
SPECIAL SECTION ARTICLE

Resilience as a dynamic concept

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Abstract

The concept of resilience has as its starting point the recognition that there is huge heterogeneity in people's responses to all manner of environmental adversities. Resilience is an inference based on evidence that some individuals have a better outcome than others who have experienced a comparable level of adversity; moreover, the negative experience may have either a sensitizing effect or a strengthening "steeling" effect in relation to the response to later stress or adversity. After noting the crucial importance of first testing for the environmental mediation of risk through "natural experiments," findings are reviewed on "steeling effects" in animal models and humans. Gene–environment interaction findings are considered, and it is noted that there is some evidence that the genetic influences concerns responsivity to all environments and not just bad ones. Life course effects are reviewed in relation to evidence on turning point effects associated with experiences that increase opportunities and enhance coping. Attention is drawn to both research implications and substantive findings as features that foster resilience.

Norm Garmezy was one of the most important pioneers in the conceptualization and study of resilience from the early 1970s onward (Garmezy, 1974, 1985). Several features made his approach distinctive. First, in keeping with Eisenberg (1977), he viewed development as the unifying concept in the study of psychopathology. This was the central element in the field of developmental psychopathology that he did so much to advance (Rutter, 2008; Rutter & Garmezy, 1983). Two key elements defined developmental psychopathology: the focus on continuities and discontinuities over time, and continuities and discontinuities between normality and mental disorder (Rutter, 1986). It was notable that this involved no presumption that either continuities or discontinuities would predominate. Rather, testing constituted an essential part of the research endeavor.

Second, Garmezy was forthright in requiring a methodologically rigorous approach to data analysis (Garmezy, Masten, & Tellegen, 1984). Resilience should not constitute a theory, nor should it be seen as equivalent to positive psychology or competence. Both of the latter are valid and useful concepts (see Masten & Tellegen, 2012 [this issue]) but they differ from resilience. However, all require longitudinal study for their rigorous investigation; all need to consider multifactor-

ial causal pathways; and all need to examine gene–environment interdependence.

Third, in his own research, Garmezy had been motivated by Bleuler's (1978) study of the children of mothers with schizophrenia, which showed that even in this high-risk group there were numerous examples of individuals who showed adaptive patterns of social behavior and work achievement. Garmezy appreciated that the high risk involved a genetic liability but, equally, he realized that being raised by a schizophrenic mother involved environmental as well as genetic risks (see Rutter, 1989, for a fuller discussion on this point). He decided that there was need to study stress resistance in high-risk groups but chose to focus on psychosocial disadvantage in community samples. In that connection, he was clear that risk and protective influences should not be defined on the basis of theoretical or ideological presumptions. Rather, the influences needed to be investigated systematically in order to understand how they *actually* operated in the samples under study. Moreover, it should not necessarily be expected that a universal answer would be found; effects were likely to be shaped by social context (Rutter, 1999).

Fourth, Garmezy appreciated the need for resilience research to include positive personality dispositions, a nurturant family milieu, and external societal support systems. This broad-based conceptualization meant that he was resistant to notions of inherent "invulnerability" that were being put forward by others in the 1970s and 1980s (Anthony, 1974; Anthony & Cohler, 1987). Resilience had to be viewed as a process and not as a fixed attribute of an individual. Of course, it was likely that some individuals would show resilience across a range of circumstances and across a range of

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outcomes, but it could not be assumed that the same features would be protective in relation to all risks. Moreover, resilience was an interactive concept and had to be inferred; it could not be measured directly as if it was a characterological trait.

Fifth and finally, although very much a user of quantitative methods, Garmezy was basically interested in what experiences meant for individuals and how research findings might be used to develop better means of helping children who experience serious stress and adversity. Accordingly, he paid attention to qualitative, as well as quantitative, research strategies (see Rutter, in press-a, for a discussion of the value of both).

It should be obvious from what I have written how much I am indebted to Norm. My own research has been greatly influenced by his input as experienced through joint collaborations and discussions (he was a great talker, full of wit as well as wisdom). What made Norm so different from others was the integrated breadth of his conceptualizations across diverse approaches, his rejection of theories that claimed to explain everything, his concern and compassion for those who were disadvantaged, and his commitment to a positive problem-solving approach. The whole field of resilience research, and of developmental psychopathology more generally, was shaped by Norm's vision; scientific papers today continue to show his imprint (albeit not always explicitly acknowledged).

