Psychological Bulletin
2004, Vol. 130, No. 4, 531–552


0033-2909/04/$12.00 DOI: 10.1037/0033-2909.130.4.531

# Psychophysiology of Aggression, Psychopathy, and Conduct Problems: A Meta-Analysis

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A meta-analysis of 95 studies was conducted to investigate the relations of heart rate (HR) and electrodermal activity (EDA) with aggression, psychopathy, and conduct problems. Analyses revealed a complex constellation of interactive effects, with a failure in some cases of autonomic patterns to generalize across antisocial spectrum behavior constructs. Low resting EDA and low task EDA were associated with psychopathy/sociopathy and conduct problems. However, EDA reactivity was positively associated with aggression and negatively associated with psychopathy/sociopathy. Low resting HR and high HR reactivity were associated with aggression and conduct problems. Physiology–behavior relations varied with age and stimulus valence in several cases. Empirical and clinical implications are discussed.

The physiological correlates of antisocial spectrum behavior—including aggression, psychopathy, conduct problems, and antisocial personality characteristics—have been the subject of a good deal of theoretical and empirical attention over the past 45 years (see Scarpa & Raine, 1997). Understanding possible links between autonomic physiology and antisocial spectrum behavior may hold special promise for advancing our understanding of these highly costly behavior patterns. Autonomic measures move beyond self-reports of psychological functioning and may be less prone to bias and measure-related error. They may also be more sensitive indexes of the brain processes that are ultimately thought to be responsible for the behavior (e.g., Fowles, 1980, 1988). Convincingly demonstrating links between antisocial spectrum behavior and autonomic physiology might elucidate relevant mechanisms and individual differences and potentially lead to treatment innovations. This is especially important with regard to antisocial spectrum behavior, because of its enormous cost to society (e.g., Moffitt, Caspi, Harrington, & Milne, 2002) and its relative resistance to treatment in many of its forms (e.g., Offord & Bennet, 1994; Richards, Casey, & Lucente, 2003), with the possible exception of conduct problems in young children (e.g., Brestan & Eyberg, 1998).

Many different theories of antisocial spectrum behavior that involve autonomic psychophysiology have been proposed (Berkowitz, 1994; Eysenck & Gudjonsson, 1989; Fowles, 1980, 1988; Gray, 1987; Hare, 1978a; Quay, 1965; Raine, 1993, 1997; Zillmann, 1994; Zuckerman & Como, 1983), some with conflicting predictions (e.g., Quay, 1965, and Zillmann, 1994). Furthermore, a wide range of experimental methods and populations have been employed and studied, respectively. Clear interpretation of the literature has proved elusive in the face of a mixed pattern of results. In an attempt to clarify these issues, the present meta-analytic review was designed to quantify the aggregate associations of heart rate (HR) and electrodermal activity (EDA) with aggression, psychopathy, conduct problems, and antisocial personality characteristics and to investigate factors that moderate these relations.

HR and EDA are variables of central interest in many theories of physiology and antisocial spectrum behavior and have historically been the most popular measures of psychophysiological response. Thus, the present analysis was limited to these two measures. The heart is subject to influence from the sympathetic (SNS) and parasympathetic (PNS) branches of the autonomic nervous system, and it is subject to neuroendocrine influences as well. Chronotropic (i.e., rate-related) cardiac effects are controlled primarily by the PNS, whereas inotropic effects such as contractile force and stroke volume are controlled primarily by the SNS. In contrast, EDA is under exclusive control of the SNS (Blascovich & Kelsey, 1990; Fowles, 1986). From a perspective dating back to the seminal work of James and Cannon, HR and EDA are thought to be tied to emotional responses, with increases reflecting general emotional arousal, specific emotions, or both (reviewed in Lang, 1994). HR and EDA may also reflect the influence of motivational systems that control behavior in response to internal and external cues (e.g., Fowles, 1988; Gray, 1987).

The present meta-analysis focused on three classes of HR and EDA measures: resting, task, and reactivity. Resting measures reflect the assessment of autonomic activity in the absence of any obvious external stimuli. HR and EDA in response to experimental stimuli are frequently measured in raw form during such presentations (task physiology) or expressed as a change from resting, baseline, or prestimulus levels (physiological reactivity).

## Problems With Interpretation of the Literature

Because of frequent nonreplications and theories that make competing predictions, the literature on the psychophysiology of antisocial spectrum behavior is often confusing and seemingly contradictory. The major thesis of this meta-analytic review is that these ambiguities are caused in part by two sources: heterogeneity

I would like to thank Theodore Beauchaine, K. Daniel O'Leary, Susan G. O'Leary, Amy Smith Slep, and Paul Wortman for their helpful comments on earlier versions of this article and Tamara Del Vecchio for her coding assistance.

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in the behavior construct and heterogeneity in experimental methods. Furthermore, many of the models of the relation between physiology and antisocial spectrum behavior have been applied to adults, adolescents, and children. The generalization of psychophysiological models across different age groups is in need of empirical evaluation given that no study of which I am aware has examined the association between antisocial spectrum behavior and physiology in both adults and children.

### *Heterogeneity in Behavior Construct*

Just what form of antisocial spectrum behavior ought to relate to what specific pattern of physiological response is unclear at present. Often, multiple behavior patterns are discussed together. For example, Scarpa and Raine's (1997) review mixed aggression, crime, psychopathy, antisocial behavior, and conduct disorder. Although there is some degree of overlap among these constructs, there is also some distinctiveness. For example, as Lynam (1996) pointed out, psychopathy and antisocial personality disorder (APD) refer to different constellations of behavioral characteristics. Following Cleckly's (1976) conceptualization, *psychopathy* refers to impulsiveness, lack of empathy and guilt, and narcissism, often accompanied by severe violence. As indicated in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994), APD is characterized by a pervasive pattern of disregard for social norms and violation of the rights of others. In fact, whereas nearly all psychopathic offenders meet APD diagnostic criteria, only approximately 25% of APD-diagnosed men meet the criteria for psychopathy (Hare, 1985), as assessed by Hare's (1991) Psychopathy Checklist.

Further confusion results from the overuse of the term *antisocial*. Not all antisocial behavior is equivalent, and not all antisocial behavior is aggressive. For example, although an individual diagnosed with APD and a one-time bar brawler may both exhibit antisocial behavior (i.e., violation of personal rights, laws, and social norms), placing them into a single category ignores the heterogeneity between these individuals. For instance, many individuals with APD are aggressive, but not all aggressors meet diagnostic criteria for APD. The non-APD aggressor may not share the callousness and pervasive violation of the rights of others exhibited by the APD-diagnosed individual.

Perhaps more important, although psychopathy and APD are associated with aggression, aggression is not the singular defining characteristic of these syndromes. It is unclear whether the psychophysiological response profiles of psychopathic and antisocial individuals are specifically related to aggression, to other characteristics, or to both. For example, aggression is frequently confounded with the personality characteristics of individuals with psychopathy. Indeed, Kelsey, Ornduff, McCann, and Reiff (2001) found that the psychophysiological responses of individuals high in narcissism—which plays a prominent part in the personality profiles of psychopaths (Kernberg, 1989, cited in Kelsey et al., 2001)—in many ways mirror those of psychopaths. Thus, narcissism may be at the root of the physiological response patterns of psychopaths.

It is perhaps not surprising, then, that findings differ between samples of psychopaths and those who exhibit other forms of antisocial or aggressive behavior, with an important caveat that psychopathic or antisocial personality characteristics are usually not assessed in studies of aggressive behavior. For example, the man who pushes his wife twice per year is probably from a different population than the felon with multiple convictions for aggravated assault who exhibits psychopathic tendencies across contexts. It follows that the psychophysiological profiles of nonpsychopathic and nonantisocial aggressors may also be different from those of their more pathological counterparts. In the present article, the term *antisocial spectrum behavior* is used as shorthand and may be thought of as a superordinate category that captures the violation of rules and the rights of others common to aggression, psychopathy, antisocial personality, and conduct problems.

### *Heterogeneity in Experimental Stimuli*

Even an incomplete list of the experimental stimuli used by researchers to study the psychophysiology of antisocial spectrum behavior would reveal substantial variability. Different pockets of research tend to use characteristic stimuli, reflecting different theoretical propositions of the circumstances under which physiological changes are associated with antisocial spectrum behavior. For example, Hinton, O'Neill, Hamilton, and Burke (1980) exposed participants to neutral orienting tones, whereas Milner (1976) used electric shocks in studying the autonomic responses of psychopaths. In family aggression research, Frodi and Lamb (1980) exposed mothers to infant cries and smiles; in studies of marital aggression (e.g., Jacobson et al., 1994), couples typically engage in conflict. These differences may be related to apparent inconsistencies in the literature.

One dimension that may hold promise for disentangling these inconsistencies is the valence of experimental stimuli. Valence is defined herein as the hedonic value of a stimulus. Valence cuts across the stimuli used in the literatures under consideration, thus allowing for parallel consideration of its impact across these disparate literatures. Furthermore, many of the theoretical perspectives on the psychophysiology of antisocial spectrum behavior make predictions specific to a negatively valenced antecedent. For example, Zillmann (1994) posited that physiological reactivity is elicited by frustrating events, triggering an aggressive response. Learning theories (e.g., Fowles, 1988) are concerned with an individual's ability to inhibit an aggressive/impulsive response given impending deleterious consequences. Thus, it was hypothesized that studies involving negatively valenced stimuli would be associated with stronger effects than would studies involving nonnegative stimuli.

### *Heterogeneity in Age*

It is unclear whether autonomic patterns associated with aggression and antisocial spectrum behaviors generalize across different age groups. On one hand, there is substantial continuity in antisocial behavior. For example, the previous presence of conduct disorder in childhood or adolescence is a diagnostic criterion for APD (American Psychiatric Association, 1994). Childhood conduct problems appear to be associated not only with later APD but with other forms of aggressive and criminal behavior as well. For example, Huesmann, Eron, Lefkowitz, and Walder (1984) found that boys identified as aggressive were more likely to abuse their spouses as adults, as well as to commit serious crimes and drive while intoxicated. Similarly, Magdol, Moffitt, Caspi, and Silva (1998) found that children with early problem behaviors were

more likely to exhibit partner aggression as adults. Thus, it may be the case that models developed to explain antisocial spectrum behavior in adulthood apply to children and adolescents as well. For instance, Lynam (1997) found evidence that children with early-onset conduct disorder also exhibit personality characteristics that resemble those of adult psychopaths.

Parallels have been found in psychophysiological research as well. Low EDA has been found in studies of youth (see Quay, 1993) and adults (see Scarpa & Raine, 1997) who exhibit antisocial spectrum behaviors. Thus, the psychophysiological profiles of children and adolescents with conduct problems, if sufficiently stable, may also be predictive of other forms of aggressive or antisocial behavior in adulthood. On the other hand, although the retrospective relation of childhood conduct problems to adult pathology is strong, prospective prediction is far weaker, with a high false-positive rate. For example, only a minority of children diagnosed with conduct disorder go on to become diagnosed with APD as adults (Hinshaw & Anderson, 1996). As illustrated in the work of Moffitt (1993), as well as Loeber and Stouthamer-Loeber (1998), this false-positive rate may be attributable to multiple developmental pathways of antisocial behavior. The majority of youth with conduct problems exhibit time-limited antisocial behavior that appears to peak in adolescence in most individuals and in the preschool–elementary school age period in others (Loeber & Stouthamer-Loeber, 1998; Moffitt, 1993). A much smaller group, which Moffitt labeled life-course-persistent antisocial individuals, exhibit a relatively continuous course of antisocial behavior beginning in childhood and enduring into adulthood. Thus, psychophysiological models developed in research on psychopathic or antisocial adults may apply to only a subset of children with conduct problems (i.e., life-course-persistent antisocial children).

## Goals of the Present Meta-Analysis

As a means of addressing the problems just outlined, the central aim of the present meta-analysis was to evaluate the extent to which the relations of antisocial spectrum behavior with HR and EDA depend on type of behavior in question, valence of experimental stimuli, and age. Furthermore, the meta-analysis provided an opportunity to document the aggregate association of each form of antisocial spectrum behavior with HR and EDA. These issues were addressed in analyses of resting, task, and reactivity measures of autonomic physiology. By quantifying these associations and elucidating potential interactions, it was hoped that some of the ambiguities that exist in the literature might be resolved.

