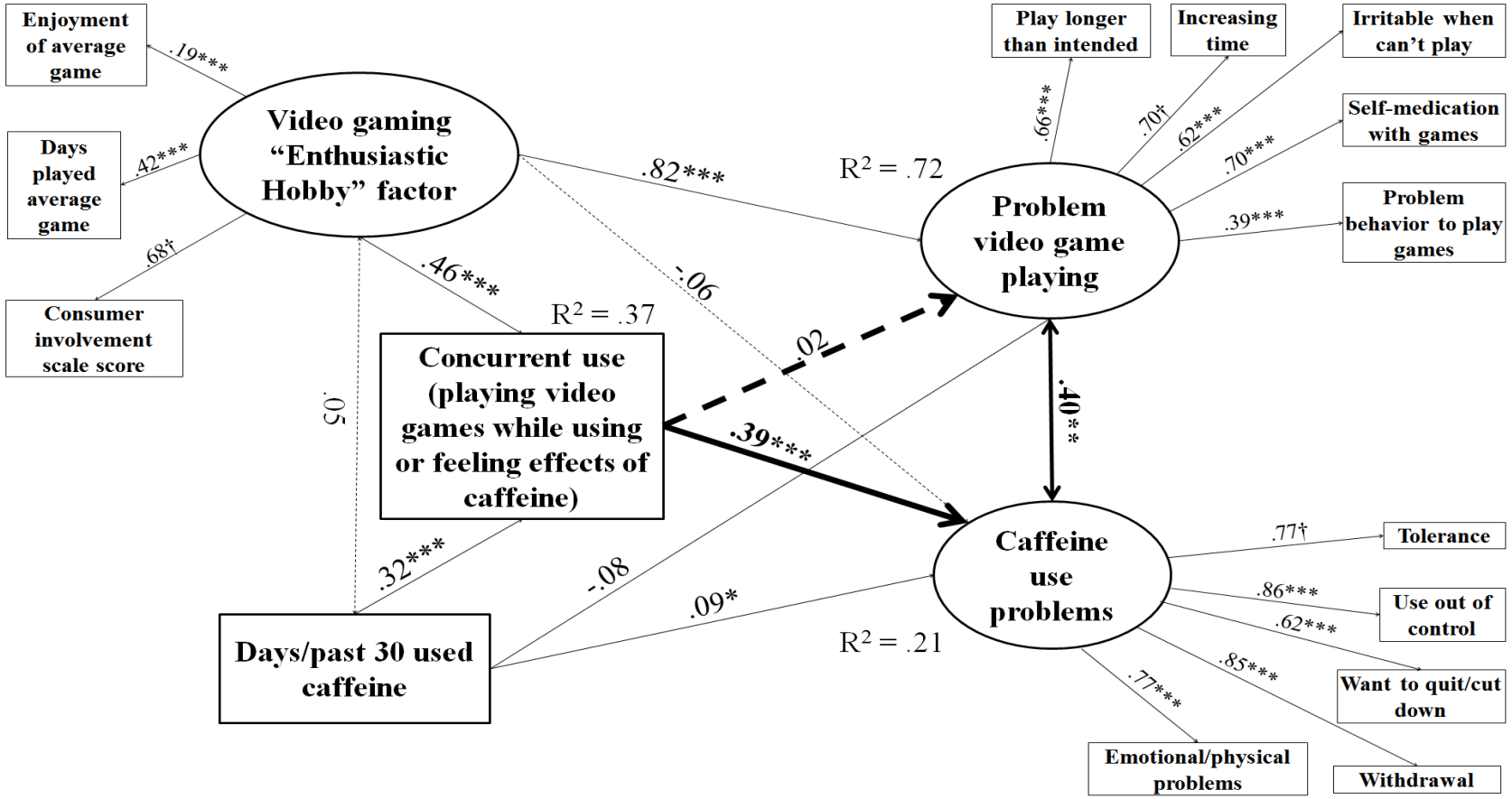
[a] Only users of the substance in question included in these substance-specific analysis. [b] Figures based on <5 real cases omitted. + $p < 0.05$, * $p < 0.01$, ** $p < 0.001$, *** $p < 0.0001$.

Table 2. Correlations among game playing, substance use, and continuous demographic variables.

	Days played	Game enjoyment	Consumer involvement	Problem play (PVP)	Caffeine days[a]	Caffeine problems[a]	Tobacco days[a]	Tobacco problems[a]	Alcohol days[a]	Alcohol problems[a]	Marijuana days[a]	Marijuana problems[a]
Enjoyment of avg. game	0.14***	1	0.18***	0.16***	0.02	0.07*	0.05	0.08+	-0.04	0.04	0.12	0.13
Consumer involvement	0.21***	0.18***	1	0.57***	-0.06*	0.07*	0.06	0.13**	-0.05	0.14***	0.07	0.19+
Problem video game play	0.28***	0.16***	0.57***	1	-0.08**	0.24***	0.08+	0.33***	-0.03	0.22***	-0.06	0.27*
Days caffeine use	0.11***	0.02	-0.06*	-0.08**	1	0.09**	0.14*	0.08	0.16***	-0.06	0.44***	-0.10
Caffeine use problems	0.01	0.07*	0.07*	0.24***	0.09**	1	0.02	0.33***	-0.12*	0.37***	0.16	0.51***
Days tobacco use	0.17***	0.05	0.06	0.08+	0.14*	0.02	1	0.48***	0.11	-0.01	0.26+	-0.21
Tobacco use problems	0.16***	0.08+	0.13**	0.33***	0.08	0.33***	0.48***	1	0.03	0.21**	0.08	-0.11
Days used alcohol	0.08+	-0.04	-0.05	-0.03	0.16***	-0.12*	0.11	0.03	1	0.23***	0.16	-0.25+
Alcohol use problems	0.02	0.04	0.14***	0.22***	-0.06	0.37***	-0.01	0.21**	0.23***	1	0.01	0.31*
Days used marijuana	0.18+	0.12	0.07	-0.06	0.44***	0.16	0.26+	0.08	0.16	0.01	1	0.24*
Marijuana use problems	0.24*	0.13	0.19+	0.27*	-0.10	0.51***	-0.21	-0.11	-0.25+	0.31*	0.24*	1
Age	0.22***	-0.13***	-0.18***	-0.13***	0.24***	-0.16***	0.13*	0.03	0.29***	-0.20***	0.11	-0.14
Education	-0.13***	-0.05*	-0.07**	-0.09***	0.00	0.00	-0.16***	-0.19***	0.03	-0.14***	-0.05	-0.13
Income	-0.11***	-0.05	-0.11***	-0.12***	0.06*	-0.04	-0.13**	-0.16***	0.00	-0.13**	-0.18+	-0.01

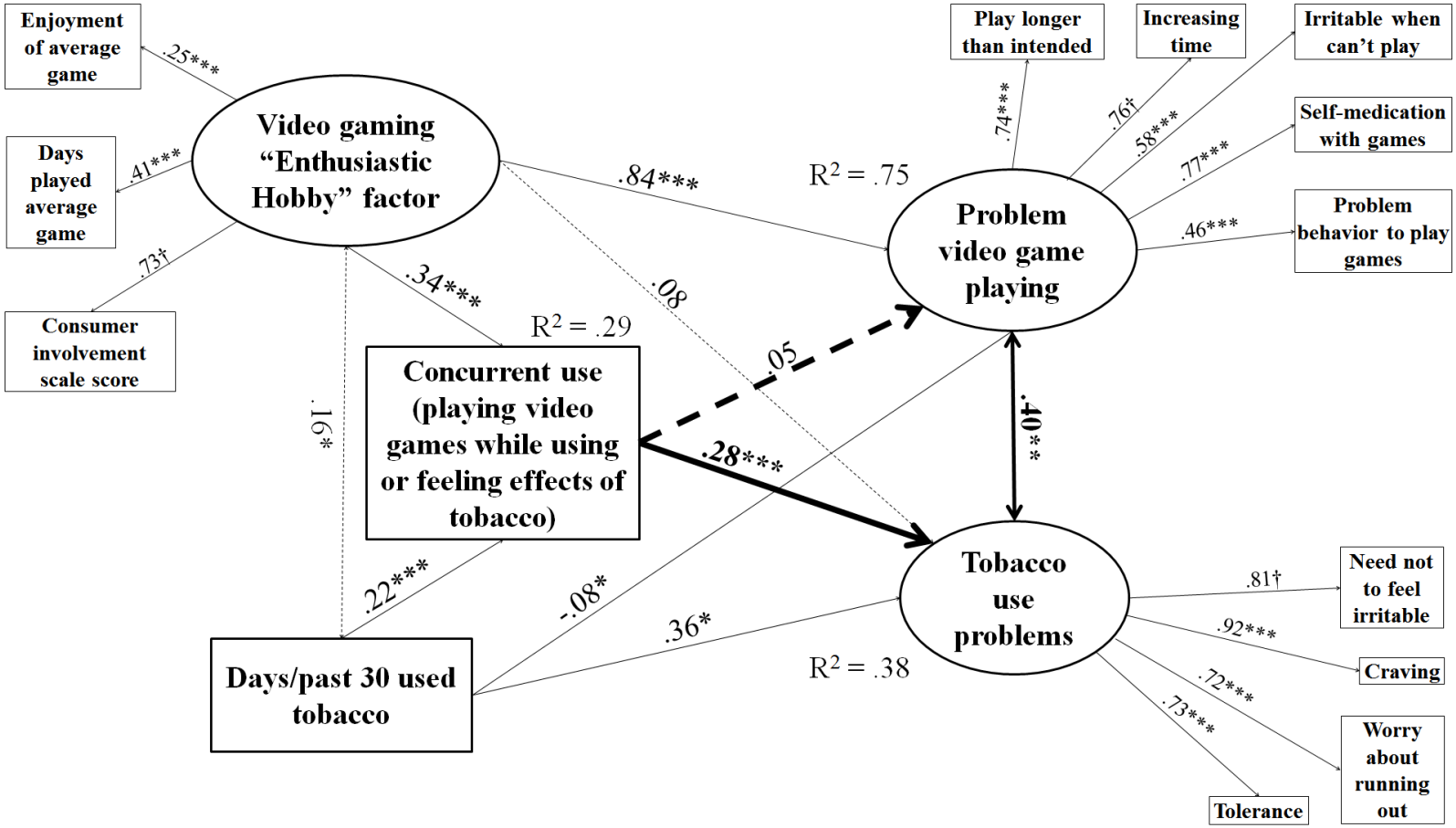
[a] Only users of the substance in question included in substance-specific analyses. + $p < 0.05$, * $p < 0.01$, ** $p < 0.001$, *** $p < 0.0001$.

Figure 1. Structural equations model for effect of concurrent use on PVP and caffeine use problems among caffeine users.