Conceptualization of Resilience

Particularly during the last two decades, there has been a marked tendency for researchers, clinicians, and policy makers to shift their focus from risk to resilience (e.g., Mohaupt, 2008). The aim was to emphasize the positive rather than the maladaptive. This was seen in the emergence of "positive psychology," as a major movement (Seligman & Csikszentmihalyi, 2000) and Layard's (2005) "happiness" agenda. The valuable aspect of this movement was the recognition that eudaimonic socioemotional well-being (including a sense of purpose and direction) was as important as economic success (Keyes, 2007). The less helpful aspect was the triviality of relabeling family conflict as a risk and family harmony as a protective factor. The most crucial point is that there was the downgrading of the seriousness of mental disorder in order to concentrate on variations in degree of happiness in the general population, and hence the downgrading of resilience in the face of severe stress and adversity. In addition, insofar as resilience is concerned, there is the misleading implication that it requires generally superior functioning, rather than relatively better functioning compared with that shown by others experiencing the same level of stress or adversity. There are also methodological problems that are inherent in the concept of "positive mental health" (Jahoda, 1959) and difficulties in differentiating between hedonic pleasure and excitement and the quiet satisfaction of a job well done (see Rutter, 2011).

The concept of psychological and social competence (Masten et al., 1999) raises a rather different set of issues. It

has two great strengths. First, it is obviously a desirable outcome, and second, it is quantifiable. Nevertheless, it has three important limitations (see Rutter, 2011). First, it implies that, usually, the causal influences will be much the same in non-stressed groups as in those suffering from extreme adversity. Nonlinear interactive effects are also systematically considered, but they have to be derived from mathematical models with all the uncertainties that these require about the assumptions (see also Parker & Maestripieri, 2011; Seery, 2011), rather than measured directly. Nevertheless, they can be examined and, when they are, promotive factors that apply outside of resilience also contribute to resilience in the face of adversity (see Bowes, Maughan, Caspi, Moffitt, & Arseneault, 2010; Masten et al., 2004). Second, it implies that outcomes generally will be explicable on the balance between risk and protective factors. That suggests that protective factors can be identified on the basis of their nature, rather than their effects. In many circumstances that works but, as discussed below, protection may come from risk experiences that lead to successful coping. Third, and most crucially, it assumes that most individuals will respond to stress and adversity in much the same way and to the same degree or that, at the very least, prevention may best be achieved by acting on that assumption.

Masten and Powell (2003) have argued that promotive factors tend to operate in the same way in all populations, and hence, that resilience can best be promoted by focusing on competence. Such promotive factors include cognitive abilities, temperament, parenting quality, and good schools. Their arguments are correct but, nevertheless, do not focus on the influences that *do* work differently in the presence of adversity; that is what defines resilience.

The concept of resilience has a quite different starting point. It has its origins in the universal finding from all research, naturalistic and experimental, human and other animals, that there is huge heterogeneity in response to all manners of environmental hazards: physical and psychosocial (Rutter, 2006). It is argued that the systematic investigations of the causes of this heterogeneity should not just throw light on the specifics of different responses to a particular hazard but, in addition, might throw light on a broader range of causal processes.

Accordingly, resilience can be defined as reduced vulnerability to environmental risk experiences, the overcoming of a stress or adversity, or a relatively good outcome despite risk experiences (Rutter, 2006). Thus, it is an *interactive* concept in which the presence of resilience has to be *inferred* from individual variations in outcome among individuals who have experienced significant major stress or adversity (Rutter, 1987).

Testing for Environmental Mediation of Risks

This interactive concept of resilience necessitates testing the postulate that the stress or adversity does entail an environmentally mediated risk. Of course, it has long been appreciated that a statistical correlation or association does not nec-

essarily mean causation. What is new is the range of quasi-experimental research strategies, employing a range of “natural experiments” that facilitate causal inferences (Rutter, 2007, 2009, in press-b). These all start with an appreciation of the various reasons why a correlation might *not* imply causation. Thus, the risk might be genetically, rather than environmentally, mediated; it might represent reverse causation (i.e., the disorder led to the supposed risk factor, rather than the other way round); it might reflect social selection (i.e., the process by which individuals select or shape their environments); or it might reflect the origins, rather than the risk actions, of the supposed risk factor.