## Method

### *Literature Searches*

Four methods were used to locate studies for the meta-analysis. First, PsycINFO (1887–2002), MEDLINE (1966–2002), and *Dissertation Abstracts International* (1861–2002) were searched through January of 2002 through the use of the key words *interbeat, heart rate, cardiac, electrodermal, skin conductance, skin potential, galvanic, physiology,* and *psychophysiology,* cross referenced with *aggression, violence, psychopathy, conduct disorder, conduct problems, antisocial, oppositional, child abuse,* and *spouse abuse*. Second, the reference lists of retrieved articles, as well as several review articles on the relation of psychophysiology with aggression and antisocial behavior (Fowles, 1980, 1988; Hare, 1978a; Kilzieh & Cloninger, 1993; Quay, 1993; Scarpa & Raine, 1997), were scanned. Third, several investigators were contacted in an attempt to locate unpublished dissertations and articles in press (A. Raine, L. Berkowitz, D. Zillmann, E. Mezzacappa, T. Zahn, R. Hare, and J. Knutson). Fourth, queries were posted via relevant Internet sites (e.g., Society for Psychophysiological Research) and electronic mail discussion groups (e.g., Society for Scientific Clinical Psychology).

### *Inclusion and Exclusion Criteria*

One hundred studies satisfied the inclusion criteria. Publication years of the retained studies ranged from 1957 to 2001. Inclusion criteria were liberal by design, in that a substantial goal of the meta-analysis was to test interactions of effect sizes with study features rather than holding these features constant. Studies had to include HR, EDA, or both, measured either during a baseline period or in an experimental task, and an assessment of aggression, antisocial personality characteristics, conduct problems, or psychopathy/sociopathy. EDA measures had to be either skin conductance level, amplitude, or frequency. Studies could either compare the physiology of an antisocial group against a control group or use correlational methods. Control groups could include less aggressive, psychopathic/sociopathic, antisocial, conduct-disordered, or normal participants. Furthermore, the studies had to include sufficient data to allow calculation of effect sizes (pairwise comparisons or correlations, sample size, and a test statistic or $p$ value for each test of interest). Longitudinal studies without relevant concurrent tests, studies of animals, articles written in languages other than English, and studies of individuals with developmental disabilities (e.g., autism) were excluded. Studies in which antisocial spectrum behavior or physiology were manipulated experimentally were also excluded, reflecting my concern with individual differences. Finally, only journal articles that were either published, in press, or in advanced stages of review (i.e., "revise and resubmit" status) and dissertations were included so as to restrict the sample to work that had undergone scientific review. When a study was available both as a dissertation and as a published article, the published article was selected.

### *Coding of Moderators*

*Age.* Age groups were coded as follows: child (1–11 years old), adolescent (12–17 years old), or adult (18 years old or older). Several studies spanned two age groups. In these cases, age category was determined by the mean age of the sample. Furthermore, age was not reported in several studies of incarcerated individuals. In these cases, a code of adult was assigned.

*Behavior type.* Behavior type was coded as one of four categories: psychopathy/sociopathy, aggression, antisocial personality characteristics, or conduct problems. The psychopathy/sociopathy code was assigned if the author(s) used (a) a measurement device purporting to measure psychopathy or sociopathy, (b) research or clinical interview data, or (c) a combination thereof to establish group membership or dimensional level of psychopathy/sociopathy. The aggression code was assigned for studies of physical behavior intended to harm or injure another person, including studies of fighting with peers, shocking laboratory confederates, and physical aggression in families. The antisocial personality characteristics code[1] was assigned for studies of antisocial personality characteristics common to APD (e.g., failure to conform to social norms, deceitfulness, manipulativeness, and lack of remorse) or diagnosed APD itself. The conduct problems code was applied for studies of diagnosed conduct disorder,

[1] I would have liked to distinguish between antisocial personality characteristics and formally diagnosed APD. However, there were not enough studies to include APD as a separate behavior category. Thus, it was included in the antisocial personality characteristics category.

delinquency, oppositionality, and other child and adolescent externalizing behavior problems. Studies of problems involving attention or hyperactivity (e.g., attention-deficit/hyperactivity disorder; American Psychiatric Association, 1994), however, were excluded from this category because other biological contributions to attention-deficit/hyperactivity disorder may confound the relation between physiology and conduct problems.

*Type of physiological measure.* Type of HR measure was coded as resting, task, or reactivity. The resting code was assigned if HR was measured in the absence of stimuli. In the case of multiple rest periods, the effect size associated with the first was selected. The task code was assigned if HR was measured (for any duration of time) while the participant performed a task, such as listening to tones or performing mental arithmetic. The reactivity code was assigned if HR was expressed as change from a baseline or prestimulus measurement to a task measurement or if baseline HR was statistically controlled. Coding of EDA followed the same procedure as HR.

*Stimulus valence.* Stimuli were coded as either negative or nonnegative. A code of negative was assigned if the stimuli used were designed to be anger provoking, very loud (e.g., tones $\geq$ 90 db), painful (e.g., electric shock), or otherwise aversive (e.g., pictures of mutilated body parts). A code of nonnegative was assigned for all other stimuli (e.g., orienting tones or slides of affectively neutral images).

*Reliability.* Coding was performed by the author, with 24 articles coded in parallel by an independent coder unaware of the hypotheses. Cohen kappa values for age and behavior type were 1.00, with kappas of .80 and .70 for stimulus valence and type of physiological measure, respectively.

## Computation of Effect Sizes

*Cohen's d.* As recommended by Hedges and Olkin (1985), Cohen's corrected *d* statistic was chosen as the measure of effect size; *d* represents the difference between treatment and control group means divided by the pooled standard deviation. The corrected *d* adjusts for bias resulting from small sample sizes. Cohen's *d* can be calculated from a variety of descriptive and inferential statistics, including means and standard deviations, test statistics, and *p* values. Positive effect sizes in the present meta-analysis represented positive associations between physiology and antisocial spectrum behavior. Effect sizes were calculated with Johnson's (1995) DSTAT program. Following Cohen's (1988) recommendations, small, medium, and large effect sizes were defined by *d* values of 0.20, 0.50, and 0.80, respectively.

*Multiple effect sizes.* It was common for studies to report enough data to compute multiple effect sizes. The assumption of independence would be violated by including more than one effect size per study in a given analysis. Many included baseline and task or reactivity measures of both HR and EDA. In such cases, no threat to the independence assumption was present, because analyses of HR and EDA and analyses of baseline and task-reactivity measures were conducted separately. However, it was common for studies to include multiple assessments within a given measure (e.g., EDA in two different tasks).

In addition, there were sometimes multiple types of EDA measurement (skin conductance level, amplitude, and frequency) and multiple measures of aggression or antisocial behavior. In these cases, one effect size was randomly selected for analysis. One exception to random selection was the case of Babcock, Yerington, Green, and Webb's (2004) study, which included analyses of psychopathy, antisocial behavior, and multiple measures of domestic violence. Because of the low number of studies of family aggression, one of the analyses pertaining to spousal aggression was randomly selected. Multiple effect sizes also occurred in studies in which other (unanalyzed) independent variables were either manipulated or selected. For example, psychopathic individuals and controls might be split into subgroups high and low in anxiety. In such cases, effects were averaged across conditions. Also, when studies reported on multiple distinguishable samples (e.g., men and women, different age cohorts, or multiple experiments), these samples were treated as independent. Finally, there were multiple experimental groups in several studies. For example, psychopathic individuals were often subclassified as exhibiting primary or secondary psychopathy (e.g., Blackburn, 1979). In the Babcock et al. (2004) study, abusive husbands were broken into high-level and low-level groups. In instances in which enough information was provided to do so, data were averaged to form pairwise comparisons (e.g., individuals with primary/secondary psychopathy vs. controls or high-level/low-level partner aggressors vs. controls). In other cases, one comparison was randomly selected for analysis.

*Nonsignificant effects.* Nonsignificant effects with inadequate information to calculate effect sizes (i.e., no test statistic or *p* value) were assigned a *d* value of zero, provided that the comparison was pairwise or a correlation and the corresponding sample size was reported. This is a conservative approach that often results in effect size estimates that are too small. However, excluding such studies leads to inflated effect sizes and artificially small *p* values (Rosenthal, 1995). As a result of the desire to maximize the number of studies and minimize Type I error, the former approach was adopted.

## Data Analysis

In the first step of the data analysis, effect sizes were calculated separately for relations of aggression, psychopathy/sociopathy, conduct problems, and antisocial personality characteristics with resting, task, and reactivity measures of HR and EDA across age and stimulus categories. As a means of assessing the impact of age, categorical analyses were performed within behavior types (for effect sizes wherein age could be determined). Categorical moderation analyses were also performed within each age group, to assess the generalization of physiology–behavior relations across behavior patterns,[2] and within behavior categories, to assess moderation by stimulus valence (for effect sizes wherein valence could be determined). To be included in moderation analyses, each moderator category had to comprise three or more studies. Although I would have liked to use a more conservative criterion, I used this more liberal criterion so that I could report as much detail as possible. Thus, many of the analyses should be regarded as preliminary.

The model for categorical moderation analyses roughly follows the logic of the *F* ratio. Variability in effect sizes is computed between and within groups defined by the categories of the moderator variable. A well-specified model results in maximized between-categories variability in effect sizes and minimized within-group variability. Between-categories homogeneity is assessed by the value $Q_B$, which is interpreted relative to a chi-square distribution (*df* = number of categories minus 1). Within-category homogeneity is indexed by $Q_W$, also interpreted relative to a chi-square distribution (*df* = number of effect sizes in each category minus 1). Significance tests are computed separately for $Q_B$ and $Q_W$.

Two strategies were adopted to address bias in effect sizes due to the tendency of authors to fail to report nonsignificant findings and editors' tendencies to reject papers with many nonsignificant findings (i.e., the "file drawer" problem; Begg, 1994). First, an attempt was made to locate as many unpublished studies as possible. Second, fail-safe *N* analyses were calculated for each statistically significant relation reported. The fail-safe *N* ($N_{fs}$) indicates the number of studies yielding null findings that would be necessary to reduce the mean effect to a negligible level. $N_{fs}$ was calculated via the following equation (Hedges & Olkin, 1985):

$$N_{fs} = \frac{k(\bar{d} - d_c)}{d_c},$$

[2] Behavior type moderation was performed within age groups to avoid confounding age and behavior. For example, studies of psychopathy almost exclusively involved adult samples; conduct problems refer specifically to child behavior.

where $k$ is the number of studies in the analysis, $\bar{d}$ is the mean weighted effect size, and $d_c$ is a critical $d$ small enough to be negligible. A $d_c$ of 0.10 (or −0.10 in the case of negative associations) was adopted in the present meta-analysis. According to Rosenthal's (1979) conservative guidelines, $N_{fs}$ should exceed $5k + 10$ to effectively overcome the file drawer problem. However, the file drawer problem may be somewhat less of an issue in the present meta-analysis, in that multiple analyses were very common and a large number of nonsignificant findings were reported. Furthermore, several of the studies analyzed were unpublished and contained nonsignificant findings.

### Final Sample

The five available studies of antisocial personality characteristics (Braggio, Pishkin, Parsons, Fishkin, & Tassey, 1992; Dinn & Harris, 2000; Levander, Schalling, Lidberg, & Lidberg, 1979; Mallory, 1983; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000) were not sufficient in number to contribute meaningfully to the meta-analysis. Therefore, these studies were excluded, and the remaining 95 studies were analyzed.

## Results

### Resting HR

Study identifying information, coded qualities, and effect sizes are reported in Tables A1–A6 of the Appendix.

*Aggression.* Results of analyses involving resting HR are presented in Table 1. The mean aggregate effect size of the association between resting HR and aggression across 16 studies was −0.38, with a 95% confidence interval that excluded zero (−0.50, −0.26). Thus, lower resting HR was associated with higher levels of aggression ($N_{fs}$ = 45). According to Cohen's (1988) standards, this is a small effect. There was substantial heterogeneity in effect sizes ($p < .01$), with effect sizes ranging from −1.20 to 0.27.