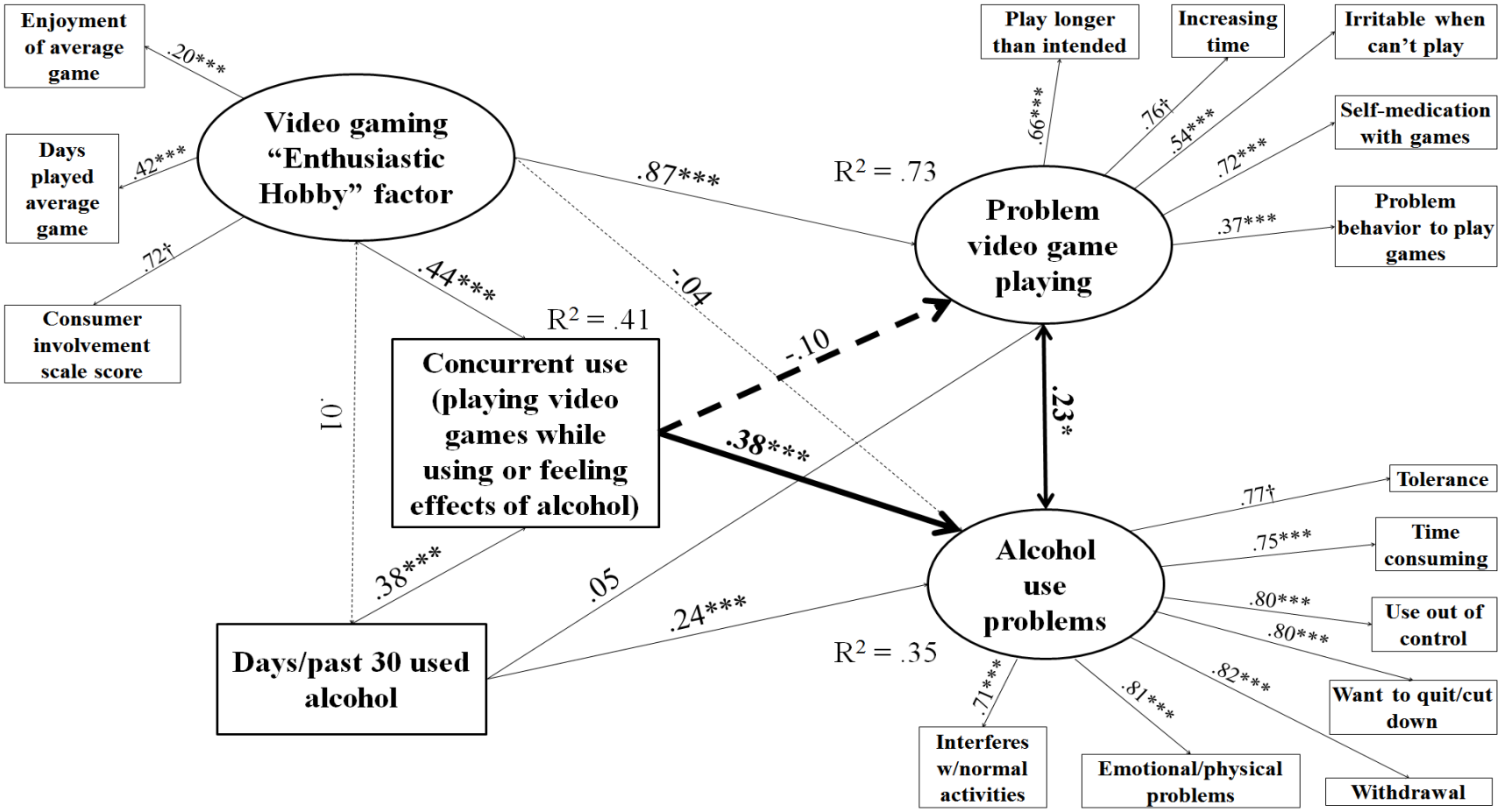
N = 1,961. Root mean square error of approximation (RMSEA) = 0.035, Comparative fit index (CFI) = 0.893. Tucker-Lewis index (TLI) = 0.850. Heavy lines indicate paths testing study hypotheses, light lines indicate control/measurement model paths. Solid lines indicate significant paths, and dashed lines indicate non-significant paths. Coefficients standardized after estimation; † indicates parameter constrained to be 1 for estimation. Model controls for age, educational level, gender, income, employment status, and race. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Figure 2. Structural equations model for effect of concurrent use on PVP and tobacco use problems among tobacco users.



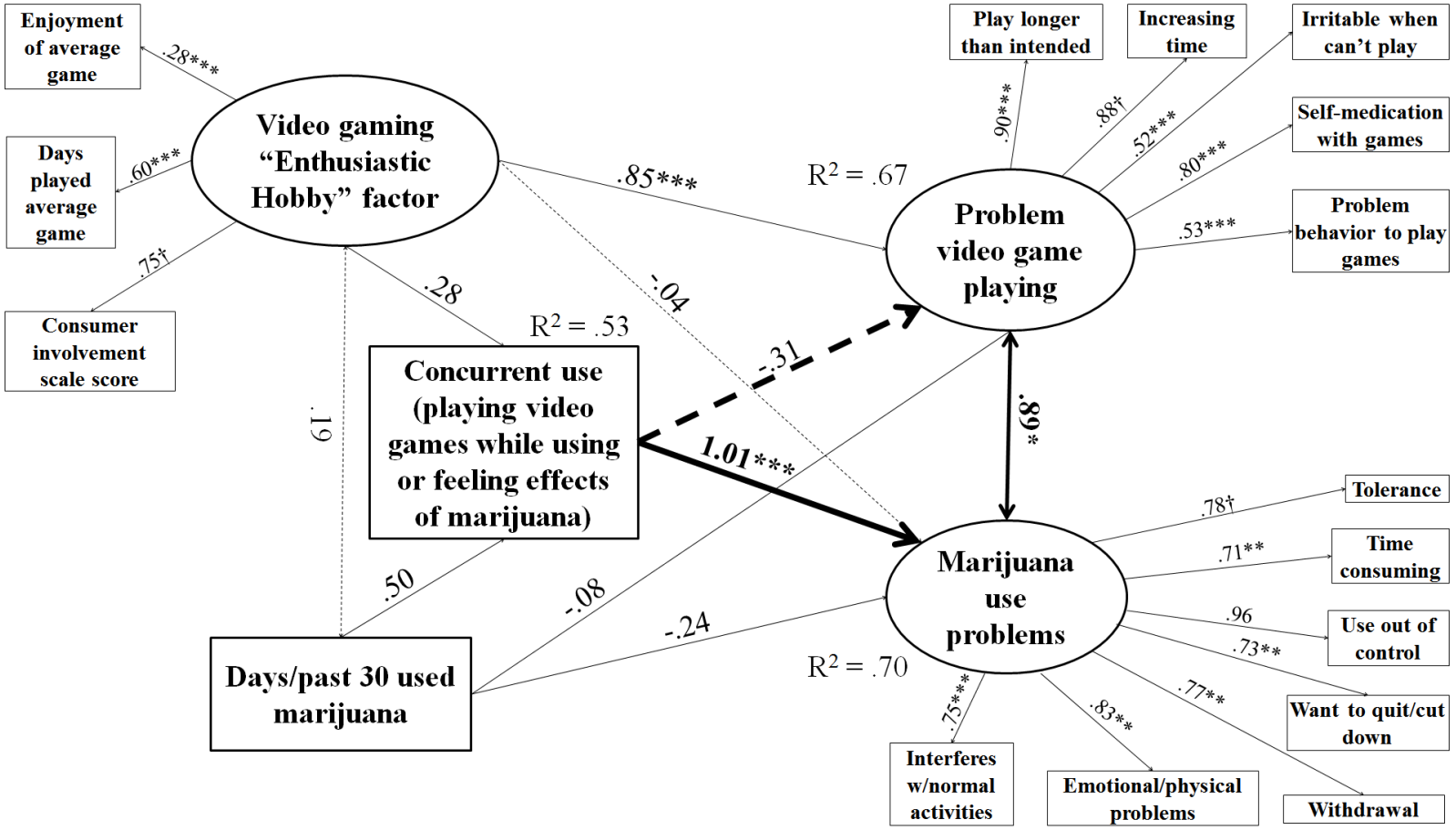
N = 683. Root mean square error of approximation (RMSEA) = 0.026, Comparative fit index (CFI) = 0.934. Tucker-Lewis index (TLI) = 0.904. Heavy lines indicate paths testing study hypotheses, light lines indicate control/measurement model paths. Solid lines indicate significant paths, and dashed lines indicate non-significant paths. Coefficients standardized after estimation; † indicates parameter constrained to be 1 for estimation. Model controls for age, educational level, gender, income, employment status, and race. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Figure 3. Structural equations model for effect of concurrent use on PVP and alcohol use problems among alcohol users.



N = 1,018. Root mean square error of approximation (RMSEA) = 0.025, Comparative fit index (CFI) = 0.936. Tucker-Lewis index (TLI) = 0.915. Heavy lines indicate paths testing study hypotheses, light lines indicate control/measurement model paths. Solid lines indicate significant paths, and dashed lines indicate non-significant paths. Coefficients standardized after estimation; † indicates parameter constrained to be 1 for estimation. Model controls for age, educational level, gender, income, employment status, and race. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Figure 4. Structural equations model for effect of concurrent use on PVP and marijuana use problems among marijuana users.



N = 133. Root mean square error of approximation (RMSEA) = 0.038, Comparative fit index (CFI) = 0.891. Tucker-Lewis index (TLI) = 0.857. Heavy lines indicate paths testing study hypotheses, light lines indicate control/measurement model paths. Solid lines indicate significant paths, and dashed lines indicate non-significant paths. Coefficients standardized after estimation; † indicates parameter constrained to be 1 for estimation. Model controls for age, educational level, gender, income, employment status, and race. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Concurrent use with all substances was prevalent. Males reported higher consumer involvement, but females report higher enjoyment, and males (within this video gamers only sample) exhibited only marginally higher PVP. Females had more frequent use of and problems with tobacco. Males were, however, more frequent concurrent users with caffeine and alcohol than females. Although Blacks, Asians, and Native Americans had higher PVP than whites, Blacks had higher enjoyment and consumer involvement but not frequency, Asians had higher frequency but not enjoyment or consumer involvement, and Native Americans had higher consumer involvement but not frequency or enjoyment. Whites, however, were the only group to exhibit clearly higher rates of concurrent use issues, and then only with caffeine. The only demographic factor consistently associated with risk for problem use patterns was non-working status, and this only held for video games, tobacco, and alcohol.

Table 2 describes bivariate correlations among continuous study variables. Frequency of game playing, enjoyment of average game, consumer involvement, and PVP were all correlated with each other. All substance use problems variables were correlated with each other except for marijuana with tobacco and PVP. The positive correlation between days played and age was not, according to follow-up analyses (not shown), because of meaningful curvilinearity—the slope of the positive relationship between age and days played was steeper for younger participants and still positive, although more shallow, for older participants. Age was, however, negatively correlated with other game playing variables, including PVP. Consistent with findings about employment in Table 1, income and education were negatively correlated with video game playing frequency, consumer involvement, PVP, tobacco use frequency, tobacco use problems, and alcohol use problems.

Figures 1–4 describe results of path analysis models for caffeine, tobacco, alcohol, and marijuana users (respectively). For all models, the path from concurrent use to substance use problems was significant, and the correlation between PVP and substance use problems also was significant. Concurrent use was not directly associated with PVP. Video gaming as an enthusiastic hobby was indirectly associated with substance use problems through two paths. The first path was via PVP, which was significant for all four substances. The second was via concurrent use, which was only significant for caffeine, tobacco, and alcohol. An effect via concurrent use with marijuana may have been hard to distinguish because of the low sample size and the high rate of concurrent use among marijuana users (see Table 1). The pattern of significant paths described here also held if a dosage variable (e.g., number of caffeinated drinks, cigarettes, or alcoholic drinks per day of alcohol use) was used instead of the substance use frequency variable.