More than a dozen different forms of “natural experiments” have been devised and found to be useful. Here, there is space only to illustrate the strategies through a few examples. The possibility of genetic mediation has been examined by discordant twin designs in which one twin experienced the risk factor and the other did not (Kendler & Prescott, 2006); by assisted reproductive technologies comparing offspring born by methods in which the child and mother share genes (e.g., sperm donation) with those such as egg donation in which that was not the case (Rice et al., 2009; Thapar et al., 2009); and by children of twins designs capitalizing on the fact that the offspring of monozygotic females are genetically half-siblings but socially cousins (D’Onofrio et al., 2003, 2008; Silberg & Eaves, 2004; Silberg, Maes, & Eaves, 2010). The findings have been informative in showing that some risks for psychopathology (such as parental negativity and prenatal smoking exposure) are partially genetically mediated but others (such as physical and sexual abuse) are largely environmentally mediated. The findings have also shown that parental mental disorder that involves a substantial genetic liability may nevertheless have environmentally mediated effects on mental disorder in the children.

Among the designs that can obviate the possibility of social selection are those that examine risks that operate on the whole population. This is exemplified by the study of the effects of prenatal starvation on the risk of developing schizophrenia brought about by the Dutch famine in World War II (Hoek, Brown, & Susser, 1998; Stein, Susser, Saenger, & Marolla, 1975); the study of the effects of stopping the use of measles, mumps, and rubella vaccines to test the hypothesis that measles, mumps, and rubella had led to an epidemic of autism, finding that it had not done so (Honda, Shimizu, & Rutter, 2005); and the investigation into the effects of poverty on psychopathology by examining the benefits for children that followed the relief of poverty as a result of the opening of a casino on a native Indian reservation (Costello, Compton, Keeler, & Angold, 2003).

The possibility for reverse causation could be examined by means of instrumental variable approaches, of which Mendelian randomization is a specific example (Davey-Smith & Ebrahim, 2003, 2005). The findings have shown that the supposed causal effect of early puberty on the liability to alcoholism was largely an artifact (Prescott & Kendler, 1999) and the postulated causal effect of early use of alcohol

on antisocial behavior reflected a genetic liability and not a causal pathway (Poulton & Moffitt, 2008). Resilience research cannot sensibly be based on statistical risk effects that have not undergone rigorous testing of the hypothesis of environmentally mediated causation.

Finally, it is crucial that the study of resilience be preceded by careful analysis of the elements in the environmental risk variable that actually involve causal influences. History shows how easy it is to misidentify these. Thus, for many years it was supposed that “broken homes” or family breakup were involved in the causation of both antisocial behavior and depression (see British Academy Working Group, 2009). Quantitative analyses of longitudinal data have shown that the risks for antisocial behavior following family separation are minor compared to the risks from family discord, after controlling for the other in each case (see Fergusson, Horwood, & Lynskey, 1992). Similarly, the proximal risks for depression were found to stem from poor parenting (brought about by family breakup) and not from the breakup as such (Harris, Brown, & Bifulco, 1986).

“Steeling” or Strengthening Effects

One of the features that particularly characterizes resilience research is the recognition of the importance of possible “steeling” effects. That is *exposure* to stresses or adversities may either increase vulnerabilities through a sensitization effect or decrease vulnerabilities through a steeling effect. A key question concerns the circumstances that lead to the one rather than the other, plus the equally important question of the mechanisms that mediate those effects. This research process is most easily illustrated through reference to research undertaken by David Lyons’ research group (Lyons et al., 2010; Lyons & Parker, 2007; Lyons, Parker, Katz, & Schatzberg, 2009; Parker, Buckmaster, Schatzberg, & Lyons, 2004) using squirrel monkeys. This built on the early studies by Gig Levine in the early 1960s and continued up to the time of his death (Levine & Mody, 2003).

In brief, they used a strategy that mimicked the normal tendency in nature for the occurrence of brief mother–infant separations brought about by the mothers going off to forage for food when the newly weaned offspring reached 3–6 months of age. Socially housed squirrel monkeys were randomized at 17 weeks of age to either brief intermittent separations or a nonseparated control condition. The separated individuals were removed from the rearing group for a 2-hr period each week for a total of 10 weeks. After 27 weeks of age, both groups were reared in identical conditions. Behavioral, hormonal, and brain imaging data were obtained at specified ages up to adulthood. At 9 months in a novel environment test, the two groups were initially similar but differences emerged over repeated sessions. Cognitive control was assessed at 1.5 years and curiosity in a stress-free situation was measured at 2.5 years. On all these measures, the separated group performed better. In addition, cortisol measures showed decreased reactivity to stress. Neuroimaging showed

that the separated monkeys had a larger ventromedial cortical volume.

