

Implications of Resilience Concepts for Scientific Understanding

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ABSTRACT: Resilience is an interactive concept that refers to a relative resistance to environmental risk experiences, or the overcoming of stress or adversity. As such, it differs from both social competence positive mental health. Resilience differs from traditional concepts of risk and protection in its focus on individual variations in response to comparable experiences. Accordingly, the research focus needs to be on those individual differences and the causal processes that they reflect, rather than on resilience as a general quality. Because resilience in relation to childhood adversities may stem from positive adult experiences, a life-span trajectory approach is needed. Also, because of the crucial importance of gene–environment interactions in relation to resilience, a wide range of research strategies spanning psychosocial and biological methods is needed. Five main implications stem from the research to date: (1) resistance to hazards may derive from controlled exposure to risk (rather than its avoidance); (2) resistance may derive from traits or circumstances that are without major effects in the absence of the relevant environmental hazards; (3) resistance may derive from physiological or psychological coping processes rather than external risk or protective factors; (4) delayed recovery may derive from “turning point” experiences in adult life; and (5) resilience may be constrained by biological programming or damaging effects of stress/adversity on neural structures.

KEYWORDS: gene–environment interactions; individual differences; stress/adversity; coping processes; turning point experiences; biological effects

INTRODUCTION

The term *resilience* is used to refer to the finding that some individuals have a relatively good psychological outcome despite suffering risk experiences that would be expected to bring about serious sequelae.¹ In other words, it implies

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relative resistance to environmental risk experiences, or the overcoming of stress or adversity.²⁻⁴ It is not, however, just social competence⁵ or positive mental health.⁶ Both of them are important concepts but they refer to something different from resilience. Essentially, resilience is an interactive concept that is concerned with the combination of serious risk experiences and a relatively positive psychological outcome despite those experiences.

There are two sets of research findings that provide a background to the resilience notion. First, there is the universal finding of huge individual differences in people's responses to all kinds of environmental hazard.¹ Before inferring resilience from these individual differences in response there are two major methodological artifactual possibilities that have to be considered. To begin with, apparent resilience might be simply a function of variations in risk exposure. This possibility means that resilience can only be studied effectively when there is both evidence of environmentally mediated risk and a quantitative measure of the degree of such risk. The other possible artifact is that the apparent resilience might be a consequence of measuring too narrow a range of outcomes. The implication is that the outcome measures must cover a wide range of possibly adverse sequelae. The details of the research strategies that need to be employed for these purposes are considered in Rutter¹ and Rutter.⁷

Second, there is the evidence that, in some circumstances, the experience of stress or adversity sometimes *strengthens* resistance to later stress⁸—a so-called “steeling” effect. Although the research literature is much more sparse than that on individual differences in response to environmental hazards, there are some empirically based examples of stress experiences increasing resistance to later stress.⁹ For example, it has been shown that experimental stress in rodents leads to structural and functional effects on the neuroendocrine system that are associated with greater resistance to later stress.⁹ Similarly, repeated parachute jumping by humans leads to physiological adaptation associated with both a change in the timing and nature of the anticipatory physiological response and also the reduced subjective feeling of stress.⁸ It is well known, of course, that exposure to infections (either by natural exposure or through vaccination or immunization) leads to relative immunity to later exposure to the same infectious agents. The experience of happy separations in early childhood may also possibly lead to a better adaptation to hospital admission.¹⁰ Older children's experience of coping successfully with family poverty seemed, in the Californian studies of the Great Depression, to lead to greater psychological strengths later.¹¹ It is important to question what are the circumstances that lead stress/adversity to result in steeling effects rather than sensitization. There is a paucity of good research data on this matter but it seems that probably the key element is some form of successful coping with the challenge or stress or hazard. This is likely to involve physiological adaptation, psychological habituation, a sense of self-efficacy, the acquisition of effective coping strategies, and/or a cognitive redefinition of the experience.

DOES RESILIENCE ADD TO RISK AND PROTECTION CONCEPTS?