4. Discussion

Results confirmed that, in models accounting for shared variance between substance use problems and PVP [8,10] and controlling for frequency of substance use, video gaming as an enthusiastic hobby, and demographics, concurrent use (playing video games while using or feeling the effects of substances) was uniquely associated with substance use problems. They did not, however, confirm the hypothesized direct association between concurrent use and PVP. The same pattern of results occurred for all four substances studied. The lack of a significant direct association between concurrent use and PVP makes these results not wholly congruent with previous research findings that alcohol exacerbates

problem gambling [14-19]. Rather, video gaming as an enthusiastic hobby emerged as a possible "third variable" associated with both concurrent use and PVP. Our models also distinguish the enthusiastic hobby factor as indirectly associated with substance use problems, through PVP (for all for substances) and concurrent use (for caffeine, alcohol, and tobacco). Although demographics are background variables in our models, our bivariate results for demographic variables echo earlier findings that socioeconomic stressors are just as relevant to problem video gaming behavior [64,81] as they are to substance addiction [82].

Factors contributing to this study's validity are its nationally representative sample collected by a provider whose data are frequently used in research [83] and its use of sampling weights calculated by the provider to correct for biases and authentically represent the population under study [76]. The online nature of the sample and 59% response rate to the screening instrument probably cannot be argued to be limitations in and of themselves: Online data collection has demonstrated validity in this area [84] and, while new communications technology has diminished response rates to all survey methods (phone and postal mail included), point estimates remain stable across methods and at much lower response rates than ours [85]. Another strength of this study was its sample of adults, filling a need noted in previous work for more studies on adults [24] and ensuring that our findings cannot be parsimoniously explained by youth problem behavior theory [61].

In order to meet the 10-minute median length requirement for this survey, established measures for PVP [31] and substance use [65] had to be abridged. We ensured validity of our measures, in part, through use of structural equation modeling – had measures really been invalid, our models would not have had adequate overall fit. Further confidence in our substance use problems and problem video gaming behavior measures can be drawn from their significant bivariate correlations with each other, which are consistent with other research using these concepts [8,10,11]. Another limitation of our measures was that operationalization of our key construct of concurrent use depended on a single question: "During the past 30 days, have you played video games while using [substance in question] or feeling its effects?" Some participants may have interpreted it either too strictly (as an exclusive rather than inclusive "or") or loosely (reporting on usual behavior rather than thinking specifically about the past 30 days). Although this variation in interpretation may have increased random error, it did not necessarily introduce bias. Further confidence in the validity of this measure can be drawn from the same pattern of associations involving it holding for all four substances under study. Other limitations of this study are those that it shares with every survey study, e.g., dependence on self-report data and participants' recall of events 30 days or even a year ago. Finally, the scope of the implications of these findings is limited by their basis in cross-sectional data. Although our graphics include arrows indicating directions of effects, structural equation modeling in the context of a study like this mainly offers a heuristic for understanding associations among variables. Our results are not meant to support firm conclusions about causality or how these factors influence each other over time.

Even with these limitations, these findings contribute to a growing understanding that behavioral and substance addictions co-occur [8-13] and may contribute to each other through influencing or complementing each other's actual use practices [14-18] and activating biologically mediated addictive processes [2-7]. Understanding concurrent engagement in self-reinforcing behavior and use of addictive substances may be an important consideration in addiction specificity [86], *i.e.*, variability in the development of co-occurring addictive patterns among those engaged in the prerequisite behaviors.

Clinical implications of these findings, to the extent that they can be drawn from a non-clinical population, are that clients who present with behavioral addictions or substance use problems should be screened for both and also assessed for concurrent use.

Acknowledgments

This research was supported by grant R01-DA027761, "Video Games' Role in Developing Substance Use", from the National Institute of Drug Abuse.

Conflict of Interest

None of the authors has any conflict of interest that might affect the integrity of the results.

References

1. Kelley, A.E.; Berridge, K.C. The neuroscience of natural rewards: Relevance to addictive drugs. *J. Neurosci.* **2002**, *22*, 3306-3311.
2. López-Moreno, J.A.; González-Cuevas, G.; Moreno, G.; Navarro, M. The pharmacology of the endocannabinoid system: Functional and structural interactions with other neurotransmitter systems and their repercussions in behavioral addiction. *Addict. Biol.* **2008**, *13*, 160-187.
3. Di Chiara, G.; Bassareo, V. Reward system and addiction: What dopamine does and doesn't do. *Curr. Opin. Pharmacol.* **2007**, *7*, 69-76.
4. Volkow, N.D.; Fowler, J.S.; Wang, G.J.; Swanson, J.M. Dopamine in drug abuse and addiction: Results from imaging studies and treatment implications. *Mol. Psychiatr.* **2004**, *9*, 557-569.
5. Kalivas, P.W.; O'Brien, C. Drug addiction as a pathology of staged neuroplasticity. *Neuropsychopharmacology* **2007**, *33*, 166-180.
6. Robbins, T.W.; Cador, M.; Taylor, J.R.; Everitt, B.J. Limbic-striatal interactions in reward-related processes. *Neurosci. Biobehav. Rev.* **1989**, *13*, 155-162.
7. De Lecea, L.; Jones, B.E.; Boutrel, B.; Borgland, S.L.; Nishino, S.; Bubser, M.; DiLeone, R. Addiction and arousal: Alternative roles of hypothalamic peptides. *J. Neurosci.* **2006**, *26*, 10372-10375.
8. Rozin, P.; Stoess, C. Is there a general tendency to become addicted? *Addict. Behav.* **1993**, *18*, 81-87.
9. El-Guebaly, N.; Patten, S.B.; Currie, S.; Williams, J.V.A.; Beck, C.A.; Maxwell, C.J.; Wang, J.L. Epidemiological associations between gambling behavior, substance use & mood and anxiety disorders. *J. Gamb. Stud.* **2006**, *22*, 275-287.
10. Greenberg, J.L.; Lewis, S.E.; Dodd, D.K. Overlapping addictions and self-esteem among college men and women. *Addict. Behav.* **1999**, *24*, 565-571.
11. Porter, G.; Starcevic, V.; Berle, D.; Fenech, P. Recognizing problem video game use. *Austr. New Zeal. J. Psychiatr.* **2010**, *44*, 120-128.
12. Wood, R.T.A.; Gupta, R.; Derevensky, J.L.; Griffiths, M.D. Video game playing and gambling in adolescents: Common risk factors. *J. Child Adol. Sub. Abuse* **2004**, *14*, 77-100.

13. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority? *Eval. Health Profess.* **2011**, *34*, 3-56.
14. Baron, E.; Dickerson, M. Alcohol consumption and self-control of gambling behaviour. *J. Gamb. Stud.* **1999**, *15*, 3-15.
15. O'Connor, J.; Dickerson, M. Definition and measurement of chasing in off-course betting and gaming machine play. *J. Gamb. Stud.* **2003**, *19*, 359-386.
16. Cronce, J.M.; Corbin, W.R. Effects of alcohol and initial gambling outcomes on within-session gambling behavior. *Experimen. Clin. Psychopharmacol.* **2010**, *18*, 145-157.
17. Ellery, M.; Stewart, S.H.; Loba, P. Alcohol's effects on video lottery terminal (vlt) play among probable pathological and non-pathological gamblers. *J. Gamb. Stud.* **2005**, *21*, 299-324.
18. Kyngdon, A.; Dickerson, M. An experimental study of the effect of prior alcohol consumption on a simulated gambling activity. *Addiction* **1999**, *94*, 697-707.
19. Dickerson, M.; Baron, E. Contemporary issues and future directions for research into pathological gambling. *Addiction* **2000**, *95*, 1145-1159.
20. King, D.; Delfabbro, P.; Griffiths, M. The convergence of gambling and digital media: Implications for gambling in young people. *J. Gamb. Stud.* **2010**, *26*, 175-187.
21. King, D.; Delfabbro, P.; Griffiths, M. Video game structural characteristics: A new psychological taxonomy. *Int. J. Mental Health Addict.* **2010**, *8*, 90-106.
22. Kuss, D.; Griffiths, M. Internet gaming addiction: A systematic review of empirical research. *Int. J. Mental Health Addict.* **2011**, Advance online publication doi: 10.1007/s11469-011-9318-5.
23. Wood, R.T.A. Problems with the concept of video game "addiction": Some case study examples. *Int. J. Mental Health Addict.* **2008**, *6*, 169-178.
24. Petry, N.M. Commentary on Van Rooij *et al*. (2011): Gaming addiction—A psychiatric disorder or not? *Addiction* **2011**, *106*, 213-214.
25. American Psychiatric Association. *Substance Use and Addictive Disorders*; American Psychiatric Association: Arlington, VA, USA, 2010. Available online: http://www.dsm5.org/ProposedRevision/Pages/SubstanceUseandAddictiveDisorders.aspx (accessed on 13 July 2011).
26. Young, K.S. Understanding online gaming addiction and treatment issues for adolescents. *Am. J. Famil. Therap.* **2009**, *37*, 355-372.
27. Griffiths, M.; Meredith, A. Videogame addiction and its treatment. *J. Contemp. Psychotherap.* **2009**, *39*, 247-253.
28. Khan, M.K.; Kantof, E.P. *Emotional and Behavioral Effects, Including Addictive Potential, of Video Games*; American Medical Association: Chicago, IL, USA, 2007. Available online: www.ama-assn.org/ama1/pub/upload/mm/467/csaph12a07.doc (accessed on 19 January 2011).
29. Griffiths, M.D. Amusement machine playing in childhood and adolescence: A comparative analysis of video games and fruit machines. *J. Adol.* **1991**, *14*, 53-73.
30. Fisher, S. Identifying video game addiction in children and adolescents. *Addict. Behav.* **1994**, *19*, 545-553.
31. Tejeiro Salguero, R.A.; Bersabé Morán, R.M. Measuring problem video game playing in adolescents. *Addiction* **2002**, *97*, 1601-1606.
32. Grüsser, S.M.; Thalemann, R.; Griffiths, M.D. Excessive computer game playing: Evidence for addiction and aggression? *Cyberpsychol. Behav.* **2007**, *10*, 290-292.