In order to test whether the benefits of intermittent brief separation were mediated by changes in maternal behavior, monkeys were randomized to three postnatal conditions, one of which involved separations of mother and offspring together as a pair. It was found that the changes in arousal regulation more closely corresponded to stress exposure than to separation-induced changes in maternal care.

Other findings showed that similar beneficial effects of brief stress exposure were found in rats. However, this was *not* found with prolonged separation experiences that instead led to increased sensitization to later stress experiences rather than steeling. Other rat studies have shown that the adverse effects are a function of uncontrollable stressors (Maier, Amat, Baratta, Paul, & Watkins, 2006), a finding that provides a link to human evidence on the benefits of coping (see below). A different randomization study with adult squirrel monkeys showed again that brief intermittent separations (in this case, from a familiar adult male companion) led to increased hippocampal neurogenesis and altered gene expression.

The overall body of evidence from animal models provides strong evidence of the reality of steeling effects from repeated brief stress experiences that are not accompanied by overall adversity or deprivation. The next question concerns the applicability of this effect to humans. Clearly, the best example is provided by the resistance to infections that comes about either through the acquisition of natural immunity through exposure to the infectious agents or through immunization in which induced immunity is brought about by administering a controlled dose of a modified version of the pathogen. This is undoubtedly a steeling effect. However, does it apply to psychosocial stressors and psychopathological outcomes?

The human evidence on this point is much weaker, largely because there have been so few attempts to investigate the matter. However, two examples of possible steeling effects warrant mentioning. First, there are Elder's (1974) longitudinal analyses of the Californian cohorts going through the economic depression of the 1920s and 1930s, in which the children had to take on new responsibilities. The follow-up showed that whereas younger children tended to fair poorly, adolescents were sometimes strengthened by the experience of having to take on adult roles and doing so successfully. Elder's proposed explanation was that the adolescents with greater maturity and experience were better able to take on responsibilities and succeed; finding that they could succeed made them more resilient. By contrast, the younger children could not cope so well (and perhaps had a less obvious relevant role) and were therefore sensitized rather than strengthened.

Second, an example is provided by Stacey, Dearden, Pill, and Robinson's (1970) finding that children who had experienced happy separations from their parents (such as staying with their grandparents or having "sleepovers" with friends)

tended to cope better with the stresses of hospital admission. Of course, admission to hospital involves multiple stressful elements other than separation. Nevertheless, the implication is that successful happy separations seem to foster resilience in dealing with unhappy separations.

The animal model findings have often been interpreted as meaning that the mechanism involves some sort of "inoculation" effect; in other words, exposure to a small dose of some hazard serves to build up resistance to a major dose through the body having had the opportunity to acquire effective defenses. That may well be involved, but the human studies suggest that that is likely to be too narrow a perspective. Thus, the adolescents in Elder's study who appeared to be strengthened by having to take on new responsibilities as a result of the great economic depression seemed to acquire a sense of self-efficacy and mastery. It did not appear that the new responsibilities "inoculated" them against greater responsibilities, rather the strengthening effect seemed to operate more broadly.

Similarly, Hauser, Allen, and Golden's (2006) qualitative study of young adults who had been institutionalized for a serious mental disorder in adolescence but who nevertheless ended up successful and optimistic was not distinctive in terms of resilience-building inoculation experiences. Rather, what stood out as different from those who were less resilient was a personal agency involving a concern to act to overcome adversity, a self-reflective style that meant that they tried to assess what was and what was not working for them, and a commitment to relationships.

These findings resonate with those from Quinton and Rutter's (1988) study of girls reared in residential group homes. As compared with girls from the same geographical area living with their families, many left their institutions in late adolescence with a feeling that there was nothing they could do to affect what happened to them. They lacked what was termed "planning" in relation to marriage or work. It was not that they planned badly, but rather that they did not feel able to plan at all. However, this did not apply to all the girls. Some did show "planning," and it was found that this was associated with earlier successes in other areas of their lives, rarely academic but in some activity important to them, such as sport, music, and positions of responsibility. Those who did show planning had better outcomes overall. As in the Hauser et al. (2006) study, their resilience seemed to stem from successes leading to self-efficacy rather than from minor stresses overcome. The findings are far too sparse for firm conclusions but they all point to the important quality being a state of mind rather than a high IQ or some particular temperamental feature.