Whenever a new term becomes fashionable, it is always necessary to consider whether it is simply a new way of repackaging old material or whether it introduces some new perspective. In other words, is resilience just a fancy way of reinventing concepts of risk and protection? It is not, because risk and protection both start with a focus on variables, and then move to outcomes, with an implicit assumption that the impact of risk and protective factors will be broadly similar in everyone, and that outcomes will depend on the mix and balance between risk and protective influences. By contrast, resilience starts with a recognition of the huge individual variation in people's responses to the same experiences, and considers outcomes with the assumption that an understanding of the mechanisms underlying that variation will cast light on the causal processes and, by so doing, will have implications for intervention strategies with respect to both prevention and treatment.

Does that mean that resilience concepts reject the traditional study of risk and protective factors? Certainly not, because there is an abundance of evidence that much of the variation in psychopathological outcomes can be accounted for by the summative effects of risk and protective factors. Also, and more importantly, resilience is an interactive concept that can only be studied if there is a thorough measurement of risk and protective factors. In short, resilience requires the prior study of risk and protection but adds a different, new dimension.

A second possibility that has to be considered is that because resilience is an inference based on evidence of an interaction, this means that it can be adequately assessed through finding a statistically significant multiplicative interaction. At first sight, it sounds obvious that that must be the case, but in fact it is wrong. That is because a statistical interaction requires variation in both variables and not just one and because synergistic interactions may involve either an additive or a multiplicative interaction.^{12,13} The point about statistical interaction requiring variation in both variables is that there are quite common circumstances in which there clearly is an interaction in a biological sense, but yet this is not reflected in the statistical interaction term. For example, there are major individual differences in people's responses to malaria, and something is known about the genes that moderate this. This will not result in a statistical interaction in areas where malaria is endemic because everyone will have been exposed to more or less the same degree of risk of infection. Similarly, there are big individual differences, again genetically influenced, in atopy. Thus, some people respond to the spring pollens by the development of hay fever, whereas others do not show this response. But, in ordinary circumstances everyone living in the same area is exposed to much the same level of pollens. Accordingly, there will be no statistical interaction, despite the obvious evidence of a biological interaction.

A further possibility that has to be considered is the assumption that it should be possible to measure resilience directly as an observed trait, rather than having to rely on an inference based on some kind of interaction, however, assessed. Numerous researchers and clinicians are searching for such questionnaire or interview measures of this postulated trait. It is a fallacious approach, however, because resilience is not a single quality. People may be resilient in relation to some sort of environmental hazards but not others. Equally they may be resilient in relation to some kinds of outcomes but not others. In addition, because context may be crucial, people may be resilient at one time period in their life but not at others.

GENE-ENVIRONMENT INTERACTION ($G \times E$)

Some of these issues are well illustrated by considering findings on gene-environment interactions ($G \times E$) in relation to some environmental risk influence, as investigated with respect to some psychopathological outcome. For a long time, behavioral geneticists tended to argue that such interactions were sufficiently rare and so minor in their effects that they could be ignored in most genetic analyses. It is clear that this was a mistaken assumption.^{14,15} $G \times E$ relies on studying an environmental risk factor for which there is good evidence of substantial risk, and of environmental mediation of that risk, as well as heterogeneity in outcome. In other words, despite conventional wisdom suggesting the opposite, the implication is that, in the present state of knowledge, the starting point has to be the study of environmental risk, and not identification of genetic risk.¹⁵⁻¹⁷

Three key findings from the Dunedin study well illustrate the phenomenon. FIGURES 1, 2, and 3 show the pattern. FIGURE 1 deals with variations in response to childhood maltreatment in terms of the outcome of antisocial behavior, according to moderation by the allelic variation in the gene that regulates MAOA activity. Considered in quantitative terms there was no main effect of genes, there was a small, significant effect of childhood maltreatment, but the big effect came from the interaction. Childhood maltreatment had a rather small effect on the individuals with high MAOA activity but it had a very big effect in relation to those with low MAOA activity. FIGURE 2 shows a comparable pattern with respect to the serotonin transporter gene again with maltreatment as the risk variable, but this time with depression as the outcome. FIGURE 3 shows the findings in relation to the valine variant of the COMT gene in relation to the effects of early heavy use of cannabis, schizophrenia being the outcome variable. In both these latter examples, there was the same overall pattern of no genetic main effect, a significant environmental effect, but with the biggest effect coming from the interaction between the identified gene and the measured risk environment. Each of these findings has now been replicated in one way or another and the serotonin transporter gene finding also has a much broader