33. Gentile, D.A.; Choo, H.; Liau, A.; Sim, T.; Li, D.; Fung, D.; Khoo, A. Pathological video game use among youths: A two-year longitudinal study. *Pediatrics* **2011**, doi: 10.1542/peds.2010-1353.
34. Gentile, D.A. Pathological video-game use among youth ages 8 to 18: A national study. *Psychol. Sci.* **2009**, *20*, 594-602.
35. Desai, R.A.; Krishnan-Sarin, S.; Cavallo, D.; Potenza, M.N. Video-gaming among high school students: Health correlates, gender differences, and problematic gaming. *Pediatrics* **2010**, doi: 10.1542/peds.2009-2706.
36. Rehbein, F.; Psych, G.; Kleimann, M.; Mediasci, G.; Moßle, T. Prevalence and risk factors of video game dependency in adolescence: Results of a german nationwide survey. *Cyberpsychol. Behav. Soc. Netw.* **2010**, *13*, 269-277.
37. Thalemann, R.; Wölfling, K.; Grüsser, S.M. Specific cue reactivity on computer game-related cues in excessive gamers. *Behav. Neurosci.* **2007**, *121*, 614-618.
38. Koepp, M.J.; Gunn, R.N.; Lawrence, A.D.; Cunningham, V.J.; Dagher, A.; Jones, T.; Brooks, D.J.; Bench, C.J.; Grasby, P.M. Evidence for striatal dopamine release during a video game. *Nature* **1998**, *393*, 266-267.
39. Wood, R.T.A.; Griffiths, M.D.; Chappell, D.; Davies, M.N.O. The structural characteristics of video games: A psycho-structural analysis. *Cyberpsychol. Behav.* **2004**, *7*, 1-10.
40. Hill, J. *Ethical Dilemmas: Exploitative Multiplayer Worlds Don't Deserve to be Called Art*; The Sydney Morning Herald: Sydney, Australia, 2007.
41. Han, D.H.; Hwang, J.W.; Renshaw, P.F. Bupropion sustained release treatment decreases craving for video games and cue-induced brain activity in patients with internet video game addiction. *Experimen. Clin. Psychopharmacol.* **2010**, *18*, 297-304.
42. Han, D.H.; Lee, Y.S.; Na, C.; Ahn, J.Y.; Chung, U.S.; Daniels, M.A.; Haws, C.A.; Renshaw, P.F. The effect of methylphenidate on internet video game play in children with attention-deficit/hyperactivity disorder. *Comprehen. Psychiatr.* **2009**, *50*, 251-256.
43. Swing, E.L.; Gentile, D.A.; Anderson, C.A.; Walsh, D.A. Television and video game exposure and the development of attention problems. *Pediatrics* **2010**, *126*, 214-221.
44. Rowan, C. Unplug—Don't drug: A critical look at the influence of technology on child behavior with an alternative way of responding other than evaluation and drugging. *Eth. Human Psychol. Psychiatr. Int. J. Crit. Inquir.* **2010**, *12*, 60-68.
45. Grant, B.F.; Stinson, F.S.; Dawson, D.A.; Chou, S.P.; Dufour, M.C.; Compton, W.; Pickering, R.P.; Kaplan, K. Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the national epidemiologic survey on alcohol and related conditions. *Arch. Gen. Psychiatr.* **2004**, *61*, 807-816.
46. Merikangas, K.R.; Mehta, R.L.; Molnar, B.E.; Walters, E.E.; Swendsen, J.D.; Aguilar-Gaziola, S.; Bijl, R.; Borges, G.; Caraveo-Anduaga, J.J.; Dewit, D.J.; *et al*. Comorbidity of substance use disorders with mood and anxiety disorders: Results of the international consortium in psychiatric epidemiology. *Addict. Behav.* **1998**, *23*, 893-907.
47. Dawe, S.; Loxton, N.J. The role of impulsivity in the development of substance use and eating disorders. *Neurosci. Biobehav. Rev.* **2004**, *28*, 343-351.
48. Bioulac, S.; Arfi, L.; Bouvard, M.P. Attention deficit/hyperactivity disorder and video games: A comparative study of hyperactive and control children. *Europ. Psychiatr.* **2008**, *23*, 134-141.

49. Chan, P.A.; Rabinowitz, T. A cross-sectional analysis of video games and attention deficit hyperactivity disorder symptoms in adolescents. *Ann. Gener. Psychiatr.* **2006**, *5*, 16:1-16:10.
50. McClernon, F.J.; Fuemmeler, B.F.; Kollins, S.H.; Kail, M.E.; Ashley-Koch, A.E. Interactions between genotype and retrospective adhd symptoms predict lifetime smoking risk in a sample of young adults. *Nicot. Tob. Res.* **2008**, *10*, 117-127.
51. Ohlmeier, M.D.; Peters, K.; Wildt, B.T.T.; Zedler, M.; Ziegenbein, M.; Wiese, B.; Emrich, H.M.; Schneider, U. Comorbidity of alcohol and substance dependence with attention-deficit/hyperactivity disorder (adhd). *Alcohol Alcohol.* **2008**, *43*, 300-304.
52. Walker, L.R.; Abraham, A.A.; Tercyak, K.P. Adolescent caffeine use, adhd, and cigarette smoking. *Child. Health Care* **2010**, *39*, 73-90.
53. Becker, S.J.; Curry, J.F. Interactive effect of substance abuse and depression on adolescent social competence. *J. Clin. Child Adol. Psychol.* **2007**, *36*, 469-475.
54. Lemmens, J.S.; Valkenburg, P.M.; Peter, J. Psychosocial causes and consequences of pathological gaming. *Comput. Human Behav.* **2011**, *27*, 144-152.
55. Anand, V. A study of time management: The correlation between video game usage and academic performance markers. *Cyberpsychol. Behav.* **2007**, *10*, 552-559.
56. Cox, R.G.; Zhang, L.; Johnson, W.D.; Bender, D.R. Academic performance and substance use: Findings from a state survey of public high school students. *J. School Health* **2007**, *77*, 109-115.
57. Gentile, D.A.; Lynch, P.J.; Linder, J.R.; Walsh, D.A. The effects of violent video game habits on adolescent hostility, aggressive behaviors, and school performance. *J. Adol.* **2004**, *27*, 5-22.
58. Madden, D.; Brueckman, J.; Littlejohn, K.V. *A Contrast of Amount and Type of Activity in Elementary School Years between Academically Successful and Unsuccessful Youth*; Education Resources Information Center: Washington, DC, USA, 1997. Available online: http://eric.ed.gov/PDFS/ED411067.pdf (accessed on 17 October 2011).
59. Anderson, C.A.; Gentile, D.A.; Buckley, K.E. *Violent Video Game Effects on Children and Adolescents: Theory, Research, and Public Policy*; Oxford University Press: Oxford; NY, USA, 2007; p.190.
60. Moss, H.B.; Tarter, R.E. Substance abuse, aggression, and violence: What are the connections? *Am. J. Addict.* **1993**, *2*, 149-160.
61. Duncan, S.C.; Duncan, T.E.; Strycker, L.A. Risk and protective factors influencing adolescent problem behavior: A multivariate latent growth curve analysis. *Ann. Behav. Med.* **2000**, *22*, 103-109.
62. Chiu, S.I.; Lee, J.-Z.; Huang, D.-H. Video game addiction in children and teenagers in Taiwan. *Cyberpsychol. Behav.* **2004**, *7*, 571-581.
63. Griffiths, M.D.; Hunt, N. Dependence on computer games by adolescents. *Psychol. Rep.* **1998**, *82*, 475-480.
64. Roberts, D.F.; Foehr, U.G. *Kids and Media in America*; Cambridge University Press: Cambridge, UK, 2004; p. 380.
65. Substance Abuse and Mental Health Statistics (SAMHSA). *Methodology Reports for the National Household Survey on Drug Abuse & the National Survey on Drug Use & Health*; SAMHSA: Rockville, MD, USA, 2009. Available online: http://oas.samhsa.gov/nsduh/methods.cfm (accessed on 25 November 2010).

66. CBS Interactive Inc. *Video Game Cheats, Reviews, Faqs, Message Boards, and More—Gamefaqs*; CBS Interactive Inc.: San Francisco, CA, USA, 2011. Available online: http://www.gamefaqs.com/ (accessed on 15 February 2011).
67. Charlton, J.P.; Danforth, I.D.W. Validating the distinction between computer addiction and engagement: Online game playing and personality. *Behav. Inform. Technol.* **2010**, *29*, 601-613.
68. Skoric, M.M.; Teo, L.L.C.; Neo, R.L. Children and video games: Addiction, engagement, and scholastic achievement. *Cyberpsychol. Behav.* **2009**, *12*, 567-572.
69. Griffiths, M. The role of context in online gaming excess and addiction: Some case study evidence. *Int. J. Mental Health Addict.* **2010**, *8*, 119-125.
70. Griffiths, M. A components' model of addiction within a biopsychosocial framework. *J. Sub. Use* **2005**, *10*, 191-197.
71. Laurent, G.; Kapferer, J.-N. Measuring consumer involvement profiles. *J. Market. Res.* **1985**, *22*, 41-53.
72. Wiley, C.G.E.; Shaw, S.M.; Havitz, M.E. Men's and women's involvement in sports: An examination of the gendered aspects of leisure involvement. *Leisure Sci.* **2000**, *22*, 19-31.
73. Gursoy, D.; Gavcar, E. International leisure tourists' involvement profile. *Ann. Tour. Res.* **2003**, *30*, 906-926.
74. Wood, R.T.A. A response to Blaszczynski, Griffiths and Turners' comments on the paper "problems with the concept of video game 'addiction': Some case study examples" (this issue). *Int. J. Mental Health Addict.* **2008**, *6*, 191-193.
75. Blaszczynski, A. Commentary: A response to "problems with the concept of video game 'addiction': Some case study examples". *Int. J. Mental Health Addict.* **2008**, *6*, 179-181.
76. DiSogra, C. *Overview of Knowledgepanel® Statistical Weighting Protocol*; Knowledge Networks: New York, NY, USA, 2009. Avaiable online: http://www.knowledgenetworks.com/ganp/docs/kn-weighting-synopsis.pdf (accessed on 25 April 2011).
77. McDonald, R.P.; Ho, M.-H.R. Principles and practice in reporting structural equation analyses. *Psychol. Methods* **2002**, *7*, 64-82.
78. Hooper, D.; Coughlan, J.; Mullen, M.R. Structural equation modelling: Guidelines for determining model fit. *Electron. J. Busin. Res. Methods* **2008**, *6*, 53-60.
79. Diamantopoulos, A.; Winklhofer, H. Index construction with formative indicators: An alternative to scale development. *J. Market. Res.* **2001**, *38*, 269-277.
80. Kenny, D.A. *Measuring Model Fit*; 2011. Available online: http://www.davidakenny.net/cm/fit.htm (accessed on 12 September 2011).
81. Brady, S.S.; Matthews, K.A. Effects of media violence on health-related outcomes among young men. *Arch. Pediatr. Adol. Med.* **2006**, *160*, 341-347.
82. Sinha, R. Chronic Stress, Drug Use, and Vulnerability to Addiction. In *Annals of the New York Academy of Science*; Uhl, G.R., Ed.; John Wiley&Sons, Inc.: Hoboken, NJ, USA, 2008; Volume 1141, pp. 105-130.
83. Knowledge Networks. *Knowledge Networks Bibliography*; Knowledge Networks: New York, NY, USA, 2011. Available online: http://www.knowledgenetworks.com/ganp/docs/KN-Bibliography.pdf (accessed on 17 September 2011).

84. Griffiths, M. The use of online methodologies in data collection for gambling and gaming addictions. *Int. J. Mental Health Addict.* **2010**, *8*, 8-20.
85. Zukin, C. *Game Change: The Challenge of Finding and Interviewing a Random Sample in 2010*; Knowledge Networks Seminars, New York, NY, USA, 28 October 2010.
86. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.