Gene–Environment Interactions (G × E)

Resilience encompasses resistance to adverse environmental influences, as well as steeling following brief intermittent stressors (Rutter, 2009). This is best illustrated by the findings on G × E. Quantitative genetic studies pointed to the likeli-

hood of $G \times E$ operating, perhaps especially with respect to depression and antisocial behavior (Rutter & Silberg, 2002). However, the situation was transformed by the availability of molecular genetic methods to identify individual susceptibility genes. $G \times E$ could then be tested for by using those genes in relation to measured environments that have been shown to involve environmentally mediated risks for psychopathology (Moffitt, Caspi, & Rutter, 2005). Moffitt, Caspi, and colleagues led the way through a series of papers based on the Dunedin Longitudinal Study (see, e.g., Caspi et al., 2002, 2003, 2005). In each case, there was no significant main effect of genes, a marginally significant main effect of environment, but a clearly significant effect of the $G \times E$. The $G \times E$ held up after rigorous control for possible alternative explanations (such as the scaling effect, an effect of a gene–environment correlation [rGE], and $G \times G$ interactions; see Rutter, Thapar, & Pickles, 2009).

The issues are most easily considered in relation to the Dunedin finding of $G \times E$ between the short allele polymorphism of the serotonin transporter promoter gene and both life events and maltreatment (Caspi et al., 2003). The finding has been replicated or partially replicated many times (Uher & McGuffin, 2008), but Risch et al. (2009) sought to claim that this was likely to be an artifact. The claim was based on a flawed meta analysis of a biased subset of studies using life events (see Caspi, Hariri, Holmes, Uher, & Moffitt, 2010; Uher & McGuffin, 2010), which ignored findings based on maltreatment and ignored all experimental biological studies in humans and in nonhuman animals (see Rutter, 2010; Rutter et al., 2009). Two examples of experimental studies may be given: one in rhesus monkeys focusing on central serotonin functioning (Bennett et al., 2002) and one in humans, using brain imaging to examine the neural effects of $G \times E$ (Hariri et al., 2002; see also Hariri, 2011). Both were strongly supportive of the reality of biological $G \times E$. The human study was additionally important because the findings derived from a sample of individuals deliberately chosen to be free of psychopathology. The implication is that the $G \times E$ biological pathway, although relevant for depression, is one present in the general population who do not have depression. The finding reinforces the epidemiological claim that the $G \times E$ does not reflect a genetic main effect on depression.

Further epidemiological studies have been highly informative in focusing down on the specifics of $G \times E$ with the serotonin transporter promoter gene. First, Karg, Burmeister, Shedden, and Sen (2011) brought together a diverse range of studies focusing on different types of stressor. The findings showed that, although there was a marginally significant $G \times E$ with life events, there was a much larger highly significant $G \times E$ with maltreatment. That is important both because it focuses on a more serious environmental risk factor and because the E applied to experiences in childhood long before the onset of a depressive episode in late adolescence or early adult life. The implication is that a causal biological pathway brought about earlier changes associated with the liability to depression, rather than a provoking effect of the onset of an

episode of depression. Second, a finding from Uher et al. (2011) showed that the $G \times E$ mainly applied to recurrent or chronic depression rather than a single episode depressive disorder. This again suggests a biological effect on liability rather than a provoking effect on onset.

Third, the next finding to emphasize is that the serotonin transporter promoter gene $G \times E$ applied to depression as an outcome, but not to antisocial behavior. Conversely, the monoamine oxidase A $G \times E$ applied to antisocial behavior but not to depression. That has two important implications. It means that resilience cannot be viewed as a general trait; the interaction found in relation to one outcome does not necessarily apply to others. In addition, just because there was no measurable effect of maltreatment on depression in the individuals with the short allele polymorphism, that does not mean that these individuals are invulnerable to the effects of maltreatment, because they may develop other adverse outcomes.

Fourth and finally, it is necessary to ask whether the genetic effect operates as an influence on vulnerability to adverse experiences or rather an influence on susceptibility to all experiences, good or bad. Pluess and Belsky (2009) and Boyce and Ellis (2005) have argued that, from an evolutionary perspective, an effect on response to all experiences is more likely. In other words the relevant genetic polymorphism that is associated with vulnerability to bad experiences may also be associated with a *better* response to good experiences in the absence of environmental adversity. There are findings that are consistent with this proposition (see Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011) but the findings in humans so far leave some important questions unanswered. Thus, the Belsky and Beaver (2011) study found the differential susceptibility only in males; Pluess and Belsky (2011) had findings that suggested possible prenatal programming of postnatal plasticity; and Obradović, Bush, and Boyce (2011) found that the effects varied according to whether the challenges were interpersonal or cognitive. Nevertheless, the balance of findings indicates the likelihood that the genetic polymorphisms that are associated with increased vulnerability to bad experiences may also be associated with greater sensitivity to good experiences.