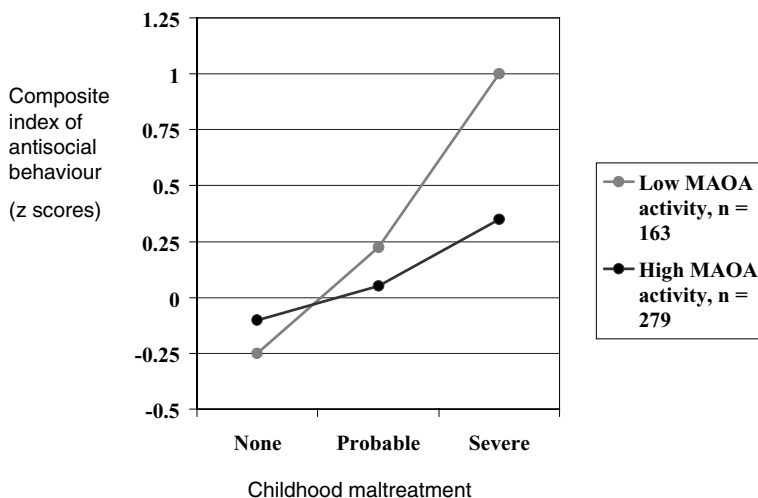


FIGURE 1. Antisocial behavior as a function of MAOA activity and a childhood history of maltreatment.²⁸

body of biological research using a range of research strategies including imaging studies of response to stress, rearing studies in rhesus monkeys, and animal models of other kinds.¹⁵ There are a series of quite important methodological checks that need to be undertaken before inferring a $G \times E$ but such steps were undertaken in a thorough and resolute fashion by the Dunedin study team and the results are compelling in showing that the interaction is valid, and not artefactual.

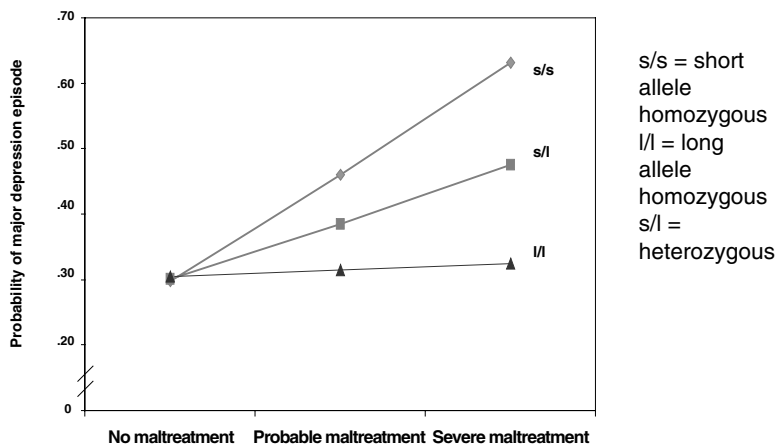


FIGURE 2. Effect of maltreatment in childhood on liability to depression moderated by 5-HTT gene.²⁹

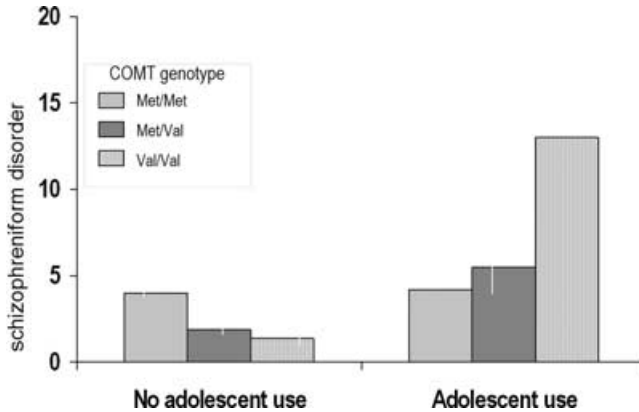


FIGURE 3. Schizophrenia spectrum disorder: cannabis use interacts with genotype.³⁰