Reprinted from *Int. J. Environ. Res. Public Health*. Cite as: Sussman, S.; Sussman, A.N. Considering the Definition of Addiction. *Int. J. Environ. Res. Public Health* **2011**, *8*, 4025-4038; doi:10.3390/ijerph8104025.

OPEN ACCESS

International Journal of
Environmental Research and Public Health
ISSN 1660-4601
www.mdpi.com/journal/ijerph

Editorial

Considering the Definition of Addiction

Steve Sussman [1,*] and Alan N. Sussman [2]

[1] Departments of Preventive Medicine and Psychology, University of Southern California, Soto Street Building, Room 302, 2001 N. Soto Street, Los Angeles, California 90032, USA
[2] 2329 Winthrop Avenue, Apt. 60, Roanoke, Virgina 24015, USA; E-Mail: alansussman67@yahoo.com

* Author to whom correspondence should be addressed; E-Mail: ssussma@usc.edu; Tel.: +1-323-442-8220; Fax: +1-323-442-8201.

Received: 21 September 2011 / Accepted: 17 October 2011 / Published: 20 October 2011

Abstract: The definition of addiction is explored. Elements of addiction derived from a literature search that uncovered 52 studies include: (a) engagement in the behavior to achieve appetitive effects, (b) preoccupation with the behavior, (c) temporary satiation, (d) loss of control, and (e) suffering negative consequences. Differences from compulsions are suggested. While there is some debate on what is intended by the elements of addictive behavior, we conclude that these five constituents provide a reasonable understanding of what is intended by the concept. Conceptual challenges for future research are mentioned.

Keywords: definition; addiction

1. Introduction

Obtaining a consensual and testable definition of a concept is useful to be able to make inferences regarding how the concept is related to other concepts, and subsequently develop useful applications (e.g., policy, services offered [1]). Unfortunately "addiction" is a concept that has been the subject of much debate [2-9]. Delineation of its definitional elements may be a step toward eventually achieving consensus and operationalization of this construct. At its origin, "addiction" simply referred to "giving over" or being "highly devoted" to a person or activity [10], or engaging in a behavior habitually [11], which could have positive or negative implications. Over the last 400 years, some

statements made about addiction began to frame it as involving strong, overpowering urges and, over the last 200 years this word has become considered more and more disease-like in connotation [12]. Many conceptualizations of the addictions pertain to imbalance of the central nervous system in some way, and these conceptualizations date back to the end of the 1700s [13]. It is more recently used as a concept having neurobiological underpinnings [14-19]. Descriptions of addiction that map onto measurable criteria could serve as phenotypes that might maximally explain gene-environment interactions in this arena [20], and best serve prevention and control efforts.

Literature Search

As a start to grappling with views on definitions of addiction, we engaged in an electronic search of Google Scholar, pairing the term "addiction" with "definition" (on April 7, 2011). The first 500 of 172,000 web pages was examined, revealing 40 relevant citations. We also engaged in the same search on OvidMEDLINE (1948-June Week 4, 2011; on July 3, 2011), revealing six citations but no new ones. Finally, we engaged in a search of PsycINFO (July 23, 2011). Simply pairing the terms revealed 2,994 peer reviewed articles. However, only two of the first 100 provided relevant information (*i.e.*, pertaining to definitions of addiction). Thus, a second search using the phrase "definition of addiction" was conducted, revealing 47 citations total and 33 peer-reviewed articles. Of these, two citations were unique and relevant to grappling with the definition of addiction. All articles found through this search, along with others obtained through previous work and word of mouth, are included in this review (total = 52 published sources that discuss the definition of addiction).

To our knowledge, this is the first systematic electronic literature review of the concept of addiction. Based on this literature review, it appears that addiction is conceptualized as being composed of multiple elements [6,21]. We highlight explicitly stated elements common across at least 20% of the 52 papers that address the definition of the addictions. However, a quantitative analysis was not provided here that examines number and type of element referred to as a function of study characteristics. Rather we took a qualitative approach that summarizes the most commonly agreed on elements of addiction and discusses philosophical concerns regarding necessary and sufficient conditions for a psychological state or pattern of behavior to *be* an addiction. It is true that over 75% (ns > 39) of the papers in the set do mention all of the criteria except for satiation as being elements of addiction. Only 20% (n = 10) of the 52 studies uncovered mentioned satiation as a definitional element of addiction; however, we view it as an essential element to the concept and one which has obvious treatment implications. While we felt that a quantitative analysis would not be of much incremental benefit, others may disagree. These five elements, which have been most popularly suggested as being constituents of addiction, are discussed next.

2. Results

2.1. Feeling Different

In most cases, an addiction does not develop overnight. In general, when contemplating addiction, one often thinks of it in terms of a process. Upon the initialization of the "addictive process" [4,22,23], one pursues some course of action for appetitive effects or motives (e.g., pain reduction, affect

enhancement, arousal manipulation, or fantasy). Different addictive behaviors have been empirically clustered as serving hedonistic (e.g., drug use, sex, gambling) or nurturant (e.g. compulsive helping, work addiction, shopping addiction, love, exercise) motives [24,25]. However, other or additional motives are plausible (e.g., to achieve fantasy or oblivion [3]), and all addictions may share in common a function to shift subjective experience of self [26].

The addiction process unfolds for some individuals but not others, and may reflect individual differences prior to engaging in the addictive behavior or as the individual continues to engage in the addictive behavior (*i.e.*, individuals may vary along a dimension of "addiction proneness"). Anecdotally, many self-described addicts have reported feeling "different" from others long before developing readily identifiable addictions. This includes feeling relatively uncomfortable, lonely, restless, or incomplete [23]. Once a behavior is tried that decreases or eliminates the baseline sense of discomfort a process begins to unfold. It is possible that 50% of the variance of addictive behavior is attributed to a genetic cause of this subjective sense of discomfort [18,27]. The extent to which there exist persons in-born for addiction remains a subject of debate [26].

Alternatively, many persons report not feeling different prior to engaging in a problematic addictive behavior. Among these individuals, a behavior may be tried that is perceived as highly valued or enjoyable, possibly with effects that occur rather rapidly, that one desires to repeat [28]. In this instance, a process begins to unfold inducing a contrast between an enhanced or potentially addictive behavior-induced state of arousal, affect or cognition, and a baseline state of arousal, affect, or cognition. The initial reaction to the potentially addictive behavior may be experienced as more positive than with other persons (among those relatively prone [8]). That is, addictive "appetites" may fall along a continuum [2], and those persons at one extreme may find certain behaviors particularly enticing. Involvement in extreme levels (frequency or valence) of these behaviors, which tend to be subject to social or other consequential restraints, may identify addictive levels of behaviors [12].

2.2. Preoccupation with the Behavior

A second aspect of addiction considers excessive thoughts about and desire to perform a behavior, excessive time spent to plan and engage in the behavior, and possibly recover from its effects (e.g., from "hangovers"), and less time spent on other activities [29], despite potentially diminishing appetitive effects [30,31]. That is, the addictive behavior "spills over" into several dimensions of one's daily life. This may be labeled more generally as "preoccupation." For example, a two-pack-a-day cigarette smoker may report often thinking about smoking cigarettes (particularly when restricted from smoking, or at certain points during the day when one is most likely to smoke), or thinking about anti-smoking control efforts, may invest a great deal of money to continue to purchase cigarettes, may have a cigarette in hand 280 minutes per day (approximately one-third of the waking day; a behavioral aspect of preoccupation), and may report experiencing discomfort upon cessation of smoking for more than a couple of hours.

Interestingly, it is not known to what extent addictive desires operate on neurobiological processes differently from regular desires [2,3]. However, addictive behavior-induced repetitive firing of certain brain systems (e.g., mesolimbic dopamine) does result in brain adaptations (e.g., activation of

glutamatergic system; decrease in production of mesolimbic dopamine), suggestive of a "hijacking" of the brain due to engagement in any of a variety of substance or process addictive behaviors [32-34].

Tolerance and withdrawal are the two hallmark criteria of physiological addiction, and, arguably, may also be considered as aspects of a more general concept of preoccupation (or as features that contribute to preoccupation). Tolerance refers to the need to engage in the behavior at a relatively greater level than in the past to achieve previous levels of appetitive effects. As tolerance increases, one likely spends more time locating and engaging in an addiction. Thus, tolerance may indicate increasing preoccupation. Withdrawal refers to physiological or acquired discomfort experienced upon abrupt termination of an addictive behavior. If withdrawal symptoms exist, and worsen, one is likely to be spending more and more time recovering from the after-effects of the addiction, and focused in thought and action on how to cope (e.g., by using again). That is, one is more preoccupied with the addiction when one is spending more time locating, engaging, and recovering from that behavior, and this may reflect processes of tolerance and withdrawal [35-37].

Possibly, related to tolerance or withdrawal, is the notion of craving. Craving or urges to engage in a target addictive behavior has been a hallmark defining feature of the addictions for a long time [13,38]. Craving is not necessarily the same thing as physiological withdrawal and may, in fact, be more central to the concept of addictions [34]. For example, several highly addictive drugs (e.g., cocaine) are thought to not have strong physiological withdrawal symptoms (e.g., such as "the shakes" as with alcohol); rather, they are identified by drug seeking behavior while in the midst of accumulative negative consequences [34]. Some addicts who are new in recovery may even maintain a subjective sense of fear that catastrophic events will occur if they continue to refrain from their addictions [23]. Craving has been proposed as a diagnostic feature of the addictions to be added to the DSM-V [39]. While there is some ambiguity regarding the definition of craving (e.g., this concept may overlap with implicit expectancy cognitive processes [19]), at least *prima facie*, the definition appears to refer to an "intense desire" to engage in a specific act [3,40], that reoccurs and about which one often conforms [40].

2.3. Temporary Satiation

A third element of the concept of addiction is "satiation". After acute engagement in an addictive behavior, some period of time may occur in which urges are not operative, addiction craving is "shut down", only to return soon [3,12,38]. This satiation period is not well studied or considered. Some thoughts regarding this period pertain to a sense of distraction from life problems or feeling temporarily self-sufficient or nurtured [41,42]. If these feelings continued, arguably, one may speculate that the individual would have achieved a resolution of the subjective sense of discomfort (or "disbalance") that precedes engagement in the addictive behavior [43].

Satiation may be examined from the perspective of the Incentive-Motivational Model [12,44,45], which examines in part how an addictive behavior may elicit satiation of emotional expectations through its incentive value (e.g., feeling "incentivized"). From this perspective, non-addictive alternatives over time may lose incentive value. That is, even though the addiction may not achieve satiety as well as it used to, its relative incentive value compared to non-addictive alternatives may increase [45]. That is, while there may be some discomfort in trying to achieve satiation, alternatives

may provide even less of a chance of achieving a sense of satiation. Therefore, an iterative pattern continues involving a period of participation in an addictive behavior followed by a period of satiation.

One other notion possibly pertaining to satiation (as well as to loss of control, discussed below) is that of psychological reversals [46,47]. This notion is the idea that people may fluctuate sharply between two or more experiential states (e.g., shifting back and forth between experiencing a sensation seeking state, to a goal-oriented/calm state). Biological needs, valence *versus* time delay of addictive behavior-related rewards, and a feeling a sense of frustration *versus* satiation, can shift one from one state to another [46,48]. That is, when frustrated the individual may seek out an addictive behavior, and when satiated the individual may temporarily avoid such temptations. The shift that may occur (e.g., Dr. Jekyl to Mr. Hyde) appears to reflect a lack of control, though behavior may appear well-controlled within each mood state.