There were enough studies to examine age as a moderator of the resting HR–aggression relation; studies in all age groups were compared. The omnibus test was marginally significant, $Q_B(2) = 5.63$, $p < .10$, and none of the pairwise post hoc tests reached significance. However, whereas aggression was reliably associated with low resting HR in children ($M = -0.51$; $N_{fs}$ = 25) and adults ($M = -0.30$; $N_{fs}$ = 12), the 95% confidence interval for adolescents included zero (−0.42, 0.12; $M = -0.15$). The significant negative association for studies of children met Cohen's criterion for a medium-sized effect (0.51). Effect sizes were heterogeneous only in studies of children, with effect sizes ranging from −1.20 to 0.01 ($p < .01$).

Table 1
*Categorical Analysis of Resting Heart Rate*

| Variable | $k$ | Mean $d_+$ | 95% CI for $d_+$ | $Q_W$ | $Q_B$ |
|---|---|---|---|---|---|
| Aggression | 16 | −0.38 | −0.50, −0.26 | 37.27** | |
| Age | | | | | 5.63† |
| Child | 6 | −0.51 | −0.68, −0.34 | 23.32** | |
| Adolescent | 4 | −0.15 | −0.42, 0.12 | 4.78 | |
| Adult | 6 | $-0.30_a$ | −0.53, −0.07 | 3.54 | |
| Psychopathy/sociopathy | 17 | $0.06_a$ | −0.08, 0.21 | 12.17 | |
| Conduct problems | 13 | −0.33 | −0.43, −0.23 | 17.32 | |
| Age | | | | | 0.00 |
| Child | 7 | −0.34 | −0.47, −0.22 | 10.21 | |
| Adolescent | 5 | −0.35 | −0.52, −0.17 | 5.72 | |

*Note.* $d_+$ denotes effect sizes weighted by the reciprocal of their variances. $Q_W$ tests within-category homogeneity of effect size; $Q_B$ tests between-categories homogeneity. Significant effect sizes signify rejection of the null hypothesis of homogeneous effect sizes. Mean effect sizes with matching subscripts are different from one another at $p < .01$. $k$ = number of data sets; CI = confidence interval.
† $p < .10$. ** $p < .01$.

*Psychopathy/sociopathy.* The mean effect size of the association between resting HR and psychopathy/sociopathy (among adults only) across 17 studies was 0.06, with a 95% confidence interval that included zero (−0.08, 0.21). Thus, there was no evidence of an association between HR and psychopathy/sociopathy in adults. Statistically significant heterogeneity in effect sizes was not obtained. With a weighted effect size of 1.06, the Taub (1972) study was an outlier (the next closest effect size was 0.33). However, its removal had no impact on the findings, and it was therefore retained.

*Conduct problems.* The mean aggregate effect size of the association between resting HR and conduct problems across 13 studies was −0.33, with a 95% confidence interval that excluded zero (−0.43, −0.23). Thus, lower resting HR was associated with conduct problems ($N_{fs}$ = 30), but the effect was small. Effect sizes were not significantly heterogeneous. Age could not be determined in one study of conduct problems (Garralda, Connell, & Taylor, 1991).

There were enough studies to examine age as a moderator of the resting HR–conduct problems relationship; studies of children and adolescents were compared. The mean effect size was nearly identical in child and adolescent studies, $Q_B(1) = 0.00$, *ns*, with confidence intervals that excluded zero and no significant heterogeneity within either age category.

*Behavior type moderation within age groups.* Categorical moderator analyses were performed to examine the association of effect size with behavior pattern in each age group (where possible). Mean effect sizes were compared in studies of child aggression and conduct problems. Although behavior type was not a significant moderator of effect size, $Q_B(1) = 2.53$, *ns*, the mean effect for conduct problems was small (−0.34; $N_{fs}$ = 17), and the mean effect for aggression (−0.51; $N_{fs}$ = 25) met Cohen's criterion for a medium effect. Confidence intervals excluded zero in both cases. There was heterogeneity among effect sizes in studies of child aggression ($p < .01$) but not in studies of child conduct problems.

Mean effect sizes were also compared in studies of adolescent aggression and conduct problems. No significant difference was obtained, $Q_B(1) = 1.47$, *ns*, despite the fact that resting HR was significantly associated only with conduct problems. Tests for heterogeneity were nonsignificant for each behavior construct.

In the case of adults, the mean effects of aggression (−0.30) and psychopathy/sociopathy (0.06) differed significantly, $Q_B(1) = 6.92$, $p < .01$, indicating that resting HR was more strongly associated with aggression. There was no significant heterogeneity in either category.

### Task HR

*Aggression.* Results of analyses involving task HR are presented in Table 2. The mean aggregate effect size of the association between task HR and aggression across 14 studies was −0.02,

Table 2
*Categorical Analysis of Task Heart Rate*

| Variable | *k* | Mean $d_+$ | 95% CI for $d_+$ | | $Q_W$ | $Q_B$ |
|---|---|---|---|---|---|---|
| Aggression | 14 | −0.02 | −0.15, | 0.11 | 47.34** | |
| Age | | | | | | 0.05 |
| Child | 5 | −0.001 | −0.22, | 0.22 | 22.84** | |
| Adult | 7 | −0.04 | −0.24, | 0.17 | 19.37* | |
| Stimulus valence | | | | | | 13.95** |
| Negative stimuli | 7 | −0.23 | −0.41, | −0.05 | 2.51 | |
| Nonnegative stimuli | 6 | 0.28 | 0.08, | 0.48 | 29.50** | |
| Psychopathy/sociopathy | 7 | −0.16 | −0.41, | 0.09 | 6.60 | |
| Conduct problems | 8 | −0.04 | −0.21, | 0.14 | 20.07** | |
| Age | | | | | | 1.58 |
| Child | 5 | 0.02 | −0.18, | 0.22 | 0.27 | |
| Adolescent | 3 | −0.25 | −0.64, | 0.13 | 18.22** | |
| Stimulus valence | | | | | | 2.66 |
| Negative stimuli | 5 | 0.06 | −0.15, | 0.27 | 4.17 | |
| Nonnegative stimuli | 3 | −0.27 | −0.60, | 0.06 | 13.25** | |

*Note.* $d_+$ denotes effect sizes weighted by the reciprocal of their variances. $Q_W$ tests within-category homogeneity of effect size; $Q_B$ tests between-categories homogeneity. Significant effect sizes signify rejection of the null hypothesis of homogeneous effect sizes. $k$ = number of data sets; CI = confidence interval.
* $p < .05$. ** $p < .01$.

with a 95% confidence interval that included zero (−0.15, 0.11). Thus, HR during task responding was not associated with aggression. There was substantial heterogeneity in effect sizes ($p < .01$), which ranged from −1.14 to 0.69. One study (Thomas, 1982) was an outlier (the effect size weighted by the reciprocal of its variance [$d_+$] was –1.14; the next nearest effect was −0.53). However, its removal did not affect the results, and it was therefore retained.

Mean effect sizes for task HR and aggression could be compared in the case of children and adults; no significant between-groups heterogeneity in effect sizes was evident, $Q_B(2) = 0.05$, *ns*. Statistically significant heterogeneity was found in child ($d_+$ range: −0.53 to 0.69, $p < .01$) and adult ($d_+$ range: −1.14 to 0.60, $p < .05$) studies.

*Psychopathy/sociopathy.* The mean effect size of the association between task HR and psychopathy/sociopathy (among adults only) across seven studies was −0.16, with a 95% confidence interval that included zero (−0.41, 0.09). Thus, there was no evidence of an association between task HR and psychopathy/sociopathy in adults. Statistically significant heterogeneity in effect sizes was not obtained.

*Conduct problems.* The mean aggregate effect size of the association between task HR and conduct problems across eight studies of children and adolescents was −0.04, with a 95% confidence interval that included zero (−0.21, 0.14). Thus, there was no association between task HR and conduct problems. There was substantial heterogeneity in effect sizes ($p < .01$), with effect sizes ranging from −1.67 to 0.44.

The difference in effects associated with conduct disorder in children and adolescents did not differ from chance, $Q_B(1) = 1.58$, *ns*, and both confidence intervals included zero. Effect sizes were heterogeneous only in studies of adolescent conduct problems ($d_+$ range: –1.67 to 0.40, $p < .01$), and these studies represented the entire effect size range for conduct problems.

*Behavior type moderation within age groups.* The comparison of child aggression and conduct problems failed to produce a significant result, $Q_B(1) = 0.02$, *ns*. Both confidence intervals included zero, indicating the lack of association in either case. Significant heterogeneity in effect sizes was observed for studies of aggression ($d_+$ range: −0.53 to 0.69, $p < .01$) but not for studies of conduct problems.

In the case of adults, the mean effects of aggression and psychopathy/sociopathy did not differ, $Q_B(1) = 0.53$, *ns*. There was no significant heterogeneity in effect sizes for studies of psychopathy/sociopathy. However, there was significant within-category heterogeneity of effect sizes in studies of aggression ($d_+$ range: −0.81 to 0.09, $p < .05$).

*Moderation by stimulus valence.* The dependence of effect size on stimulus valence was examined in studies of aggression and conduct problems. Valence was a significant moderator in studies of aggression, $Q_B(1) = 13.95$, $p < .01$. The mean effect for negative stimuli was negative ($M = -0.23$; $N_{fs} = 9$), whereas the mean effect for nonnegative stimuli was positive ($M = 0.28$; $N_{fs} = 11$). Both confidence intervals excluded zero. Thus, the direction of the association depended on stimulus valence. Significant heterogeneity was found only in studies of aggression that employed nonnegative stimuli ($p < .01$), with effects ranging from −1.14 to 0.69 (i.e., the highest and lowest effect sizes for task HR and aggression were found in this category).

Stimulus valence did not moderate the association between task HR and conduct problems, $Q_B(1) = 2.66$, *ns*, and could not be examined with regard to psychopathy/sociopathy (only $k = 2$ studies with nonnegative stimuli). Valence could not be determined in two studies of psychopathy/sociopathy (Pham, Philippot, & Rime, 2000; Tharp, Maltzman, Syndulko, & Ziskind, 1980) and in one study of aggression (Chiang, Schuetz, & Soyka, 2001).

### *HR Reactivity*

*Aggression.* Results of analyses involving HR reactivity are presented in Table 3. The mean aggregate effect size of the association between HR reactivity and aggression across 14 studies was 0.10, with a 95% confidence interval that included zero (−0.03, 0.22). Thus, HR reactivity was not significantly associated with aggression. There was substantial heterogeneity in effect sizes ($p < .01$), with effect sizes ranging from −0.91 to 1.40.

Mean effect sizes for HR reactivity and aggression were compared among adolescents and adults; no significant between-groups heterogeneity in effect sizes was evident, $Q_B(1) = 0.81$, *ns*. Yet, HR reactivity was related significantly to aggression in studies of adults ($M = 0.27$; $N_{fs} = 14$), and the effect was small. Statistically significant heterogeneity was also found in adult studies ($d_+$ range: −0.06 to 1.40, $p < .01$).