There are four main lessons from the body of research on $G \times E$. First, as in the three Dunedin study examples, the influence of the genes was only shown through demonstration of the interaction with the environmental hazard. Second, in each case, the $G \times E$ was specific to a particular psychopathological outcome. The finding underlines the fact that there is not, and cannot be, a single universally applicable resilience trait. Third, the implication of the $G \times E$ is that both the G and the E share the same causal pathophysiological pathway. Of course, that suggestion needs to be tested. Nevertheless, the point is that the resilience finding has causal process implications for both genes and environment. Fourth, the genetic variant is neither a risk nor a protective factor in itself. That is, there is little or no effect on psychopathology in the absence of the environmental risk factor. There could scarcely be any better example of the value of a resilience concept in studying causal processes because it identified a significant and important genetic effect that would not have been detected in the absence of studying the interaction.

OTHER LESSONS FROM RESILIENCE FINDINGS

Obviously, resilience is not just a feature of $G \times E$. There are numerous other circumstances in which resilience is evident. The findings from such studies bring out four more important lessons for scientific understanding. First, resistance to environmental hazards may come from *exposure* to risk in controlled circumstances, rather than *avoidance* of risk. This is best demonstrated, of course, in the natural immunity to infections and that brought about by immunization and vaccination. It is also evident in the rodent studies of stress to which reference has already been made. The Californian studies of the great economic depression¹¹ provide an interesting example of the benefits of adolescents coping successfully, the contrast being with the findings

of adverse effects in younger children who were not able to cope in the same way. Treatment studies of fears and phobias have also shown that exposure is an important (although not necessarily essential) element in their successful treatment.^{18,19} Avoidance of the feared object is the action most likely to lead to persistence of the fear. It has to be said that there is a paucity of good evidence on the protective effect of controlled exposure to stress/adversity in relation to psychopathological outcomes, and clearly there is a need to consider both physiological mediation and cognitive/affective mediation. Nevertheless, the parallels with internal medicine are sufficiently compelling to indicate that it is quite likely that there are psychological parallels to the immunity example.

Second, protection may derive from circumstances that are either neutral or risky in the absence of the key environmental hazard. For example, it is apparent in the protection against malaria provided by heterozygote sickle-cell status.²⁰ Being a carrier of the sickle-cell is not in itself a good thing but it happens to be protective against malaria. It has no particular benefits for people living in a malaria-free area but it has important benefits for those in areas where malaria is endemic. Adoption may well constitute a psychological example. Adoption is an experience that probably carries some risks (albeit small ones) that stem from it being atypical in all societies. If children who are adopted come from a low-risk background, there are no particular advantages to being adopted. By sharp contrast, however, for children who have been exposed in early life to parental abuse or neglect, adoption can be highly advantageous.²¹

FIGURE 4 illustrates the point in a somewhat different way by its indication that actions that are protective in depriving circumstances may be of no particular benefit in advantageous conditions. Quinton and Rutter²² showed that the