Arguably, there may be instances in which a person suffering from an addiction reports no longer being able to achieve satiation. If so, some researchers might suggest that satiation should not be considered a defining element of addiction. It might rather be considered as a construct which interacts with addiction [49]. Alternatively, it is feasible that satiation almost always is achieved even if for brief periods, possibly directly following the first moments of onset of an addictive behavior, and when not satiated, satiation is still sought.

2.4. Loss of Control

Among the defining elements of addiction, loss of control has a rather long history [12,13]. One may report desiring to stop an addictive behavior but, even so, not having the ability to precisely predict when a bout with the behavior will be initiated, how it will be manifested, or when it will stop. That is, the addictive behavior may become increasingly more automatic [4,12,15,19,50-54]. Difficulty in refraining from an addictive behavior despite attempting to do so may be central to a loss of control aspect of addiction (see Heather [19] on "akrasia"). Many persons claim to be struggling with an addiction; feeling compelled, sensing incomplete control; and it is observed that they may disregard even basic self-care, suggestive of a loss of will [8].

Incomplete memory access appears to be a common feature of addictions. According to Campbell [29], the "cognitive impairment" associated with an addiction emerges only when a specific addiction associated with harmful consequences produces a simultaneous positive emotional response. This attentional narrowing minimizes or negates the memory of the negative effects or consequences of previous addictive behavior experiences (or access to aversive memory). Phenomenologically, due to these memory effects, recovering addicts with some "sober time" may look back at their using days as being disordered, illogical, fragmented, destructive, and nonsensical [41].

Impulsiveness is another descriptor that has been used to indicate addiction-related loss of control [55]. This aspect of the addictive process might be identified as including spontaneous urges to engage in the addictive behavior about which executive inhibitory processes fail to operate due to actions of addiction-related reinforcers on separate memory systems (implicit *versus* declarative systems [56]). Possibly, the existence of separate memory systems may account for both "incomplete memory" and "impulsiveness" descriptions of the loss of control aspect of addiction [51]. That is, the implicit system may facilitate attentional narrowing and impulsive behavior associated with previously

reinforcing addiction-related events that are strongly embedded in memory, whereas the declarative system may fail to be operative in "trigger" (high risk) situations, to inhibit a relatively automatic (self-destructive) chain of behavior [51,56].

On the other hand, executive planning (declarative cognitive) processes are involved in the addiction seeking process (e.g., one may need to be innovative to acquire their drug of choice [2,43]), many people appear to mature out of addictions [2], and many times if sufficient justification is provided a person may stop engagement in an addictive behavior [2]. Such examples could suggest that persons suffering from addictions simply are making choices for pleasure in accordance with their lifestyle preferences (and they may have very strong desires for pleasure), contrary to normative expectations [2]. That is, people may have very strong, regular, appetitive desires that outweigh other alternatives and cause negative consequences; but are engaged in not due to an obvious lack of behavioral control *per se*. The relative emphasis placed on appetitive *versus* loss of control aspects of addiction apparently vary as a function of age; adults tend to view the loss of control aspect as more important than do adolescents [36].

2.5. Negative Consequences

A fifth defining element of the concept of addiction is the existence of negative consequences. In general, at some point, negative consequences tend to ensue due to engaging in an addictive behavior (e.g., physical discomfort, social disapproval, financial loss, or decreased self-esteem [57]). Continuing to engage in the addictive behavior after suffering numerous negative consequences often has been a criterion of dependence on the addictive behavior [4,29]. Stopping the behavior becomes difficult for several reasons, including influence of the cognitive salience of immediate gratification resulting from the addictive behavior (*i.e.*, satiation) relative to its delayed adverse effects. The individual also may fear having to cope with day-to-day perceived stress and other life experiences upon cessation (possibly due to accumulation of addiction-related consequences, or having to endure "raw" emotional experiences without concurrent self-medication, having failed to learn to cope without use of the addiction [23]), as well as suffer withdrawal-related phenomena [22,58,59]. Thus, the addiction persists, incurring negative effects while also providing maintenance functions.

Negative consequences may vary across contexts. For example, arrests for drinking and driving may not be well-enforced in some countries (e.g., some rural areas in Southeast Asia), or may be enforced very strictly in other countries (e.g., Sweden). Thus, the legal consequences related to drinking alcohol may vary across contexts. Physical consequences may vary likewise (e.g., there may be fewer injuries and deaths related to drinking and driving in locations where drinking-diving laws are well-enforced [27]). Also, the social consequences of alcohol use and other addictions vary across history [11] and cultures [60], for example, due to differential tolerance of public display of drunken (or high) behavior. Possibly, role consequences (e.g., difficulty fulfilling one's role as parent, spouse, or coworker) may be a relatively invariant aspect of negative consequences that operates across different addictions [27]. That is, if a person is unable to perform their societally-defined roles in the world (which sets the parameters of their overall contribution to self and others) due to their problematic behavior, they are maximally likely to be labeled as "addicted".

2.6. Differentiating Addiction from Compulsion

Some people view non-drug use addictive behaviors, such as pathological gambling or shopping, as being "compulsions" [41], that involve (a) spontaneous desires to act a particular way, (b) a subjective sense of feeling temporarily out of control, (c) psychological conflict pertaining to the imprudent behavior, (d) "settling for less" to achieve the same ends, and (e) a disregard for negative consequences. Others use the term "compulsion" more narrowly. Some may define this term as a simple but intense urge to do something; only one aspect of addictions but centrally definitive of obsessive-compulsive disorders [61]. It may be defined even more precisely as an intense egodystonic (separate from self) urge to engage in a simple, repetitive activity, to remove anxiety [55]. Such activities may include repeated washing of hands, tying of shoes, or bathing, or restricting areas in which one will travel (e.g., not walking on cracks). A narrow definition of compulsion does not, primarily, consider the interplay of higher-order cognitive processes, such as the planning that may go into completion of a cycle of addictive behavior. (Arguably, however, someone may decide to wash their hands where there is plenty of soap available and the facility is considered very clean; this may involve planning). Also, the act may accomplish a temporary removal of anxiety, but it tends not to be experienced as pleasurable at any point in the engagement of the behavior [61]. Conversely, an addiction, by definition, involves the attempt to achieve some appetitive effect and satiation through engagement in some behavior. In fact, a whole constellation of purposeful behavior may be involved in attempts to achieve satiation [60].

3. Discussion and Conclusion

A series of complex, associated behaviors may be engaged in to continue to achieve appetitive effects. The problem with continued engagement in addictive-related behaviors is that over time they lead to negative side effects. The "addict" may then try to figure out new behaviors to achieve similar appetitive effects, while trying to avoid negative results. Over time, negative consequences may be greater than the positive consequences of engaging in any number of addictive behaviors. However, the participant may continue to engage in the behavior for several reasons. These reasons may include considering the behavior as a compromise between aspects of daily experience about which the participant feels a lack of control or accomplishment and aspects of experience the participant can manipulate. The behavior may be of a sort the participant can engage in relatively easily, and may still serve as a "short cut" to obtaining affective goals. The behavior may become a lifestyle, a means of existence. The stance the participant may take depends in part on the other activities about which the participant has access, or involvement. In the midst of the engagement in the addictive behavior, other competing behaviors may or may not be of interest unless woven into the fabric of the addictive behavior [45].

Formalized treatment may become imperative. Several overarching models have been proposed, contingent on whether or not the individual is considered to be held responsible for acquiring (yes/no) or resolving (yes/no) the addiction problem. Marlatt [57] categorized the acquiring-resolving typology as moral (where the individual is held personally responsible for both; considered weak-willed), medical/disease (responsible for neither; being physiologically disordered; must rely on external

support for prevention of addiction and to achieve and maintain sobriety, if addiction has set in), enlightenment (responsible only for the development of the problem; the person made repeated mistakes, leading to the problem which demands external support), and compensatory (being vulnerable, and responsible only for the solution; must take responsibility for change). Of course, individuals may vary along quantitative dimensions of voluntariness regarding acquiring and resolving an addictive disorder. Placing persons at different points along two such dimensions may be a more accurate portrayal of individual differences. More thinking on how conceptualizations of addiction interface with treatment implications is needed.

3.1. Philosophical Concerns

Since this is an inquiry into the content of a concept, or perhaps into the essence of the phenomenon denoted by that concept, a glance at some of the philosophical concerns this inquiry presents may be in order [1]. A natural first step is to look for the necessary and sufficient conditions for a psychological state or pattern of behavior to *be* an addiction. But it must be emphasized that there may be no such necessary and sufficient conditions for defining the term "addiction" or for being an addiction. Nevertheless, when we speak of "addictions" we are speaking of something rather than nothing and an inquiry into what it is may be illuminating.

It can be misleading to say we want to *define* "addiction"; that is, this may appear to say that our interest is in lexicography, in the *word* "addiction". But, of course, we all know how to use the term; we do not have to run for a dictionary whenever one speaks of addiction. Rather, our concern is to say what addiction *is*. Ideally, we would like to discover the necessary and sufficient conditions for someone to have an addiction, and to do so in such a way as to provide real illumination about the sort of phenomena we have in mind when thinking about addiction. We *know* we are talking about *something* that we recognize in certain clear cases and we want to know just what it is. Of course, we must be careful. There was a time when our ancestors wanted illuminating definitions of the ether that filled space, the spheres in which heavenly bodies were embedded as they spun around the earth, and the caloric that brought heat and left a burning object as it reduced to ash. As it happens, while our aforesaid ancestors were talking about *something* that they clearly did observe, the ether, spheres, and caloric did not exist. Natural philosophers may have tried mightily to define these "phenomena" in scientifically illuminating ways, but their efforts were hopeless for, again, these "things" did not exist. As we learn about the world we experience we do not merely learn more about things we know exist; we also change our beliefs about what things exist, even things as obvious as caloric and the ether [62].

Our "ontology" is the set of (kinds of) things that must exist if our beliefs are true. The point to note is that scientific progress involves not just changes in beliefs about the properties of and relations between the entities in our ontology—the ontology itself may well have to change. One often enlightening fact, or even guide, about those elements of our ontology that are destined for elimination is that efforts to provide illuminating necessary and sufficient conditions for their occurrence are frustrated. It is possible that, at some point in the evolution of our scientific arena, "addiction" *per se* may have to be eliminated from our scientific ontology. Of course, it would not follow that when we speak of addiction, we are speaking of *nothing*, just that we do not yet know *what* we really are talking about. We don't even know if there is one natural phenomenon involved [62,63].