The finding from the Suomi group studies (Suomi, personal communication, 2011) has been striking in finding that all the $G \times E$ interactions they have found show this “crossover” effect. Moreover, it is even more striking that the less efficient transcription allele is the one associated with the greater vulnerability to adversities, whereas the more efficient transcription allele is the one with the vulnerability effect in advantageous circumstances. The mechanisms involved have yet to be determined but the phenomenon certainly seems to have a biological plausibility.

Life Course Effects

The research discussed so far has mainly concerned circumstances in early life, and it is necessary to ask whether the overcoming of adversity can be influenced by experiences

in adult life. There is clear evidence from both animal models and human studies that adult experiences have both behavioral and neural effects (Keating, 2011). However, the issue here is the rather different one of whether such experiences can counter or alleviate the effects of earlier adversities. Findings from Lyons et al. (2010), studying brief separation experiences in adult squirrel monkeys, has already been noted. Such experiences not only provided a behavioral and neuroendocrine steeling effect with respect to later stresses, but also there were measurable neural and epigenetic effects. Although the findings provide a convincing demonstration of steeling effects in adult life in relation to *later* stresses, they do not address the question of effects that serve to counter *earlier* adversities. There is other animal research that suggests that this does occur but it may be more fruitful to turn to human studies on life span effects.

Probably the best research is that undertaken by Sampson and Laub (1993; Laub & Sampson, 2003). Their starting point was boys in residential institutions for delinquents, first studied by the Gluecks (Glueck & Glueck, 1950). Laub and Sampson followed the sample up to 70 years of age, and Laub personally interviewed a purposively chosen subsample of 52, chosen to represent those who had an outcome much better than expected on the basis of their previous behavior and psychological background, as well as those who had done averagely well or as expected. One of the adult experiences they investigated was marriage, which had been postulated to have a protective effect (Laub, Nagin, & Sampson 1998). Their first approach was a multivariate analysis that did indeed show that marriage had a protective effect. They then argued that if this was a true effect, it should follow that crime rates should vary over time according to whether the individuals were currently married or not married. A propensity score approach was used to create groups equivalent with respect to a propensity to marriage. The findings showed that marriage was associated with a reduction in crime of approximately 36% to 43% (Sampson, Laub, & Wimer, 2006).

The final step lay in Laub's qualitative interviews. These showed that marriage constituted far more than the provision of a stable attachment relationship. It brought about a new kinship group and a new peer group, as well as a strong guiding influence from the wife. Marriage was a life-changing experience and not just an "event."

A second adult life experience examined was military service. Perhaps unexpectedly, this was associated with a substantially better outcome in the socially disadvantaged youths studied (Sampson & Laub, 1996). The meaning of this finding has to take account of both the social context (many of the youths had dropped out of schooling and viewed their future in very negative terms) and the benefits (both intended and incidental) that came with military service. The intended benefit came from the educational opportunities provided by the GI bill and the unintended from the postponement of marriage that resulted in having a much wider choice of partners, many of whom did not have the same disadvantaged background, and marriage at a time of having established some

sort of career. It may be concluded that positive experiences in adult life can do much to counter the effects of early adversities provided that they serve to both "cut off" the past and provide new opportunities.

Although not planned as a study of turning points, the adult follow-up of Masten et al.'s (2004) Project Competence produced strikingly comparable findings. The seven individuals who made a dramatic change from maladaptive to resilient over the transition to adulthood differed from their peers in terms of "planfulness," future motivation, autonomy, and adult support outside the family. Thus, both mental qualities and new opportunities seemed important. Individual cases suggested that the latter happened through moving to a good job, marrying into better functioning families, experiencing religious conversion, and/or the pursuit of higher education. Bowes et al.'s (2010) separate study also showed the role of positive family relationships in fostering resilience.

Resilience Versus the Summative Effect of Risk and Protective Factors

It is necessary now to return to the starting point of the differences between the *interactive* concept of resilience and the *summative* vulnerability and competence approach. The conceptual differences were outlined in Rutter (2006), but here we focus on the rather different issues of the research implications and substantive findings.