*Psychopathy/sociopathy.* The mean effect size of the association between HR reactivity and psychopathy/sociopathy across 14 studies was 0.06, with a 95% confidence interval that included zero (−0.11, 0.23). Thus, there was no evidence of an association between HR reactivity and psychopathy/sociopathy. Only marginally significant heterogeneity in effect sizes was found ($p < .10$), with effects ranging from −0.66 to 0.99. The only age category containing 3 or more studies was adult. The mean effect size of the association between HR reactivity and psychopathy/sociopathy across these 13 studies was 0.07, with a 95% confidence interval

Table 3
*Categorical Analysis of Heart Rate Reactivity*

| Variable | *k* | Mean $d_+$ | 95% CI for $d_+$ | | $Q_W$ | $Q_B$ |
|---|---|---|---|---|---|---|
| Aggression | 14 | 0.10 | −0.03, | 0.22 | 69.20** | |
| Age | | | | | | 0.81 |
| Adolescent | 4 | 0.10 | −0.21, | 0.42 | 5.04 | |
| Adult | 8 | 0.27 | 0.10, | 0.43 | 30.16** | |
| Stimulus valence | | | | | | 18.76** |
| Negative stimuli | 8 | 0.31 | 0.15, | 0.47 | 26.34** | |
| Nonnegative stimuli | 5 | −0.34 | −0.59, | −0.10 | 23.76** | |
| Psychopathy/sociopathy | 14 | 0.06 | −0.11, | 0.23 | 23.19† | |
| Adult | 13 | 0.07 | −0.11, | 0.25 | 23.13* | |
| Stimulus valence | | | | | | 0.03 |
| Negative stimuli | 8 | −0.04 | −0.17, | 0.26 | 9.74 | |
| Nonnegative stimuli | 5 | 0.01 | −0.27, | 0.30 | 9.85† | |
| Conduct problems | 7 | 0.20 | 0.05, | 0.36 | 19.51** | |
| Age | | | | | | 0.05 |
| Child | 3 | 0.19 | 0.002, | 0.39 | 5.95 | |
| Adolescent | 3 | 0.23 | −0.05, | 0.51 | 13.42** | |
| Stimulus valence | | | | | | 3.76† |
| Negative stimuli | 4 | 0.26 | 0.10, | 0.43 | 14.32** | |
| Nonnegative stimuli | 3 | −0.20 | −0.63, | 0.24 | 1.43 | |

*Note.* $d_+$ denotes effect sizes weighted by the reciprocal of their variances. $Q_W$ tests within-category homogeneity of effect size; $Q_B$ tests between-categories homogeneity. Significant effect sizes signify rejection of the null hypothesis of homogeneous effect sizes. *k* = number of data sets; CI = confidence interval.
† $p < .10$. * $p < .05$. ** $p < .01$.

that included zero (−0.11, 0.25). Thus, there was no evidence of an association between HR reactivity and psychopathy/sociopathy in adults. Statistically significant heterogeneity in effect sizes was found ($p < .05$), with effects representing the entire range for studies of psychopathy/sociopathy. Age could not be determined in one study of psychopathy/sociopathy (Borkovec, 1970).

*Conduct problems.* The mean aggregate effect size of the association between HR reactivity and conduct problems across seven studies of children and adolescents was 0.20, with a 95% confidence interval that excluded zero (0.05, 0.36). Thus, greater HR reactivity was associated with conduct problems ($N_{fs} = 7$). There was substantial heterogeneity in effect sizes ($p < .01$), with effect sizes ranging from –1.24 to 0.49.

Similar effects were found in studies of child and adolescent conduct disorder, $Q_B(1) = 0.05$, *ns*. However, whereas the confidence interval for adolescent studies included zero, the lower bound of the confidence interval for child studies hovered just above zero (0.002), indicating that HR reactivity is positively associated with conduct problems in children ($N_{fs} = 3$). Significant effect size heterogeneity was found only in adolescent studies ($p < .01$), with effect sizes in this category comprising the entire range of studies of conduct problems. Age could not be determined in one study (Garralda, Connell, & Taylor, 1990).

*Behavior type moderation within age groups.* In the case of adolescents, the mean effects for aggression and conduct problems did not differ significantly, $Q_B(1) = 0.35$, *ns*. Significant within-category heterogeneity in effect sizes was found in studies of conduct problems ($p < .01$) but not in studies of aggression.

The difference in mean effects for aggression and psychopathy/sociopathy in studies of adults did not reach statistical significance, $Q_B(1) = 2.55$, *ns*; only aggression was significantly associated with HR reactivity (95% confidence interval: 0.10, 0.43). Significant heterogeneity in effect sizes was found in both categories (*p*s < .05 and .01 for psychopathy/sociopathy and aggression, respectively).

*Moderation by stimulus valence.* The dependence of effect size on stimulus valence was examined in studies of aggression, conduct problems, and psychopathy/sociopathy. Valence was a significant moderator in studies of aggression, $Q_B(1) = 18.76$, $p < .01$. In studies of aggression, positive effect sizes were associated with negative stimuli ($M = 0.31$; $N_{fs} = 17$), whereas negative effect sizes were associated with nonnegative stimuli ($M = -0.34$; $N_{fs} = 12$). Significant effect size heterogeneity was evident in both categories (*p*s < .01). Effect sizes ranged from −0.06 to 1.40 for negative stimuli and from −0.91 to 0.84 for nonnegative stimuli.

A similar sign reversal was obtained in studies of conduct problems. However, the interaction of valence and HR reactivity in predicting conduct problems was only marginally significant, $Q_B(1) = 3.76$, $p < .10$. Mean effects were 0.26 ($N_{fs} = 6$) for negative stimuli and −0.20 for nonnegative stimuli. However, the confidence interval for studies that employed nonnegative stimuli included zero. Significant effect size heterogeneity was found only in studies that employed negative stimuli ($p < .01$), with effects that ranged from −1.24 to 0.49.

Stimulus valence did not moderate the association between HR reactivity and psychopathy/sociopathy, $Q_B(1) = 0.03$, *ns*. Stimulus valence could not be determined in one study of psychopathy/sociopathy (Schachter & Latane, 1964) and in one study of aggression (Smith & Gallo, 1999).

## *Resting EDA*

*Aggression.* Results of analyses involving resting EDA are presented in Table 4. Four studies of aggression met criteria for analysis. The mean aggregate effect size of the association between resting EDA and aggression across these studies was 0.10, with a 95% confidence interval that included zero (−0.14, 0.34). Thus, resting EDA was not significantly associated with aggression. Effect sizes were homogeneous. The only age category containing three or more studies was adult. The mean aggregate

Table 4
*Categorical Analysis of Resting Electrodermal Activity*

| Variable | *k* | Mean $d_+$ | 95% CI for $d_+$ | | $Q_W$ | $Q_B$ |
|---|---|---|---|---|---|---|
| Aggression | 4 | 0.10 | −0.14, | 0.34 | 0.08 | |
| Adult | 3 | 0.08 | −0.27, | 0.44 | 0.07 | |
| Psychopathy/sociopathy | 18 | −0.30 | −0.46, | −0.15 | 27.31† | |
| Adult | 14 | −0.29 | −0.46, | −0.12 | 23.10† | |
| Conduct problems | 11 | −0.15 | −0.28, | 0.001 | 23.81** | |
| Age | | | | | | 2.81† |
| Child | 8 | −0.30 | −0.49, | −0.07 | 16.72* | |
| Adolescent | 3 | −0.03 | −0.23, | 0.17 | 4.28 | |

*Note.* $d_+$ denotes effect sizes weighted by the reciprocal of their variances. $Q_W$ tests within-category homogeneity of effect size; $Q_B$ tests between-categories homogeneity. Significant effect sizes signify rejection of the null hypothesis of homogeneous effect sizes. *k* = number of data sets; CI = confidence interval.
† $p < .10$. * $p < .05$. ** $p < .01$.

effect size of the association between resting EDA and aggression across these three studies was 0.08, with a 95% confidence interval that included zero (−0.27, 0.44) and no significant variation in effect sizes, further confirming the lack of a reliable relation between resting EDA and aggression.

*Psychopathy/sociopathy.* The mean effect size of the association between resting EDA and psychopathy/sociopathy across 18 studies was −0.30 ($N_{fs}$ = 36), with a 95% confidence interval that excluded zero (−0.46, −0.15). Thus, lower resting EDA was significantly associated with psychopathy/sociopathy, but the effect was small. Marginally significant within-category heterogeneity of effect sizes was found ($p < .10$), with effects that ranged from –1.87 to 0.57. The only age category containing 3 or more studies was adult. The mean effect of the association between resting EDA and psychopathy/sociopathy in these 14 studies was −0.29 ($N_{fs}$ = 27), with a 95% confidence interval that excluded zero (−0.46, −0.12) and marginal within-category heterogeneity of effect sizes ($p < .10$), representing the entire range for studies of psychopathy/sociopathy. This did not differ from the overall pattern just reported. Age could not be determined in 2 studies of psychopathy/sociopathy (Fox & Lippert, 1963; Hare, 1965b). With an effect size of –1.87, the Hinton et al. (1980) study was a significant outlier (the nearest effect size was –1.17). However, its removal did not affect the findings reported here, and it was therefore retained.

*Conduct problems.* The mean aggregate effect size of the association between resting EDA and conduct problems across 11 studies of children and adolescents was −0.15, with a 95% confidence interval that included zero (−0.28, 0.001). Thus, there was no reliable association between resting EDA and conduct problems. There was substantial heterogeneity in effect sizes ($p < .01$), with effect sizes ranging from −1.02 to 0.89.

There were enough studies to examine age as a moderator of the resting EDA–conduct problems relationship; studies of children and adolescents were compared. The mean effect size was lower in child ($M$ = −0.30; $N_{fs}$ = 16) than in adolescent ($M$ = −0.03) studies, $Q_B(1) = 2.81$, $p < .10$, although this difference only approached statistical significance. Furthermore, the 95% confidence interval excluded zero (−0.49, −0.07) in studies of children but not adolescents, indicating that low resting EDA is reliably associated with conduct problems only in the case of children. Significant effect size heterogeneity was found only in studies of children ($p < .05$), with these effects representing the entire range for conduct problems.

The effect size contributed by the investigation of Garralda, Connell, and Taylor (1989) to studies of conduct problems in children was an outlier ($d_+$ = 0.89; the next highest effect size was 0.21). However, this study was retained because it did not substantially leverage the results.

*Behavior type moderation within age groups.* Mean effect sizes were compared in studies of adult psychopathy/sociopathy and aggression. The difference in mean effects for aggression ($M$ = 0.08) and psychopathy/sociopathy ($M$ = −0.29) fell just short of statistical significance, $Q_B(1) = 3.54$, $p < .10$; only psychopathy/sociopathy was associated significantly with resting EDA (i.e., its confidence interval excluded zero). Marginal within-category effect size heterogeneity was found in studies of psychopathy/sociopathy ($p < .10$) but not in studies of aggression.

## *Task EDA*

*Aggression.* Results of analyses involving task EDA are presented in Table 5. The mean aggregate effect size of the association between task EDA and aggression across four studies was 0.07, with a 95% confidence interval that included zero (−0.18, 0.32). Thus, task EDA was not associated with aggression. Effect sizes were significantly heterogeneous ($d_+$ range: −0.18 to 1.77, $p < .05$). The only age category containing three or more studies was adult. The mean aggregate effect size of the association between task EDA and aggression across these three studies was 0.31, with a 95% confidence interval that included zero (−0.04, 0.65). Thus, EDA was not associated with adult aggression. Effect sizes were homogeneous. One study (Wolfe, Fairbank, Kelly, & Bradlyn, 1983) produced an outlier ($d_+$ = 1.77; the next nearest effect size was 0.42). Its removal did not affect the results, and it was therefore retained.

*Psychopathy/sociopathy.* The mean effect size of the association between task EDA and psychopathy/sociopathy across 28 studies was −0.25, with a 95% confidence interval that excluded zero (−0.34, −0.15). Thus, lower task EDA was associated with psychopathy/sociopathy, but the effect was small ($N_{fs}$ = 42). Statistically significant heterogeneity in effect sizes was found ($p < .01$) with effects ranging from −1.12 to 0.57.

Age was a significant moderator; studies of adult psychopathy/sociopathy yielded a significant and larger mean effect ($M$ = −0.43; $N_{fs}$ = 69), whereas studies of adolescents yielded a nonsignificant and smaller mean effect ($M$ = −0.07), $Q_B(1) = 12.24$, $p < .01$. Effect sizes were marginally heterogeneous only in adolescents ($d_+$ range: −0.62 to 0.06, $p < .10$). Age could not be determined in two studies of psychopathy/sociopathy (Borkovec, 1970; Hare, 1965b).