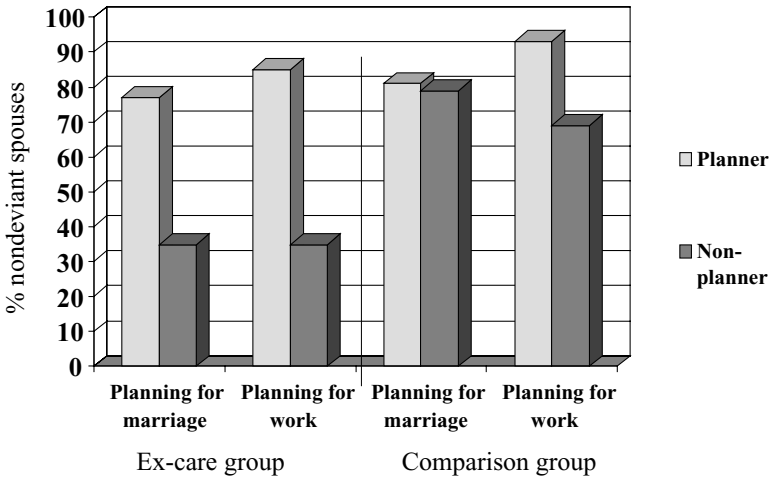


FIGURE 4. Planning and nondeviant spouses.²²

phenomenon of planning (meaning no more than taking considered decisions rather than acting impulsively) made it much more likely that young people who had been reared in institutions would marry a nondeviant spouse. Moreover, this effect was evident, not just with respect to planning for marriage, but also planning as evident in the work context. By contrast, the planning tendency made no significant difference in a comparison population sample. The point is that the peer group for the children who had been raised in residential group homes was largely a deviant one and when they left the institutions and returned to discordant families there was considerable pressure to marry to get out of the arena of conflict. This did not apply in the comparison group who, if they married entirely by random selection, were most likely to land up with a nondeviant spouse and their circumstances provided no particular pressure to marry in haste.

The third message, therefore, is that protection may derive from what people do to deal with stress or adversity. That is, the notion of resilience focuses attention on coping mechanisms, mental sets, and the operation of personal agency. In other words, it requires a move from a focus on external risks to a focus on *how* these external risks are dealt with by the individual. More generally, this means that resilience, unlike risk and protective factor approaches, forces attention on dynamic *processes*, rather than static factors that act in summative fashion. Such processes may involve neurotransmitters as in the $G \times E$ example, neuroendocrine effects as seen in stress adaptation, or cognitive/emotional mechanisms. It should be noted that the study of cognitive/emotional mechanisms may require qualitative methods to generate hypotheses (although quantitative measures will still be required to test the hypothesis so generated). Thus, Hauser *et al.*,²³ in their study of resilience in young people who had had a prolonged psychiatric hospitalization, found that three features were strongly characteristic of resilience (as compared with average outcomes). These were: personal agency and a concern to overcome adversity; a self-reflective style; and a commitment to relationships.

Fourth, protection may derive from circumstances that come about long after the risk experience. In other words, resilience may sometimes reflect later recovery, rather than an initial failure to succumb. Thus, Laub and Sampson,²⁴ in their follow-up of the Gluecks' institutionalized sample, showed that a beneficial turning point effect was seen with a supportive marriage. It might have been supposed that the beneficial effect derived solely from a secure attachment relationship, but their findings indicated that the benefits also stemmed from the new extended kin network and friendship group that marriage brought, providing hitherto lacking positive role models. Also, the spouses frequently exerted informal controls as well as support, marital obligations often cut off the antisocial individual from the delinquent peer group, and marriage brought expectations of providing financial support (so that regular employment also provided social controls). Much the same complex mix of influences was seen with the parallel finding of the turning point effect of armed services for young

people from a severely disadvantaged background.²⁵ It was not that serving in the armed services was of itself beneficial but, rather, it provided opportunities for continuing education in a more adult environment. It brought a widening of the peer group and it often delayed marriage until careers were more effectively managed.

IS RESILIENCE UNLIMITED?

Resilience notions have generally been interpreted as conveying great optimism regarding the possibility of surviving adversity. Such optimism is well justified but it is necessary to ask whether resilience is limited. Findings indicate that it is. Thus, our follow-up study of children from profoundly depriving residential institutions in Romania, who were adopted into well-functioning UK families, showed remarkable persistence of adverse sequelae even after more than 7.5 years in the adoptive home.^{26,27} As FIGURE 5 indicates, there were no persisting sequelae that could be detected when the children had left the institutions before the age of 6 months but there was then a marked increase in multiple impairments that occurred even within the group of children spending just 6 to 12 months in depriving institutions. Curiously, there was no further increase in risk with persistence of the depriving circumstances beyond the 6-month period (at least up to the age of 42 months). The implication seems to be that the pervasively depriving circumstances took some months to have an effect but when they lasted beyond the age of 6 months, they tended to have effects that endured many years. The inference is that there may have been some form of intraorganismic change—either neural damage or biological programming of some kind.