Well, if addiction does not exist, what *are* we talking about when we use the word "addiction"? First, we may be talking about various things that can occur separately. For example, if we administer a lot of morphine over some time to an individual, he will become subject to a nasty withdrawal if we suddenly stop administering it (an aspect of preoccupation). He might also want us to stop the morphine immediately and go "cold turkey", to get the "monkey off his back". In these cases it is common to say that the individual is "addicted", although he did nothing to bring it about and does not want it to continue (no appetitive effect). Also, quitting may not be particularly difficult (no or little loss of control). On the other hand, some researchers would define addiction, in part, as a behavioral pattern that the individual's choices led to [2], which now has led to a personal sense of loss of control [8]. Moreover, some others would speak of addictions in which there is no real withdrawal syndrome at all (but there would be another manifestation of preoccupation)—a relatively new usage [33]. Very recently the emphasis has changed from addicting substances to various other addictive behaviors [59,60]. There may be a tendency to group all such instances together when in reality they may be different entities. Thus, possibly, the five definitional elements presented in this paper may represent different phenomena—they may not, only in conjunction, lead to a whole concept that we term "addiction".

Conversely, perhaps addiction is a disjunctive concept; that is, its definitional elements serve to create the concept. That is, perhaps one is addicted if he or she is demonstrates withdrawal-like phenomena or other aspects of preoccupation with an addictive behavior, even if he or she fails to demonstrate other definitional elements, such as loss of control. Certainly, the way addiction is used in everyday language is diverse. Its use in science may be less so, but still may refer to one definitional element *or* another, rather than one definitional element *and* another.

Thus, there are several possibilities regarding what the relationship is between "addiction" and the five aforementioned definitional elements. We note at least four. First, each of the elements alone, or in any combination with one or more of the others—perhaps all—may be a necessary condition of addiction. Second, each of the elements alone, or in any combination with one or more of the others—perhaps all—may be a sufficient condition of addiction. Third, the five criteria elements may provide a family resemblance concept of the phenomenon or phenomena denoted by "addiction" [64]. A family resemblance is best exemplified by, well, a family. We may say that all of the Smith's appear similar (e.g., on any number of elements contained within the family set, such as weight, eye color, hair, last name) even though no two are identical in descriptors (elements) examined and two of them within the group (set) may have no feature in common. Say Al and Mary have no one feature in common, and yet somehow Al and Mary both "look like members of the Smith family" (e.g., Al may have Smith weight and hair, but not eye color and last name; whereas Mary may have Smith eye color and last name, but not weight and hair). If there are two people who are labeled as addicted, but share none of the same elements in common (e.g., Al may experience loss of control and negative consequences whereas Mary may experience appetitive effects and preoccupation), by the way, we can expect to discover new phenomena of addiction that bear only a family resemblance to the ones we now recognize [64]. There is a fourth possibility; the definitional elements may turn out to be a grab bag of notions that fails to name any real kind of thing, any part of "the furniture of the universe".

A deeper understanding of the relevant phenomena may well lead us to see the *essence* of addiction as a certain brain state—say a particular degradation of the pleasure system—however that state came

about [18]. As an analogy, consider "water". Once chemistry did its job, we came to use "water" to denote a certain natural kind, H_2O [63]. We now say that in the old days folks were often mistaken when they called certain liquids "water"—even many of the constituents of the Chicago River. In summary, of course, there are phenomena of interest and importance at issue here, but the effort to define addiction in a way so as to make it a respectable scientific term is, at present, likely to commit errors of inclusion or exclusion.

There is another respect in which the concept of addiction may fall short of scientific respectability. Often, at least, one does not call something an addiction unless the relevant behavior is considered in some way "bad". That is, typically, for example, one does not claim to be addicted to breathing (c.f., [38]). Of course, there is also talk of "positive" addictions, such as work or exercise, but generally such talk is employed to show how these behaviors can lead to (the definitional element of) negative consequences [58,59], along with associated "bad" connotations (e.g., the person being selfish or out of control (a "loose cannon")). There is yet another related problem with the notion of addiction. The concept of an addiction is inextricably embedded in an interconnected matrix of common sense concepts such as desire, will, compulsion, pleasure and more. None of these concepts have the kind of clarity we want from terms in a well developed theory, terms such as H_2O, electron, glutamate or dendrite [62].

3.2. Limitations and Conclusions

From this discussion, and additional thought, one may infer at least four limitations with the presentation of the definitional elements provided in this paper. First, there was no attempt made in this paper to discuss how to measure each definitional element. For example, any of the definitional elements might be measured through use of self-report, implicit cognitive techniques, or through participant observation [27,51]. Rather, the goal was more modest, and future empirical work is needed (though some direction exists [58]).

Second, and a related point, is that it is not clear to what extreme a behavior must fall before it would be considered an "addiction". That is, one may ponder whether or not merely occasional (e.g., a couple of gambling sprees) or constant engagement (e.g., gambling to the point of bankruptcy) in an addictive behavior could indicate appetitive motive, preoccupation, loss of control, temporary satiation, and occurrence of negative consequences of sufficient magnitude or frequency to label the behavior as an addiction.

Third, there was no discussion of how the five elements might be related to each other. (This assumes that all five elements are necessary and sufficient constituents of addiction.) The reader may infer that we intended the operation of a linear process (appetitive motives to preoccupation to temporary satiation to loss of control to negative consequences). However, that was not intended. It is possible for example, that several feedback loops may be operative (e.g., appetitive engagement to temporary satiation to preoccupation to appetitive engagement again, eventually leading to loss of control and negative consequences). It is also possible that each element impacts in some way all other elements, a rather complex process.

A final limitation is that we did not place emphasis on the knowledge that addiction may be context-dependent. That is, what is considered an addiction in one social-environmental location may

not be considered addiction in another location [27]. For example, daily use of marijuana may be considered an addiction in the United States but not in some settings in Jamaica. Likewise, at a more specific level of inquiry, the five definitional elements offered herein (appetitive engagement, preoccupation, satiation, loss of control, and negative consequences) may vary in application in different social-environmental locations. It is likely that more extreme levels of each of these elements would be considered definitive of addiction when examining marijuana use in settings in Jamaica in comparison to the US; that is, it may be difficult to remove addiction and its defining elements from varying normative standards of behavior. A common scenario is one in which a person forgets where they parked their car after a drinking episode. This scenario might be interpreted as a negative consequence of drinking, or as a humorous story, depending on the social norms in operation when the event is being vocalized (e.g., at church *versus* a college fraternity gathering).

Of course, if there were no negative consequences, and if the experience was always fulfilling, one may speculate that the addictive behavior would continue, and may or may not even be considered addictive (c.f., or as a positive addiction). However, continued engagement in the addictive experience may limit the options to engage in alternative experiences. While not leading to dramatic negative consequences *per se*, the experience may conflict with other desired patterns of behavior. Arguably, most addictive behaviors do not simply terminate when one desires to stop. There is a "recovery period" in which one anticipates feeling better physically, financially, or otherwise. Then one may consider other pathways of action. These other pathways may be harder to achieve the more entrenched one has become in the addictive behavior. In fact, involvement in addictive behavior may reflect a dialectic; that is, an approach-avoidance response. That is, the participant desires the behavior, its satiation, but also desires its avoidance; one equivocates in preference. This equivocation may serve as the experiential substrate of an appetitive motive and satiation, and loss of control and preoccupation, and may itself be experienced as a negative consequence.

It is hoped that the five definitional elements of the concept of addiction presented herein provide a heuristic basis for greater consensus and forward thinking. Certainly, to define addiction in a meaningful, important way (*i.e.*, not mere lexicography) it is necessary to develop a well confirmed, substantive *theory*—just as the definition of water requires chemical theory. Definition, theory construction and confirmation are not separate activities. And it is quite possible that there is no theory that could justify one precise concept of addiction that would cover all the phenomena of concern, including encompassment of all five definitional elements. Or not—it depends on further empirical and theoretical work. Much work remains to be done to fine-tune the definition of addiction, which will entail theoretical modeling.

Acknowledgments

The authors would like to acknowledge Steven E. Wallis and John Monterosso for their very helpful comments on an earlier draft of this manuscript.

Conflict of Interest

The authors declare no conflict of interest.

References

1. Rudner, R.S. *Philosophy of Social Science*; Prentice Hall: Englewood Cliffs, NJ, USA, 1966.
2. Foddy, B.; Savulescu, J. A liberal account of addiction. *Philos. Psychiatry Psychol.* **2010**, *17*, 1-22.
3. Foddy, B.; Savulescu, J. Relating addiction to disease, disability, autonomy, and the good life. *Philos. Psychiatry Psychol.* **2010**, *17*, 35-42.
4. Goodman, A. Addiction: Definition and implications. *Br. J. Addict.* **1990**, *85*, 1403-1408.
5. Horne, M. Johnny Wilkinson's addiction. *Philos. Psychiatry Psychol.* **2010**, *17*, 31-34.
6. Larkin, M.; Griffiths, M.D. Response to Shaffer (1996): The case for a 'complex systems' conceptualization of addiction. *J. Gambl. Stud.* **1998**, *14*, 73-82.
7. Mathews, E. Explaining addiction. *Philos. Psychiatry Psychol.* **2010**, *17*, 23-26.
8. Nordenfelt, L. On concepts and theories of addiction. *Philos. Psychiatry Psychol.* **2010**, *17*, 27-30.
9. Shaffer, H.J. The most important unresolved issue in the addictions: Conceptual chaos. *Subst. Use Misuse* **1997**, *32*, 1573-1580.
10. Alexander, B.K.; Schweighofer, A.F. Defining "addiction". *Can. Psychol.* **1988**, *29*, 151-162.
11. Levine, H.G. The discovery of addiction: Changing conceptions of habitual drunkenness in America. *J. Stud. Alcohol* **1978**, *39*, 143-174.
12. Orford, J. Addiction as excessive appetite. *Addiction* **2001**, *96*, 15-31.
13. Meyer, R.E. The disease called addiction: Emerging evidence in a 200-year debate. *Lancet* **1996**, *347*, 162-166.
14. Bechara, A. Risky business: Emotion, decision making, and addiction. *J. Gambl. Stud.* **2003**, *19*, 23-51.
15. Bechara, A. Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nat. Neurosci.* **2005**, *8*, 1458-1463.
16. Di Chiara, G. Nucleus accumbens shell and core dopamine: Differential role in behavior and addiction. *Behav. Brain Res.* **2002**, *137*, 75-114.
17. Feltenstein, M.W.; See, R.E. The neurocircuitry of addiction: An overview. *Br. J. Pharmacol.* **2008**, *154*, 261-274.
18. Goodman, A. The neurobiological development of addiction: An overview. *Psychiatr. Times* **2009**, *26*, 1-14.
19. Heather, N. A conceptual framework for explaining drug addiction. *J. Psychopharmacol.* **1998**, *12*, 3-7.
20. Barry, D.T. Review of the globalization of addiction. A study in poverty of the spirit. *J. Nerv. Ment. Dis.* **2010**, *198*, 462.
21. Griffiths, M.S.; Larkin, M. Conceptualizing addiction: The case for a "complex systems" account. *Addict. Res. Theory* **2004**, *12*, 99-102.
22. Hatterer, L.J. The addictive process. *Psychiatr. Q.* **1982**, *54*, 149-156.
23. Jacobs, D.F. A general theory of addictions: A new theoretical model. *J. Gambl. Stud.* **1986**, *2*, 15-31.
24. Haylett, S.A.; Stephenson, G.M.; Lefever, R.M.H. Covariation in addictive behaviors: A study of addictive orientations using the shorter PROMIS Questionnaire. *Addict. Behav.* **2004**, *29*, 61-71.