Research considerations

Four key research implications stand out. First, the focus on individual differences in outcome requires a specific identification of the key risk factors and, second, it demands rigorous testing of the hypothesis that the risks are truly environmentally mediated. Neither of these needs tend to be met in even the best of the vulnerability/competence studies (Fergusson & Horwood, 2003; Masten & Powell, 2003; Sameroff, Gutman, & Peck, 2003), although its importance has been recognized (Luthar & Brown, 2007). Third, the examination of individual differences calls for experimental testing (preferably combined with biological measures). Fourth, it also calls for the use of animal models. This was evident in the research into "steeling" effects and also that into gene-environment interactions. Of course, this is not to say that none of these needs are ever met in vulnerability/competency research; but it is to say that they are to the forefront as a priority in resilience research, whereas that is less commonly the case in other approaches. The need for a perspective that spans biology and psychosocial influences has become accepted (see Cicchetti, 2010), but the crucial differences between resilience research and risk and protective factors research are less well appreciated.

Substantive findings

The resilience research has given rise to several substantive findings that would have been unlikely to arise if the focus

had not been on the individual differences in response to the same level and type of adversities. First, there are the steeling effects finding that intermittent *exposure* to brief periods of stress, far from being damaging, increases *resistance* to later stresses. This has been shown by both behavioral and neuroendocrine effects. The conclusion is in keeping with the biological understanding that developmental benefits from meeting, and successfully coping with, challenges (Rutter & Rutter, 1992). It is also in keeping with the consistent finding that resistance to infectious agents comes through exposure to, and not avoidance of, contact with those agents. In addition, it is consonant with the clinical evidence that the treatment of phobias benefits from exposure to and is hampered by avoidance of, the feared object. It may be concluded that these “steeling” effects needed a resilience approach for their identification.

Second, the genetic findings on $G \times E$ could only have come from a focus on *interactive* effects. In all the replicated findings, no main genetic effect has been found. The genetic influence was *not* on a liability for a particular mental disorder, but rather on a susceptibility to environmental influences. The findings were also important in showing that the main risk effect came from serious, chronic adversities (such as maltreatment) rather than acute stresses. However, this is even more important in showing that the $G \times E$ operated in relation to environmental experiences long before the onset of disorder. Some sort of biological pathway (reflecting $G \times E$) that predisposed to a psychopathological liability, rather than the provoking of an onset, seemed to be operative.

Third, the life course findings indicate that appropriate experiences in adult life can do much to counter the effects of earlier adversities. It needs to be noted, however, that such experiences are not simply pleasurable happenings but, rather, experiences that create a helpful discontinuity with the past, and increase opportunities and enhance coping.

Fourth, resilience is accompanied by important biological changes, neuroendocrine and neural. Resilience is a dynamic concept in which successful coping may involve a complicated mixture of psychological habituation, changes in mental set, alterations in perceived and actual self-efficacy, hormonal changes (especially in the hypothalamic–pituitary–adrenal axis) and neural alterations.

Fifth, the findings on ideas, attributions, self-reflection, and planning emphasize the importance of mental phenomena in the response to stress and adversity (i.e., in the *processes* of coping and not just in terms of enduring temperamental features).

Sixth, the interactive resilience approach emphasizes that resilience needs to be judged, not in terms of superior overall functioning as judged in relation to the population as a whole, but rather in terms of functioning that is relatively better than that shown by others experiencing the same level of adversity. As the study of Romanian adoptees indicated (Rutter & Sonuga-Barke, 2010), that may well mean a mixture of important real-life successes in the context of some continuing difficulties.

Seventh and finally, although recognizing the huge strides in understanding resilience that have been made since Garmezy’s pioneering concepts and findings, it has to be accepted that many key questions have still to be addressed. It is crucially important that, in pointing to new research successes, the results are not oversold. All research builds on a broad base of investigations by other research groups, and it is rare indeed for a single finding to constitute a true “break-through.”

Conclusions

Resilience research has as its starting point the universal finding of huge heterogeneity in outcomes after all types of environmental adversity, together with the evidence that, in some circumstances, exposure to stress may be followed by an increased resistance to later stress (a steeling effect), rather than a sensitization or increased vulnerability. In other words, the focus is on individual differences in response to adversity rather than an assumption that outcomes can be accounted for in terms of the balance between positive and negative influences, with the assumption that they will affect most people in the same way and to the same degree.

There are some nine features that serve to characterize resilience research as distinctive from the overall field of risk and protective factors. First, there is a *direct* analysis of the features associated with heterogeneity in response to adversity, rather than a reliance on statistical approaches to detect nonlinear interactive effects. The statistical power to detect interactions is inevitably less than the power to determine the associations with heterogeneity of outcomes. In addition, there is the requirement to test for environmental mediation of risk effects, rather than relying on quantifying a heterogeneous mixture of risks that may be either genetically mediated or environmentally mediated or both.