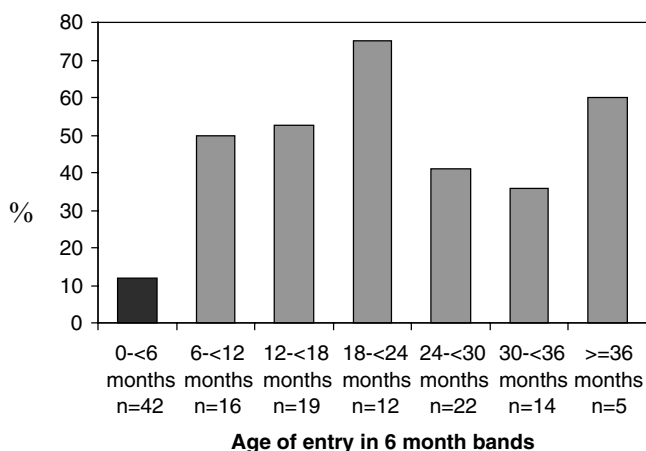


FIGURE 5. Rates of children with 2+ impairments by age of entry pooled in 6-month bands (institution-reared Romanian adoptees) (from Kreppner *et al.*, submitted).

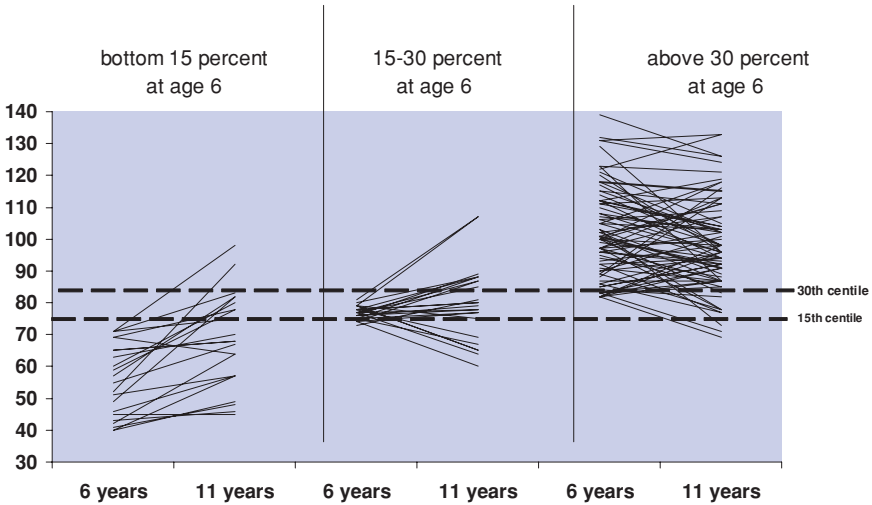


FIGURE 6. Change in IQ between ages 6 and 11.²⁶

On the other hand, although the effects were remarkably persistent, there was change between 6 and 11 years as illustrated in FIGURE 6 showing a line scattergram for cognitive change between 6 and 11 years. It is notable that the changes, however, were largely confined to the group that was most impaired at 6 years. Also, it is striking that there was huge heterogeneity in outcome with some children showing superior cognitive functioning despite the prolonged institutional deprivation. Factors promoting resilience in the face of this extraordinarily pervasive and profound deprivation remain unclear.

CONCLUSIONS

There are three broad research implications that derive from resilience findings. Because resilience is not a general quality that represents a trait of the individual, research needs to focus on the processes underlying individual differences in response to environmental hazards, rather than resilience as an abstract entity. Second, because resilience in relation to adverse childhood experiences may stem from positive adult experiences, it is necessary to adopt a life-span trajectory approach that can investigate later turning point effects. Third, because of the importance of $G \times E$, it will be necessary to combine psychosocial and biological research approaches and to use a diverse range of research strategies. These should include functional imaging of cognitive processing, neuroendocrine studies, investigation of mental sets and models, and the use of animal studies of various kinds.

Resilience findings also provide five key implications for scientific understanding of substantive effects. First, resistance to hazards may derive from

controlled exposure to risk (rather than its avoidance). Second, resistance may derive from traits or circumstances that are either risky or neutral in the absence of the relevant environmental hazard. Third, resistance may derive from physiological or psychological coping processes, rather than external risk or protective factors. Fourth, delayed recovery may derive from “turning point” effects in adult life. Fifth, resilience may be constrained by biological programming or by the damaging effects of stress/adversity on neural structures.

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