25. MacLaren, V.V.; Best, L.A. Multiple addictive behaviors in young adults: Student norms for the shorter PROMIS Questionnaire. *Addict. Behav.* **2010**, *35*, 252-255.
26. Larkin, M.; Wood, R.T.A.; Griffiths, M.D. Toward addiction as relationship. *Addict. Res. Theory* **2006**, *14*, 207-215.
27. Sussman, S.; Ames, S.L. *Drug Abuse: Concepts, Prevention and Cessation*; Cambridge University Press: New York, NY, USA, 2008.
28. Haertzen, C.A.; Kocher, T.R.; Miyasato, K. Reinforcements from the first drug experience can predict later drug habits and/or addiction: Results with coffee, cigarettes, alcohol, barbiturates, minor and major tranquilizers, stimulants, marijuana, hallucinogens, heroin, opiates and cocaine. *Drug Alcohol Depend.* **1983**, *11*, 147-165.
29. Campbell, W.G. Addiction: A disease of volition caused by a cognitive impairment. *Can. J. Psychiatry* **2003**, *48*, 669-674.
30. Robinson, T.E.; Berridge, K.C. Mechanisms of action of addictive stimuli. Incentive-sensitization and addiction. *Addiction* **2001**, *96*, 103-114.
31. Robinson, T.E.; Berridge, K.C. Review. The incentive sensitization theory of addiction: Some current issues. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* **2008**, *363*, 3137-3146.
32. Goodman, A. Neurobiology of addiction: An integrative review. *Biochem. Pharmacol.* **2008**, *75*, 266-322.
33. Leshner, A.I. Addiction is a brain disease, and it matters. *Science* **1997**, *278*, 45-47.
34. Potenza, M.N. What integrated interdisciplinary and translational research may tell us about addiction. *Addiction* **2010**, *105*, 790-796.
35. American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*; American Psychiatric Association (APA): Washington, DC, USA, 2000.
36. Chassin, L.; Presson, C.C.; Rose, J.; Sherman, S.J. What is addiction? Age-related difference in the meaning of addiction. *Drug Alcohol Depend.* **2007**, *87*, 30-38.
37. Greenberg, J.L.; Lewis, S.E.; Dodd, D.K. Overlapping addictions and self-esteem among college men and women. *Addict. Behav.* **1999**, *24*, 565-571.
38. Marks, I. Behaviour (non-chemical) addictions. *Br. J. Addict.* **1990**, *85*, 1389-1394.
39. American Psychiatric Association. Proposed Revisions, 2010. http://www.dsm5.org/ ProposedRevisions/Pages/proposedrevision.aspx?rid=452 (accessed on 28 June 2011).
40. Pelchat, M.L. Of human bondage: Food craving, obsession, compulsion, and addiction. *Physiol. Behav.* **2002**, *76*, 347-352.
41. Hirschman, E.C. The consciousness of addiction: Toward a general theory of compulsive consumption. *J. Consum. Res.* **1992**, *19*, 155-179.
42. Pearson, M.M.; Little, R.B. The addictive process in unusual addictions: A further elaboration of etiology. *Am. J. Psychiatry* **1969**, *125*, 1166-1171.
43. Sussman, S.; Unger, J.B. A "drug abuse" theoretical integration: A transdisciplinary speculation. *Subst. Use Misuse* **2004**, *39*, 2055-2069.
44. Cox, W.M.; Klinger, E. A motivational model of alcohol use. *J. Abnorm. Psychol.* **1988**, *97*, 168-180.
45. Glasner, S.V. Motivation and addiction: The role of incentive processes in understanding and treating addictive disorders. In *Handbook of Motivational Counseling*; Cox, W.M., Klinger, E., Eds.; John Wiley & Sons: Hoboken, NJ, USA, 2004; pp. 29-47.

46. Loonis, E.; Apter, M.J.; Sztulman, H. Addiction as a function of active system properties. *Addict. Behav.* **2000**, *25*, 477-481.
47. Miller, W.R. Addictive behavior and the theory of psychological reversals. *Addict. Behav.* **1985**, *10*, 177-180.
48. Ainslee, G. *Picoeconomics: The Strategic Interaction of Successive Motivational States within the Person*; Cambridge University Press: New York, NY, USA, 1992.
49. Iannaccone, L.R. Addiction and satiation. *Econ. Lett.* **1986**, *21*, 95-99.
50. Cassin, S.E.; von Ranson, K.M. Is binge eating experienced as an addiction? *Appetite* **2007**, *49*, 687-690.
51. Stacy, A.W.; Ames, S.L.; Knowlton, B. Neurologically plausible distinctions in cognition relevant to drug abuse etiology and prevention. *Subst. Use Misuse* **2004**, *39*, 1571-1623.
52. Tiffany, S.T. A cognitive model of drug urges and drug-use behavior: The role of automatic and non-automatic processes. *Psychol. Rev.* **1990**, *97*, 147-168.
53. Walters, G.D.; Gilbert, A.A. Defining addiction: Contrasting views of clients and experts. *Addict. Res. Theory* **2000**, *8*, 211-220.
54. Wiers, R.W.; Ames, S.L.; Hoffmann, W.; Krank, M.; Stacy, A.W. Impulsivity, impulsive and reflective processes and the development of alcohol use and misuse in adolescents and young adults. *Front. Psychology* **2010**, *1*, 1-12.
55. Brewer, J.A.; Potenza, M.N. The neurobiology and genetics of impulse control disorders: Relationships to drug addictions. *Biochem. Pharmacol.* **2008**, *75*, 63-75.
56. White, N.M. Addictive drugs as reinforcers: Multiple partial actions on memory systems. *Addiction* **1996**, *91*, 921-949.
57. Marlatt, G.A. Relapse prevention: Theoretical rationale and overview of the model. In *Relapse Prevention*, 1st ed.; Marlatt, G.A., Gordon, J.R., Eds.; The Guilford Press: New York, NY, USA, 1985; pp. 3-70.
58. Schneider, J.P.; Irons, R.R. Assessment and treatment of addictive sexual disorders: Dependency relapse. *Subst. Use Misuse* **2001**, *36*, 1795-1820.
59. Sussman, S.; Lisha, N.; Griffiths, M. Prevalence of the addictions: A problem of the majority or the minority. *Eval. Health Prof.* **2010**, *34*, 3-56.
60. Sussman, S.; Leventhal, A.; Bluthenthal, R.N.; Freimuth, M.; Forster, M.; Ames, S.L. A framework for the specificity of addictions. *Int. J. Environ. Res. Public Health* **2011**, *8*, 3399-3415.
61. Hartney, E. The difference between an addiction and a compulsion, 2011. Available online: http://addictions.about.com/od/howaddictionhappens/a/addcompulsion.htm?rd=1 (accessed on 30 July 2010).
62. Churchland, P.M. Eliminative materialism and the propositional attitudes. *J. Philos.* **1981**, *78*, 67-90.
63. Ellis, B. *Scientific Essentialism, Cambridge Studies in Philosophy*; Cambridge University Press: Cambridge, UK, 2001.
64. Wittgenstein, L. *Philosophical Investigations*; Basil Blackwell Publishing: Oxford, UK, 1953.

International Journal of
Environmental Research and Public Health

Aims and Scope

International Journal of Environmental Research and Public Health (IJERPH) is a peer-reviewed scientific journal that publishes original articles, critical reviews, research notes, and short communications in the interdisciplinary area of environmental health sciences and public health. It links several scientific disciplines including biology, biochemistry, biotechnology, cellular and molecular biology, chemistry, computer science, ecology, engineering, epidemiology, genetics, immunology, microbiology, oncology, pathology, pharmacology, and toxicology, in an integrated fashion, to address critical issues related to environmental quality and public health. Therefore, IJERPH focuses on the publication of scientific and technical information on the impacts of natural phenomena and anthropogenic factors on the quality of our environment, the interrelationships between environmental health and the quality of life, as well as the socio-cultural, political, economic, and legal considerations related to environmental stewardship and public health. The primary areas of research interests to the IJERPH include:

- Gene-environment interactions
- Environmental genomics and proteomics
- Environmental toxicology, mutagenesis and carcinogenesis
- Environmental epidemiology and disease control
- Health risk assessment and management
- Ecotoxicology, and ecological risk assessment and management
- Natural resources damage assessment
- Environmental chemistry and computational modeling
- Environmental policy and management
- Environmental engineering and biotechnology
- Emerging issues in environmental health and diseases
- Environmental education and public health

Therefore this international journal covers a broad spectrum of important topics which are relevant to environmental health sciences and public health protection. It provides comprehensive and unique information with a worldwide readership. Emphasizing holistic approach, the journal serves as a comprehensive and multidisciplinary platform, addressing important public health issues associated with environmental pollution and degradation. A large number of eminent professors and scientists from all over the world serve as guest reviewers for the journal.

Indexing & Abstracting

International Journal of Environmental Research and Public Health is covered by following indexing and abstracting databases:

- CAB Abstracts
- Chemical Abstracts (CAS)
- Current Contents
- Directory of Open Access Journals (DOAJ)
- e-Helvetica (Swiss National Library Archive)
- EBSCOhost
- EMBASE
- Food Science and Technology Abstracts (FSTA)
- Global Health
- Google Scholar
- IndexCopernicus
- Journal Citation Report
- Open-J-Gate
- PubMed / MEDLINE
- PubMed Central
- Science Citation Index Expanded (SCIE) / Web of Science
- Scopus / SciVerse
- Ulrich's™

International Journal of Environmental Research and Public Health is published by MDPI monthly. If you would like to submit a paper, please first visit the Instructions for Authors website (www.mdpi.com/journal/ijerph/instructions/). Manuscripts should be submitted through the online manuscript submission and editorial system at www.mdpi.com/user/manuscripts/upload.

MDPI publishes several peer-reviewed, open access journals listed at www.mdpi.com. All MDPI journals maintain a rapid, yet rigorous, peer-review and editorial process.

MDPI AG
Postfach, CH-4005 Basel, Switzerland
Office: Kandererstrasse 25, CH-4057 Basel, Switzerland
Tel. +41 61 683 77 34
Fax +41 61 302 89 18
www.mdpi.com

Final Acknowledgements

I would like to thank the Guest Editor Professor Steve Sussman of this special issue "Substance and Behavioral Addictions: Co-Occurrence and Specificity" for his excellent contribution and effort. I would also like to thank the authors for so many relevant and high quality papers within the specific topic of this special issue, which together are an important contribution to studies on addictions.

I would like to use this opportunity to extend a general invitation to scholars whose expertise is relevant to environmental health sciences and public health protection to submit their original research articles or reviews to our journal. Finally, the journal also welcomes proposals from scholars interested in guest editing a special issue; please contact the Editorial Office at ijerph@mdpi.com.

Sincerely,

Dr. Shu-Kun Lin
Publisher

This book contains the printed edition of the special issue on Substance and Behavioral Addictions: Co-Occurrence and Specificity that appeared in *International Journal of Environmental Research and Public Health* (http://www.mdpi.com/journal/ijerph/). *International Journal of Environmental Research and Public Health* (ISSN 1660-4601; CODEN: IJERGQ; ISSN 1661-7827 for printed edition) is an interdisciplinary open access journal, published by MDPI online monthly.

Multidisciplinary Digital Publishing Institute (MDPI)
Basel, Switzerland

ISBN 3-906980-32-4