Table 5
*Categorical Analysis of Task Electrodermal Activity*

| Variable | $k$ | Mean $d_+$ | 95% CI for $d_+$ | $Q_W$ | $Q_B$ |
|---|---|---|---|---|---|
| Aggression | 4 | 0.07 | −0.18, 0.32 | 8.38* | |
| Adult | 3 | $0.31_a$ | −0.04, 0.65 | 4.62 | |
| Psychopathy/sociopathy | 28 | −0.25 | −0.34, −0.15 | 48.59* | |
| Age | | | | | 12.24** |
| Adolescent | 4 | −0.07 | −0.21, 0.06 | 8.78† | |
| Adult | 21 | $-0.43_a$ | −0.57, −0.28 | 26.26 | |
| Stimulus valence | | | | | 10.98** |
| Negative stimuli | 13 | −0.47 | −0.65, −0.29 | 16.45 | |
| Nonnegative stimuli | 13 | −0.11 | −0.23, 0.01 | 15.89 | |
| Conduct problems | 14 | −0.23 | −0.35, −0.10 | 18.81 | |
| Age | | | | | 6.62* |
| Child | 8 | −0.46 | −0.67, −0.24 | 6.03 | |
| Adolescent | 6 | −0.10 | −0.26, 0.05 | 6.16 | |
| Stimulus valence | | | | | 0.18 |
| Negative stimuli | 4 | −0.07 | −0.30, 0.16 | 6.27 | |
| Nonnegative stimuli | 9 | −0.27 | −0.44, −0.12 | 9.50 | |

*Note.* $d_+$ denotes effect sizes weighted by the reciprocal of their variances. $Q_W$ tests within-category homogeneity of effect size; $Q_B$ tests between-categories homogeneity. Significant effect sizes signify rejection of the null hypothesis of homogeneous effect sizes. Mean effect sizes with matching subscripts are different from one another at $p < .01$. $k$ = number of data sets; CI = confidence interval.
† $p < .10$. * $p < .05$. ** $p < .01$.

*Conduct problems.* The mean aggregate effect size of the association between task EDA and conduct problems across 14 studies of children and adolescents was −0.23, with a 95% confidence interval that excluded zero (−0.35, −0.10). Thus, lower task EDA was associated with conduct problems ($N_{fs}$ = 18). There was no evidence of heterogeneity in effect sizes after removal of an extreme outlier (Mangina, Beuzeron-Mangina, & Grizenko, 2000; $d_+$ = −6.71).

The difference in effects associated with conduct problems in children ($M$ = −0.46; $N_{fs}$ = 29) and adolescents ($M$ = −0.10) was statistically significant, $Q_B(1)$ = 6.62, $p < .05$. The association of conduct problems with low task EDA was reliable only with respect to children (95% confidence interval: −0.67, −0.24). There was no evidence of effect size heterogeneity in either age group.

*Behavior type moderation within age groups.* In the case of adolescents, the mean effects for psychopathy/sociopathy and conduct problems did not differ significantly, $Q_B(1)$ = 0.08, *ns.* Marginally significant within-category heterogeneity in effect sizes was found only in studies of psychopathy/sociopathy ($d_+$ range: −0.62 to 0.06, $p < .10$).

Among adults, effect size was moderated by behavior type, $Q_B(1)$ = 15.06, $p < .01$. A significant negative relation was found for the 21 studies of psychopathy/sociopathy ($M$ = −0.43; $N_{fs}$ = 69); this was not the case for aggression, in which a nonsignificant positive mean effect was obtained ($M$ = 0.31). There was no evidence of effect size heterogeneity in either group.

*Moderation by stimulus valence.* Dependence of effect size on stimulus valence was examined in studies of psychopathy/sociopathy and conduct problems. Valence was a significant moderator only in studies of psychopathy/sociopathy, $Q_B(1)$ = 10.98, $p < .01$. The mean effect for negative stimuli ($M$ = −0.47; $N_{fs}$ = 48) was stronger than the mean effect for nonnegative stimuli ($M$ = −0.11). Furthermore, the task EDA–psychopathy/sociopathy association was reliable only when negative stimuli were employed (95% confidence interval: −0.65, −0.29). There was no evidence of effect size heterogeneity for stimuli of either valence category.

Valence was not a significant moderator of effect sizes in studies of conduct problems, $Q_B(1)$ = 0.18, *ns*; a reliable association was obtained only for studies that employed nonnegative stimuli (mean $d_+$ = −0.27; 95% confidence interval: −0.44, −0.12; $N_{fs}$ = 15). There was no evidence of effect size heterogeneity for either valence category. Stimulus valence could not be determined in two studies of psychopathy/sociopathy (Hare, 1978b; Herpertz, Werth, et al., 2001) and in one study of conduct problems (Harden, Pihl, Vitaro, Gendreau, & Tremblay, 1995).

## *Electrodermal Reactivity*

*Aggression.* Results of analyses involving EDA reactivity are presented in Table 6. Comparatively fewer studies ($k$ = 20) reported EDA reactivity. All but two of these were studies of adults. The one study of conduct problems was not included in EDA reactivity analyses. The aggregate association across the five studies of aggression was small ($M$ = −0.07), with a 95% confidence interval that included zero (−0.24, 0.10). Thus, there was no aggregate association between EDA reactivity and aggression. Significant effect size heterogeneity was found ($d_+$ range: −0.72 to 0.91, $p < .01$). The only age class with 3 or more studies was adult. The aggregate association across these three studies was small ($M$ = 0.34; $N_{fs}$ = 7), but its 95% confidence interval excluded zero (0.10, 0.59). Thus, adult aggression was associated with greater EDA reactivity, with no significant heterogeneity in effect sizes.

Table 6
*Categorical Analysis of Electrodermal Reactivity*

| Variable | $k$ | Mean $d_+$ | 95% for $d_+$ | $Q_W$ |
|---|---|---|---|---|
| Aggression | 5 | −0.07 | −0.24, 0.10 | 29.62** |
| Adult | 3 | $0.34_a$ | 0.10, 0.59 | 2.89 |
| Psychopathy/sociopathy | 14 | −0.31 | −0.48, −0.13 | 29.30** |
| Adult | 13 | $-0.33_a$ | −0.52, −0.15 | 28.23** |

*Note.* $d_+$ denotes effect sizes weighted by the reciprocal of their variances. $Q_W$ tests within-category homogeneity of effect size. Significant effect sizes signify rejection of the null hypothesis of homogeneous effect sizes. Mean effect sizes with matching subscripts are different from one another at $p < .01$. $k$ = number of data sets; CI = confidence interval.
** $p < .01$.

*Psychopathy/sociopathy.* Across 14 studies, EDA reactivity was negatively associated with psychopathy/sociopathy ($M$ = −0.31; $N_{fs}$ = 29), with a 95% confidence interval that excluded zero (−0.48, −0.13). Effect sizes varied significantly ($p < .01$), from −1.35 to 0.82. The only age class with 3 or more studies was adult. Across these 13 studies, EDA reactivity was significantly negatively associated with psychopathy/sociopathy ($M$ = −0.33; $N_{fs}$ = 30), with a 95% confidence interval that excluded zero (−0.52, −0.15). Effect sizes varied significantly ($p < .01$), representing the entire range for psychopathy/sociopathy.

*Behavior type moderation within age groups.* Studies of adult psychopathy/sociopathy and aggression yielded nearly identical effect sizes with opposite signs (−0.33 and 0.34, respectively). This difference was statistically significant, $Q_B(1)$ = 18.95, $p < .01$.

*Moderation by stimulus valence.* Because the sample consisted almost exclusively of effects based on the use of negatively valenced stimuli, moderation by stimulus valence could not be evaluated.

## Discussion

The present meta-analytic review evaluated the associations of HR and EDA with aggressive, psychopathic, and conduct-disordered behavior across 95 studies.[3] Analyses were conducted to examine moderation of effect size by behavior type and age, as well as valence of experimental stimuli. Results suggest that aggression, conduct problems, and psychopathy/sociopathy are reliably though modestly associated with HR and EDA in many cases. However, results were mixed and revealed a complex constellation of interactive effects. Many of the findings are summarized in Table 7.

With rare exceptions, the effect sizes corresponding to each of the significant findings reported herein were small according to Cohen's (1988) standards. Furthermore, fail-safe $N$ analyses indi-

[3] Unfortunately, there were not enough studies of antisocial personality characteristics to meaningfully contribute to the meta-analysis.

cated that there might be a file drawer problem. A relatively small group of studies with very small or opposite-signed effect sizes could render many of the aggregate findings nonsignificant. Thus, due caution should be exercised in interpreting the present results. There were generally more studies of psychopathy/sociopathy available for analysis, as compared with studies of aggression and conduct problems. Therefore, with due acknowledgment of the file drawer problem, confidence in findings involving psychopathy/sociopathy is greater.

The most simple and compelling results were obtained with respect to EDA and psychopathy/sociopathy, studied primarily in adults. Psychopathy/sociopathy was associated with lower EDA at rest and during tasks and as a change from baseline. Consistent with predictions, the negative association between psychopathy/sociopathy and EDA during tasks was evident only for stimuli of negative valence; this interaction achieved statistical significance.

A similar pattern was obtained for child conduct problems, which were associated with attenuated EDA in the absence of stimulation. As was psychopathy/sociopathy, conduct problems were associated with low task EDA. However, this was found only in the case of studies of conduct problems that involved *nonnegative* stimuli. This finding was the opposite of what was predicted and was in contrast to the findings for psychopathy/sociopathy. The attenuated EDA of psychopathic individuals relative to controls was evident only under conditions of exposure to negatively valenced stimuli. Thus, despite the considerable overlap in these two constructs and the status of childhood conduct problems as a risk factor for antisocial adult behavior (Loeber, 1982), the present meta-analytic results suggest that the nature of underlying psychopathology may differ between children with conduct problems and adults with psychopathy. The latter are thought to almost invariably exhibit conduct problems as children (Lynam, 1996). However, the majority of children with conduct problems will not become psychopathic or even antisocial adults (Robins, 1978); thus, if a group of "fledgling psychopaths" exists (with autonomic characteristics of psychopathic adults), their contribution to the relations between task EDA and conduct problems would be washed out by the majority.

A very different pattern of results was obtained with respect to EDA and aggression. Although aggression was not associated with either resting or task EDA in any age group, adult aggression was positively associated with EDA reactivity, the opposite of findings for psychopathy/sociopathy. Considering the EDA findings as a whole, and in spite of overlaps in the behavior constructs being studied (e.g., violations of personal rights, laws, and social norms are common to all of them), a degree of behavioral specificity was evidenced in this aspect of autonomic functioning.

In contrast to the consistent relation between EDA and psychopathy/sociopathy, psychopathy/sociopathy was not associated with HR in any analysis, irrespective of the valence of experimental stimuli. On the other hand, HR proved to be a reliable correlate of conduct problems and aggression. Conduct problems in children and adolescents were associated with lower resting HR. Conduct problems were also associated with greater HR reactivity, but only among children. Because of the small effect size difference between studies of children and adolescents, however, future meta-analyses may indeed reveal a positive relation between HR reactivity and conduct problems in adolescents. Consistent with the valence hypothesis, reliable associations between conduct problems and HR reactivity were found only for negative stimuli. The association of conduct problems with HR, combined with a lack of any HR findings for psychopathy/sociopathy, further highlights the distinctiveness of the psychopathologies constituting these two behavior patterns.

As with conduct problems, both child and adult aggression were associated with low resting HR, but only adult aggression was marked by greater HR reactivity. Although no aggregate association between task HR and aggression was found, negative task HR–aggression relations emerged for stimuli of negative valence, whereas positive relations were obtained for nonnegative stimuli. The opposite pattern was found with respect to HR reactivity.

Table 7
*Summary of Main Effects*

| Category | Resting HR | Task HR | HR reactivity | Resting EDA | Task EDA | EDA reactivity |
|---|---|---|---|---|---|---|
| Aggression | ↓ | — | — | — | — | — |
| Child | ↓ | — | ? | ? | ? | ? |
| Adolescent | ? | — | — | ? | ? | ? |
| Adult | ↓ | — | ↑ | — | — | ↑ |
| Negative stimuli | | ↓ | ↑ | | ? | ? |
| Nonnegative stimuli | | ↑ | ↓ | | ? | ? |
| Psychopathy/sociopathy | — | — | — | ↓ | ↓ | ↓ |
| Child | ? | ? | ? | ? | ? | ? |
| Adolescent | ? | ? | ? | ? | — | ? |
| Adult | — | — | — | ↓ | ↓ | ↓ |
| Negative stimuli | | ? | — | | ↓ | ? |
| Nonnegative stimuli | | ? | — | | — | ? |
| Conduct problems | ↓ | — | ↑ | — | ↓ | ? |
| Child | ↓ | — | ↑ | ↓ | ↓ | ? |
| Adolescent | ↓ | — | — | — | — | ? |
| Negative stimuli | | — | ↑ | | — | ? |
| Nonnegative stimuli | | — | — | | ↓ | ? |

*Note.* ↑ denotes a significant (i.e., 95% confidence interval excludes zero) positive main effect; ↓ denotes a significant negative main effect. Dashes denote nonsignificant main effects (i.e., 95% confidence interval includes zero). Question marks indicate insufficient number of studies for analysis. HR = heart rate; EDA = electrodermal activity.

## *Clinical Implications*

Limitations notwithstanding, the results of this meta-analysis may have clinical implications. Iacono (1991) pointed out the value of psychophysiological assessment in assisting with differential diagnoses and identification of different forms of psychopathology. From this perspective, the autonomic differentiation of adult aggression and psychopathy/sociopathy provides the clearest information. Broadly, aggression appears to be more tightly tied to HR than to EDA, with the converse being true of psychopathy/sociopathy. Differences between mean effects were significant in only half of the contrasts reported. However, in no case were aggression and psychopathy/sociopathy reliably associated with either EDA or HR in the same direction. This set of findings further accentuates the distinctiveness of these two constructs.

In contrast to the relative autonomic distinctiveness of aggression and psychopathy/sociopathy in adults, there was no evidence of differing physiology–behavior relations between conduct prob-

lems and aggression in children and adolescents. The strongest findings concern conduct problems in children. Children with conduct problems had autonomic patterns in common with adult aggressors (low resting HR; also found with regard to child aggression) and psychopathic/sociopathic individuals (low resting EDA). This pattern is consistent with the theoretical and empirical literature indicating that children with conduct problems are at risk for both adult aggression and psychopathy/sociopathy (e.g., Lynam, 1996; Moffitt et al., 2002). HR and EDA appear to mark nonspecific risk, with other factors determining which developmental trajectory children follow.

To the extent that autonomic measures reflect the presence of specific symptoms or dimensional attributes that underlie a disorder (Iacono, 1991), the autonomic variables identified here may also be useful indices of change. Where psychopathology is concerned, treatment outcome studies most often assess changes in behavioral indicators only. However, if part of the pathology of antisocial spectrum behavior is inherently biological—and the results of the present meta-analysis support this contention—behavioral indicators cannot capture all of the relevant aspects of change. Changes in autonomic measures may ultimately prove to be most feasible with children (e.g., Raine et al., 2001), given their relative neural plasticity.

### *Contributors to Variability in Effect Sizes*

The present analyses of effect size heterogeneity have several empirical implications. Researchers of antisocial spectrum behavior may in many cases assume a degree of specificity in physiology–behavior relations, these being tied to theory. For example, Lykken (1957) hypothesized that psychopathic individuals have deficient fear conditioning. This was tested by measuring EDA responses to a conditioned stimulus that had previously been associated with shocks. In contrast, Hare (1968) hypothesized that psychopathic individuals are deficient in orienting. This was tested by measuring EDA in response to novel tones. However, this degree of specificity may not always exist. Despite the fact that the antisocial constructs were measured differently, different stimuli were employed, and different subclasses of physiological measures were used, effect sizes did not significantly vary in many cases. Significant heterogeneity was found in 42.86% of the cases in which it was tested (30.43% for EDA, 51.52% for HR). In the cases in which significant aggregate effects were accompanied by nonsignificant heterogeneity in effect sizes, a degree of generality is suggested. That is, these associations were potentially insensitive to construct measurement, stimuli used, and subtype of physiological measure. Also, in cases in which aggregate effects were not reliable, with nonsignificant heterogeneity in effect sizes, methodological differences could not have caused false nonsignificant findings.

Of course, in instances in which heterogeneity was found, it could have been due to any one of these factors or to other methodological and substantive differences among studies. The presence of such interactive effects could not be evaluated here because of the relatively low number of studies included in each subanalysis; however, single studies or future quantitative reviews might address this issue.

One methodological contributor to unexplained variability in effect sizes may have been construct heterogeneity within coding definitions. For example, studies by Hare and colleagues, who used a consistent operationalization of psychopathy over several studies of psychopathy and EDA reactivity, enjoyed a notably high replication rate relative to the pool of studies contributed by other investigators, within which varied psychopathy assessment methods were used. One of the main themes in this article is that physiology–behavior relations depend on which behavior construct is being studied. Therefore, heterogeneity in behavior operationalizations seems to be an important candidate as a contributor to variability in effect sizes. More studies will be required to make finer distinctions in future quantitative reviews.

Within behavior patterns, the mixed degree of clinical deviation in participants of the studies included in the present meta-analysis may also have contributed to effect size heterogeneity. Physiology–behavior relations may apply only to more "clinical" groups, such as psychopathic individuals low in anxiety (vs. all psychopathic individuals) or those in the upper ranges of aggression (vs. simply higher than average). Single studies and future meta-analyses could address this concern. For example, Babcock et al. (2004) found that the relation between HR reactivity and psychopathy differed among low- and high-level domestically violent men.

Gender is another potentially important moderator of the relations investigated herein, and it may have contributed to effect size heterogeneity. Although the majority of studies included in the present meta-analysis were conducted exclusively with male participants, female participants were included in several of the studies, probably more so in studies of children and adolescents. Most of these studies, however, did not report separate findings by gender. This prevented examination of gender interactive effects. Such questions are important, and they should be addressed in future studies and, eventually, meta-analyses.

With some notable exceptions (e.g., Raine, Brennan, Farrington, & Mednick, 1997; Raine & Venables, 1981), the field has focused primarily on main effects. Identification of methodologically and theoretically warranted moderators will continue to advance this area of inquiry.

### *Further Implications for Research*

Cross-sectional work with multiple age cohorts would also be helpful to resolve issues pertaining to generalization of findings across development. Longitudinal studies would help in this regard as well, with both aggressive/antisocial behavior and autonomic patterns measured contemporaneously at all time points. Longitudinal studies have begun to yield findings similar to the cross-sectional ones reported here. For example, Raine and his colleagues have found that low resting HR at the age of 3 years predicts aggression at 11 years (Raine, Venables, & Mednick, 1997). This group has also found evidence that poor conditioning (judged by electrodermal responses) in adolescence is a risk factor for adult crime (Raine, Venables, & Williams, 1996). More prospective longitudinal studies will be necessary to address important, as-of-yet-unresolved developmental problems, such as identification of autonomic variables that predict the transition from childhood conduct problems to adult psychopathy.

The impact of stimulus valence on physiology–behavior relations also merits further study. Broad support was not found for the hypothesis that stimuli of negative valence would produce stronger physiology–behavior relations. In fact, the only clear support for the valence hypothesis was found with respect to task EDA and psychopathy/sociopathy. Significant valence moderation was found in other cases. However, the results were mixed, with frequent sign reversals. Although these results did not support a priori hypotheses in most cases, the impact of stimuli valence is clearly worthy of attention. To my knowledge, there is no existing theory that predicts such patterns. Thus, revisions to theory may be in order. On the other hand, it is possible that valence was confounded with design characteristics that were ultimately responsible for the observed patterns. This possibility could perhaps best be tested in future studies and should be clarified before theory modification.

In addition to paying more attention to moderators in future quantitative reviews, measuring multiple forms of antisocial and aggressive behavior within studies would allow for comparisons of autonomic patterns. The Babcock et al. (2004) study is an excellent example of this approach. The present results suggest that, in many cases, autonomic patterns differ according to the behavior under consideration; however, the comorbidity of different forms of antisocial spectrum behavior decreases clarity of interpretation. For example, it is impossible to know to what degree aggressive samples were populated with psychopathic individuals or individuals with multiple antisocial personality characteristics.

Another implication for future empirical work is tied to enervation of the heart. As reviewed in the introduction, HR is controlled by both the SNS and the PNS. However, the PNS ordinarily exerts a stronger influence on HR—via the vagus nerve—than the SNS (Porges, 2001). Because aggression was found to be associated with HR (low resting HR and high HR reactivity), it follows that aggression may be tied to vagal functioning, particularly that which originates in the nucleus ambiguus of the medulla, an area tied to social affective behavior (Beauchaine, 2001; Porges, 1995, 2001). At present, however, the literature assessing such relations (e.g., Umhau et al., 2002) is too limited to allow any firm conclusions. Investigations of vagal functioning and antisocial spectrum behavior may hold a great deal of promise for advancing our understanding of these forms of psychopathology, particularly when studied in the context of other aspects of autonomic functioning (see Beauchaine, 2001).

Finally, there is a critical need for studies focusing on psychophysiological correlates of antisocial personality characteristics. I was surprised at the relative lack of such studies, too few to have made a meaningful contribution to the present meta-analysis. It is ironic that the most commonly invoked construct in this area of inquiry—"antisocial"—has apparently been studied the least.

### *Conclusion*

This meta-analysis highlights the significance of autonomic markers of antisocial spectrum psychopathology. Although aggregate relations were small, HR and EDA measures proved to be reliable correlates of aggression, psychopathy/sociopathy, and conduct problems in enough cases to suggest that they be taken seriously. At the same time, the present meta-analysis underscores the complexity of research in this area. This too is a useful contribution inasmuch as it points to several open empirical issues, the resolution of which will doubtlessly advance our knowledge of this most socially costly set of behaviors.

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# Appendix

## Characteristics and Effects Sizes for Studies in the Meta-Analysis

Table A1
*Coded Qualities and Weighted Effect Sizes of Studies of Resting Heart Rate*

| Study | Age | Behavior pattern | $N_e$ | $N_c$ | $d_+$ |
|---|---|---|---|---|---|
| Arnett et al. (1993) | Adult | Psychopathy/sociopathy | 31 | 32 | 0.00 |
| Babcock et al. (2004) | Adult | Aggression | 72 | 30 | −0.32 |
| Blackburn (1979) | Adult | Psychopathy/sociopathy | 18 | 14 | −0.23 |
| Chiang et al. (2001) | Adult | Aggression | 14 | 16 | −0.79 |
| Cohn (1995)[a] | Adolescent | Aggression | 21 | 21 | −0.31 |
| Cohn (1995)[a] | Adolescent | Aggression | 19 | 19 | 0.19 |
| Cohn (1995)[a] | Adolescent | Aggression | 21 | 21 | 0.27 |
| Davies & Maliphant (1971) | Adolescent | Conduct problems | 14 | 14 | −0.73 |
| Delamater & Lahey (1983) | Child | Conduct problems | 19 | 17 | −0.58 |
| Frodi & Lamb (1980) | Adult | Aggression | 14 | 14 | 0.00 |
| Garralda et al. (1989) | Child | Conduct problems | 7 | 8 | −1.02 |
| Garralda et al. (1991) | Uncodable | Conduct problems | 25 | 25 | 0.00 |
| Gerra et al. (1997) | Adult | Aggression | 15 | 15 | −0.08 |
| Haldeman (1973) | Adult | Psychopathy/sociopathy | 30 | 10 | 0.08 |
| Hare & Craigen (1974) | Adult | Psychopathy/sociopathy | 17 | 17 | 0.00 |
| Hare (1968) | Adult | Psychopathy/sociopathy | 39 | 12 | 1.00 |
| Hare (1982) | Adult | Psychopathy/sociopathy | 16 | 15 | 0.00 |
| Hare et al. (1978) | Adult | Psychopathy/sociopathy | 24 | 40 | 0.00 |
| Herpertz, Wenning, et al. (2001) | Child | Conduct problems | 26 | 21 | −0.47 |
| House & Milligan (1976) | Adult | Psychopathy/sociopathy | 32 | 32 | 0.00 |
| Ingersoll (1977) | Adult | Psychopathy/sociopathy | 9 | 11 | −0.32 |
| Kindlon et al. (1995)[a] | Child | Aggression | 51 | 0 | −0.52 |
| Kindlon et al. (1995)[a] | Adolescent | Aggression | 44 | 0 | −0.42 |
| Maliphant et al. (1990) | Adolescent | Conduct problems | 12 | 20 | −1.11 |
| Marchel (1993) | Adolescent | Conduct problems | 40 | 40 | −0.16 |
| McBurnett (1989) | Child | Conduct problems | 15 | 37 | −0.18 |
| McLaughlin (1995) | Child | Aggression | 29 | 0 | −0.14 |
| McNamara & Ballard (1999)[a] | Adult | Psychopathy/sociopathy | 49 | 0 | 0.02 |
| McNamara & Ballard (1999)[a] | Adult | Psychopathy/sociopathy | 47 | 0 | 0.26 |
| Milner (1976) | Adult | Psychopathy/sociopathy | 29 | 0 | 0.33 |
| Ogloff & Wong (1990) | Adult | Psychopathy/sociopathy | 16 | 16 | −0.29 |
| Pham et al. (2000) | Adult | Psychopathy/sociopathy | 14 | 15 | −0.32 |
| Pitts (1993) | Child | Aggression | 65 | 38 | −0.75 |
| Raine & Jones (1987) | Child | Aggression | 40 | 0 | −0.64 |
| Raine & Venables (1984a) | Adolescent | Conduct problems | 101 | 0 | −0.28 |
| Rogeness et al. (1990)[a] | Child | Conduct problems | 374 | 0 | −0.39 |
| Rogeness et al. (1990)[a] | Adolescent | Conduct problems | 61 | 145 | −0.34 |
| Scarpa et al. (2000) | Adult | Aggression | 54 | 0 | −0.36 |
| Schachter & Latane (1964)[a] | Adult | Psychopathy/sociopathy | 15 | 15 | 0.00 |
| Schachter & Latane (1964)[a] | Adult | Psychopathy/sociopathy | 9 | 9 | 0.00 |
| Smithmyer (2001) | Child | Aggression | 67 | 0 | 0.01 |
| Taub (1972) | Adult | Psychopathy/sociopathy | 15 | 15 | 1.06 |
| Tucker (1991) | Child | Conduct problems | 32 | 44 | −0.25 |
| van Goozen et al. (1998) | Child | Aggression | 52 | 0 | −1.20 |
| Wolfe et al. (1983) | Adult | Aggression | 7 | 7 | 0.18 |
| Zahn & Kruesi (1993) | Child | Conduct problems | 27 | 31 | 0.37 |

*Note.* *N* for studies that reported correlations or other single-group analyses is reported in the $N_e$ column. $N_e$ = number of participants in experimental (i.e., aggressive, psychopathic/sociopathic, conduct problems) group; $N_c$ = number of participants in control group; $d_+$ = effect size weighted by reciprocal of variance.
[a] Multiple independent samples from a single study.

(*Appendix continues*)

Table A2
*Coded Qualities and Weighted Effect Sizes of Studies of Task Heart Rate*

| Study | Age | Behavior pattern | Stimulus valence | $N_e$ | $N_c$ | $d_+$ |
|---|---|---|---|---|---|---|
| Babcock et al. (2004) | Adult | Aggression | Negative | 72 | 30 | −0.32 |
| Chiang et al. (2001) | Adult | Aggression | Uncodable | 14 | 16 | −0.45 |
| Davies & Maliphant (1971) | Adolescent | Conduct problems | Negative | 14 | 14 | −0.47 |
| Edguer & Janisse (1994) | Adult | Aggression | Nonnegative | 50 | 0 | 0.60 |
| Eisenberg et al. (1996) | Child | Conduct problems | Negative | 92 | 0 | 0.00 |
| Friedrich et al. (1985) | Adult | Aggression | Negative | 14 | 15 | 0.00 |
| Haldeman (1973) | Adult | Psychopathy/sociopathy | Negative | 10 | 10 | −0.06 |
| Harden & Pihl (1995) | Adolescent | Conduct problems | Negative | 28 | 0 | 0.44 |
| Harden et al. (1995) | Child | Conduct problems | Negative | 18 | 15 | 0.19 |
| House & Milligan (1973) | Adult | Psychopathy/sociopathy | Nonnegative | 32 | 32 | 0.00 |
| Ingersoll (1977) | Adult | Psychopathy/sociopathy | Nonnegative | 9 | 11 | −0.80 |
| Jacobson et al. (1994) | Adult | Aggression | Negative | 60 | 32 | −0.04 |
| Jennings & Matthews (1984) | Child | Aggression | Nonnegative | 17 | 17 | −0.53 |
| Maliphant et al. (1990) | Adolescent | Conduct problems | Nonnegative | 12 | 20 | −1.67 |
| Marchel (1993) | Adolescent | Aggression | Negative | 80 | 0 | −0.20 |
| McBurnett (1989) | Child | Conduct problems | Nonnegative | 15 | 37 | 0.00 |
| McLaughlin (1995) | Child | Aggression | Nonnegative | 29 | 0 | −0.30 |
| Milner (1976) | Adult | Psychopathy/sociopathy | Negative | 29 | 0 | 0.09 |
| Ogloff & Wong (1990) | Adult | Psychopathy/sociopathy | Negative | 16 | 16 | −0.81 |
| Pelham et al. (1991) | Child | Aggression | Negative | 10 | 10 | −0.49 |
| Pham et al. (2000) | Adult | Psychopathy/sociopathy | Uncodable | 14 | 15 | −0.15 |
| Pitts (1993) | Child | Aggression | Negative | 65 | 38 | −0.41 |
| Tharp et al. (1980) | Adult | Psychopathy/sociopathy | Uncodable | 15 | 15 | 0.00 |
| Thomas (1982) | Adult | Aggression | Nonnegative | 13 | 0 | −1.14 |
| Tucker (1991) | Child | Conduct problems | Nonnegative | 32 | 44 | 0.00 |
| van Goozen et al. (1998) | Child | Conduct problems | Negative | 21 | 31 | 0.05 |
| Winkel et al. (1987) | Adolescent | Aggression | Nonnegative | 28 | 0 | 0.51 |
| Wolfe et al. (1983) | Adult | Aggression | Negative | 7 | 7 | −0.10 |
| Zahn & Kruesi (1993) | Child | Aggression | Nonnegative | 63 | 0 | 0.69 |

*Note.* *N* for studies that reported correlations or other single-group analyses is reported in the $N_e$ column. $N_e$ = number of participants in experimental (i.e., aggressive, psychopathic/sociopathic, conduct problems) group; $N_c$ = number of participants in control group; $d_+$ = effect size weighted by reciprocal of variance.

Table A3
*Coded Qualities and Weighted Effect Sizes of Studies of Heart Rate Reactivity*

| Study | Age | Behavior pattern | Stimulus valence | $N_e$ | $N_c$ | $d_+$ |
|---|---|---|---|---|---|---|
| Arnett et al. (1997) | Adult | Psychopathy/sociopathy | Nonnegative | 13 | 19 | 0.62 |
| Babcock et al. (2004) | Adult | Aggression | Negative | 102 | 0 | −0.06 |
| Borkovec (1970) | Uncodable | Psychopathy/sociopathy | Nonnegative | 19 | 46 | 0.00 |
| Cohn (1995)[a] | Adolescent | Aggression | Negative | 21 | 21 | 0.54 |
| Cohn (1995)[a] | Adolescent | Aggression | Nonnegative | 21 | 21 | −0.10 |
| Cohn (1995)[a] | Adolescent | Aggression | Nonnegative | 19 | 19 | 0.32 |
| Davies & Maliphant (1971) | Adolescent | Conduct problems | Negative | 7 | 7 | −1.24 |
| Eisenberg et al. (1996) | Child | Conduct problems | Negative | 95 | 0 | −0.02 |
| Eisenberg et al. (1996) | Child | Conduct problems | Negative | 92 | 0 | 0.47 |
| Frodi & Lamb (1980) | Adult | Aggression | Negative | 14 | 14 | 0.73 |
| Garralda et al. (1990) | Uncodable | Conduct problems | Nonnegative | 4 | 5 | 0.00 |
| Gerra et al. (1997) | Adult | Aggression | Nonnegative | 15 | 15 | 0.84 |
| Gerra et al. (2001)[a] | Adult | Aggression | Negative | 20 | 0 | 1.14 |
| Gerra et al. (2001)[a] | Adult | Aggression | Negative | 20 | 0 | 1.40 |
| Hare (1968) | Adult | Psychopathy/sociopathy | Nonnegative | 21 | 12 | −0.65 |
| Hare (1982) | Adult | Psychopathy/sociopathy | Negative | 14 | 12 | −0.66 |
| Hare & Craigen (1974) | Adult | Psychopathy/sociopathy | Negative | 17 | 17 | 0.00 |
| Hare et al. (1978) | Adult | Psychopathy/sociopathy | Negative | 24 | 40 | 0.59 |
| House & Milligan (1976) | Adult | Psychopathy/sociopathy | Negative | 32 | 32 | 0.00 |
| Ishikawa et al. (2001) | Adult | Psychopathy/sociopathy | Negative | 13 | 26 | 0.35 |
| Johnson & Rule (1986) | Adult | Aggression | Negative | 40 | 0 | 0.51 |
| Keltikangas-Jarvinen & Keinonen (1988) | Adolescent | Aggression | Nonnegative | 19 | 16 | −0.41 |
| Levenston et al. (2000) | Adult | Psychopathy/sociopathy | Nonnegative | 17 | 18 | 0.56 |
| Maliphant et al. (1990) | Adolescent | Conduct problems | Nonnegative | 12 | 20 | −0.55 |
| Marchel (1993) | Adolescent | Conduct problems | Negative | 80 | 0 | 0.49 |
| McBurnett (1989) | Child | Conduct problems | Nonnegative | 15 | 37 | 0.00 |
| Meehan et al. (2001) | Adult | Aggression | Negative | 19 | 39 | 0.00 |
| Milner (1976) | Adult | Psychopathy/sociopathy | Negative | 29 | 0 | −0.05 |
| Ogloff & Wong (1990) | Adult | Psychopathy/sociopathy | Negative | 16 | 16 | −0.39 |
| Patrick et al. (1994) | Adult | Psychopathy/sociopathy | Nonnegative | 35 | 18 | −0.33 |
| Raskin & Hare (1978) | Adult | Psychopathy/sociopathy | Negative | 10 | 7 | 0.00 |
| Schachter & Latane (1964) | Adult | Psychopathy/sociopathy | Uncodable | 9 | 9 | 0.99 |
| Smith & Gallo (1999) | Adult | Aggression | Uncodable | 60 | 0 | 0.00 |
| Smithmyer (2001) | Child | Aggression | Negative | 67 | 0 | 0.23 |
| Zahn & Kruesi (1993) | Child | Aggression | Nonnegative | 63 | 0 | −0.91 |

*Note.* *N* for studies that reported correlations or other single-group analyses is reported in the $N_e$ column. $N_e$ = number of participants in experimental (i.e., aggressive, psychopathic/sociopathic, conduct problems) group; $N_c$ = number of participants in control group; $d_+$ = effect size weighted by reciprocal of variance.
[a] Multiple independent samples from a single study.

(*Appendix continues*)

Table A4
*Coded Qualities and Weighted Effect Sizes of Studies of Resting Electrodermal Activity*

| Study | Age | Behavior pattern | $N_e$ | $N_c$ | $d_+$ |
|---|---|---|---|---|---|
| Arnett et al. (1993) | Adult | Psychopathy/sociopathy | 31 | 32 | −0.30 |
| Babcock et al. (2004) | Adult | Aggression | 72 | 30 | 0.11 |
| Beauchaine et al. (2001) | Adolescent | Conduct problems | 20 | 22 | −0.65 |
| Blackburn (1979) | Adult | Psychopathy/sociopathy | 18 | 14 | 0.57 |
| Blair (1999) | Adolescent | Psychopathy/sociopathy | 16 | 15 | −0.37 |
| Delamater & Lahey (1983) | Child | Conduct problems | 21 | 0 | −1.02 |
| Fox & Lippert (1963) | Uncodable | Psychopathy/sociopathy | 10 | 10 | −1.17 |
| Frodi & Lamb (1980) | Adult | Aggression | 14 | 14 | 0.00 |
| Garralda et al. (1989) | Child | Conduct problems | 7 | 8 | 0.89 |
| Haldeman (1973) | Adult | Psychopathy/sociopathy | 30 | 10 | −0.42 |
| Harden et al. (1995) | Child | Conduct problems | 18 | 15 | 0.00 |
| Hare (1965b) | Uncodable | Psychopathy/sociopathy | 10 | 10 | −0.28 |
| Hare (1968) | Adult | Psychopathy/sociopathy | 39 | 12 | −0.51 |
| Hare (1982) | Adult | Psychopathy/sociopathy | 16 | 15 | 0.00 |
| Hare & Craigen (1974) | Adult | Psychopathy/sociopathy | 17 | 17 | −0.57 |
| Hare et al. (1978) | Adult | Psychopathy/sociopathy | 24 | 40 | 0.00 |
| Herpertz, Wenning, et al. (2001) | Child | Conduct problems | 26 | 21 | −0.09 |
| Hinton et al. (1980) | Adult | Psychopathy/sociopathy | 7 | 7 | −1.86 |
| House & Milligan (1976) | Adult | Psychopathy/sociopathy | 32 | 32 | −0.70 |
| Ingersoll (1977) | Adult | Psychopathy/sociopathy | 9 | 11 | −0.34 |
| Ishikawa et al. (2001) | Adult | Psychopathy/sociopathy | 29 | 26 | −0.36 |
| Lippert & Senter (1966) | Adolescent | Psychopathy/sociopathy | 21 | 21 | 0.00 |
| Marchel (1993) | Adolescent | Conduct problems | 40 | 40 | 0.07 |
| McBurnett et al. (1993) | Child | Conduct problems | 31 | 26 | −0.53 |
| Milner (1976) | Adult | Psychopathy/sociopathy | 30 | 0 | 0.11 |
| Ogloff & Wong (1990) | Adult | Psychopathy/sociopathy | 16 | 16 | −0.91 |
| Schmidt et al. (1985) | Child | Conduct problems | 11 | 11 | −0.29 |
| Smithmyer (2001) | Child | Aggression | 69 | 0 | 0.11 |
| Steinberg & Schwartz (1976) | Adult | Psychopathy/sociopathy | 10 | 12 | 0.00 |
| Tucker (1991) | Child | Conduct problems | 32 | 44 | −0.56 |
| Witkin-Lanoil (1977) | Adolescent | Conduct problems | 128 | 0 | 0.04 |
| Wolfe et al. (1983) | Adult | Aggression | 5 | 5 | 0.08 |
| Zahn & Kruesi (1993) | Child | Conduct problems | 27 | 31 | 0.21 |

*Note.* *N* for studies that reported correlations or other single-group analyses is reported in the $N_e$ column. $N_e$ = number of participants in experimental (i.e., aggressive, psychopathic/sociopathic, conduct problems) group; $N_c$ = number of participants in control group; $d_+$ = effect size weighted by reciprocal of variance.

Table A5
*Coded Qualities and Weighted Effect Sizes of Studies of Task Electrodermal Activity*

| Study | Age | Behavior pattern | Stimulus valence | $N_e$ | $N_c$ | $d_+$ |
|---|---|---|---|---|---|---|
| Aniskiewicz (1979) | Adult | Psychopathy/sociopathy | Negative | 13 | 13 | −0.81 |
| Arnett et al. (1997) | Adult | Psychopathy/sociopathy | Negative | 29 | 29 | −0.78 |
| Babcock et al. (2004) | Adult | Aggression | Negative | 72 | 30 | 0.13 |
| Blackburn (1979) | Adult | Psychopathy/sociopathy | Negative | 18 | 14 | 0.00 |
| Blair (1999) | Child | Psychopathy/sociopathy | Negative | 16 | 16 | −0.12 |
| Blair et al. (1997) | Adult | Psychopathy/sociopathy | Nonnegative | 18 | 18 | −0.03 |
| Borkovec (1970) | Uncodable | Psychopathy/sociopathy | Nonnegative | 19 | 46 | −0.49 |
| Delamater & Lahey (1983) | Child | Conduct problems | Nonnegative | 19 | 17 | −0.63 |
| Garralda et al. (1989) | Child | Conduct problems | Nonnegative | 15 | 7 | 0.04 |
| Garralda et al. (1991) | Child | Conduct problems | Nonnegative | 25 | 25 | −0.52 |
| Haldeman (1973) | Adult | Psychopathy/sociopathy | Negative | 30 | 10 | −0.16 |
| Harden & Pihl (1995) | Adolescent | Conduct problems | Negative | 28 | 0 | 0.00 |
| Harden et al. (1995) | Child | Conduct problems | Uncodable | 18 | 15 | −0.56 |
| Hare (1965a) | Uncodable | Psychopathy/sociopathy | Negative | 10 | 10 | −0.52 |
| Hare (1965b) | Adult | Psychopathy/sociopathy | Nonnegative | 12 | 12 | −0.84 |
| Hare (1972) | Adult | Psychopathy/sociopathy | Nonnegative | 16 | 16 | −1.00 |
| Hare (1978b) | Adult | Psychopathy/sociopathy | Uncodable | 32 | 32 | −0.66 |
| Herpertz, Wenning, et al. (2001) | Adult | Psychopathy/sociopathy | Uncodable | 25 | 24 | −0.69 |
| Herpertz, Werth, et al. (2001) | Child | Conduct problems | Negative | 26 | 21 | −0.74 |
| Hinton et al. (1980) | Adult | Psychopathy/sociopathy | Nonnegative | 7 | 7 | 0.00 |
| Ingersoll (1977) | Adult | Psychopathy/sociopathy | Nonnegative | 9 | 11 | −0.28 |
| Ishikawa et al. (2001) | Adult | Psychopathy/sociopathy | Nonnegative | 29 | 26 | −0.24 |
| Levenston et al. (2000) | Adult | Psychopathy/sociopathy | Nonnegative | 17 | 16 | −0.44 |
| Lippert & Senter (1966) | Adolescent | Psychopathy/sociopathy | Negative | 21 | 21 | −0.62 |
| Lykken (1957) | Adult | Psychopathy/sociopathy | Negative | 14 | 11 | −0.82 |
| Mangina et al. (2000) | Child | Conduct problems | Nonnegative | 10 | 10 | −6.71 |
| Marchel (1993) | Adolescent | Conduct problems | Negative | 40 | 40 | −0.05 |
| McBurnett et al. (1993) | Child | Conduct problems | Nonnegative | 31 | 26 | −0.74 |
| Milner (1976) | Adult | Psychopathy/sociopathy | Negative | 30 | 0 | 0.20 |
| Plovnick (1976) | Adolescent | Psychopathy/sociopathy | Nonnegative | 311 | 0 | 0.00 |
| Raine (1987) | Adult | Psychopathy/sociopathy | Nonnegative | 36 | 0 | −1.00 |
| Raine & Venables (1981)[a] | Adolescent | Psychopathy/sociopathy | Negative | 41 | 0 | −0.55 |
| Raine & Venables (1981)[a] | Adolescent | Conduct problems | Negative | 52 | 0 | 0.16 |
| Raine & Venables (1984b)[a] | Adolescent | Psychopathy/sociopathy | Nonnegative | 49 | 0 | 0.06 |
| Raine & Venables (1984b)[a] | Adolescent | Conduct problems | Nonnegative | 44 | 0 | −0.36 |
| Schmidt et al. (1985) | Child | Conduct problems | Nonnegative | 11 | 11 | 0.00 |
| Siddle et al. (1973) | Adolescent | Conduct problems | Nonnegative | 15 | 20 | −0.69 |
| Sutker (1970) | Adult | Psychopathy/sociopathy | Nonnegative | 12 | 12 | 0.00 |
| Tharp et al. (1980) | Adult | Psychopathy/sociopathy | Negative | 15 | 15 | −0.74 |
| Tong (1959) | Adult | Aggression | Nonnegative | 19 | 22 | 0.42 |
| Tucker (1991) | Child | Conduct problems | Nonnegative | 32 | 44 | −0.18 |
| Waid (1975) | Adult | Psychopathy/sociopathy | Nonnegative | 10 | 17 | −0.77 |
| Waid & Orne (1982) | Adult | Psychopathy/sociopathy | Nonnegative | 9 | 11 | −1.12 |
| Waid et al. (1979) | Adult | Psychopathy/sociopathy | Negative | 12 | 13 | −0.82 |
| Witkin-Lanoil (1977) | Adolescent | Conduct problems | Nonnegative | 128 | 0 | −0.09 |
| Wolfe et al. (1983) | Adult | Aggression | Negative | 5 | 5 | 1.76 |
| Zahn & Kruesi (1993) | Child | Aggression | Nonnegative | 63 | 0 | −0.18 |

*Note.* *N* for studies that reported correlations or other single-group analyses is reported in the $N_e$ column. $N_e$ = number of participants in experimental (i.e., aggressive, psychopathic/sociopathic, conduct problems) group; $N_c$ = number of participants in control group; $d_+$ = effect size weighted by reciprocal of variance.
[a] Multiple independent samples from a single study.

(*Appendix continues*)

Table A6
*Coded Qualities and Weighted Effect Sizes of Studies of Electrodermal Reactivity*

| Study | Age | Behavior pattern | Stimulus valence | $N_e$ | $N_c$ | $d_+$ |
|---|---|---|---|---|---|---|
| Babcock et al. (2004) | Adult | Aggression | Negative | 102 | 0 | 0.32 |
| Friedrich et al. (1985) | Adult | Aggression | Negative | 14 | 15 | 0.00 |
| Frodi & Lamb (1980) | Adult | Aggression | Negative | 14 | 14 | 0.91 |
| Garralda et al. (1990) | Uncodable | Conduct problems | Nonnegative | 4 | 6 | 0.00 |
| Hare (1965c) | Adult | Psychopathy/sociopathy | Negative | 11 | 11 | −0.88 |
| Hare (1982) | Adult | Psychopathy/sociopathy | Negative | 14 | 12 | −0.66 |
| Hare & Craigen (1974) | Adult | Psychopathy/sociopathy | Negative | 17 | 17 | −0.71 |
| Hare et al. (1978) | Adult | Psychopathy/sociopathy | Negative | 12 | 52 | −0.50 |
| Hare & Quinn (1971) | Adult | Psychopathy/sociopathy | Negative | 18 | 18 | −0.67 |
| House & Milligan (1976) | Adult | Psychopathy/sociopathy | Negative | 32 | 32 | −0.77 |
| Lippert & Senter (1966) | Adolescent | Psychopathy/sociopathy | Nonnegative | 21 | 21 | 0.00 |
| Lykken (1957) | Adult | Psychopathy/sociopathy | Negative | 18 | 11 | −0.76 |
| Marchel (1993) | Adolescent | Aggression | Negative | 80 | 0 | −0.72 |
| Mathis (1970) | Adult | Psychopathy/sociopathy | Uncodable | 20 | 20 | 0.00 |
| Milner (1976) | Adult | Psychopathy/sociopathy | Negative | 29 | 0 | 0.21 |
| Patrick et al. (1994) | Adult | Psychopathy/sociopathy | Nonnegative | 35 | 18 | 0.00 |
| Raskin & Hare (1978) | Adult | Psychopathy/sociopathy | Negative | 24 | 24 | 0.00 |
| Schmauk (1970) | Adult | Psychopathy/sociopathy | Negative | 10 | 10 | −1.35 |
| Smithmyer (2001) | Child | Aggression | Nonnegative | 69 | 0 | −0.12 |
| Sutker (1970) | Adult | Psychopathy/sociopathy | Negative | 12 | 12 | 0.82 |

*Note.* *N* for studies that reported correlations or other single-group analyses is reported in the $N_e$ column. $N_e$ = number of participants in experimental (i.e., aggressive, psychopathic/sociopathic, conduct problems) group; $N_c$ = number of participants in control group; $d_+$ = effect size weighted by reciprocal of variance.

Received March 7, 2002
Revision received October 18, 2003
Accepted October 20, 2003 ■