Second, there is an interest in variables that are without effect in the general population of lower risk individuals but which have substantial effects in the presence of adversity. Adoption is the obvious example of this kind. Of course, it could be identified in risk and protective factor studies but it has not been so identified, probably because of its infrequency in the total population. Planning constitutes a further example in which its origins lay in good experiences outside the family. That would be unlikely to have been picked up in the usual type of risk and protective factor study. However, it is both relevant and noteworthy that the importance of planning was detected in Masten et al.’s (2004) competence study once there was a focus on individual differences.

Third, there is an interest in the steeling effects of successfully coping with stress or challenge. That could have arisen as a result of risk/protective studies but it has not been a prominent feature, probably because what was needed was a focused hypothesis-testing approach, a feature of resilience research but not other approaches, at least not to the same extent.

Fourth, as a specific example of hypothesis-driven strategies, there is the group of studies of $G \times E$ interactions. Once

more, the need (that was met) was for hypotheses driven by biological findings (see Rutter et al., 2009). In addition, however, there was the explicit acceptance that epidemiological findings had to be put to the test through human experimental studies, animal models, and basic science.

Fifth, that brings in the central importance in resilience research of animal models, of which the squirrel monkey studies of possible stress inoculation, represent a good example. The focus is explicitly on possible steeling effects and an experimental approach is used.

Sixth, there is the study of possible turning point effects, as illustrated by the study of the beneficial effects of marriage and of early service in the Armed Forces for individuals from a disadvantaged background living through the Great Depression of the 1930s. General population longitudinal studies provided the data but it was the focus on individual differences that brought this research into the resilience arena.

Seventh, a key feature of resilience research has been the use of qualitative data to determine the *meaning* of experiences. The research into marriage constitutes one example of this and the study of positive outcomes following inpatient psychiatric care in adolescence constitutes another rather different example.

References

- Anthony, E. J. (1974). The syndrome of the psychologically invulnerable child. In E. J. Anthony & C. Koupernik (Eds.), *The child in his family: Children at psychiatric risk* (pp. 529–545). New York: Wiley.
- Anthony, E. J., & Cohler, B. J. (Eds.). (1987). *The invulnerable child*. New York: Guilford Press.
- Bennett, A. J., Lesch, K. P., Heils, A., Long, J. C., Lorenz, J. G., Shoaf, S. E., et al. (2002). Early experience and serotonin transporter gene variation interact to influence primate CNS function. *Molecular Psychiatry*, 7, 118–122.
- Belsky, J., & Beaver, K. M. (2011). Cumulative–genetic plasticity, parenting and adolescent self-regulation. *Journal of Child Psychology and Psychiatry*, 52, 619–626.
- Bleuler, M. (1978). *The schizophrenic disorders: Long-term patient and family studies*. New Haven, CT: Yale University Press.
- Bowes, L., Maughan, B., Caspi, A., Moffitt, T. E., & Arseneault, L. (2010). Families promote emotional and behavioural resilience to bullying: Evidence of an environmental effect. *Journal of Child Psychology and Psychiatry*, 51, 809–817.
- Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, 17, 271–301.
- British Academy Working Group Report. (2009). *Social science and family policies*. London: British Academy Policy Centre.
- Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry*, 167, 509–527.
- Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 297, 851–854.
- Caspi, A., Moffitt, T. E., Cannon, M., McClay, J., Murray, R., Harrington, H., et al. (2005). Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-*o*-methyltransferase gene: Longitudinal evidence of a gene environment interaction. *Biological Psychiatry*, 57, 1117–1127.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301, 386–389.
- Cicchetti, D. (2010). Resilience under conditions of extreme stress: A multi-level perspective. *World Psychiatry*, 9, 145–154.
- Eighth, there are the basic science findings on brain plasticity (see Rutter, in press-c), which underline the dynamic nature of plasticity in terms of its temporal limits and its openness to external influences.
- Ninth and finally, resilience is defined in terms of a better outcome than that seen in other individuals from a similarly adverse background. In short, there is no requirement of superior functioning in relation to the nondeprived population as a whole. The study of Romanian adoptees who experienced profoundly depriving care (Rutter & Sonuga-Barke, 2010) constitutes a good illustration. Although, in the group as a whole, deficits were apparent, some individuals fared surprisingly well. There was marked relative success of a meaningful kind. Once again, the risks were shown to be environmentally mediated and a hypothesis-testing approach was followed.
- As indicated in the introductory section, resilience concepts accept, and build on, the importance of risk and protective factors research (and require its operation), but they add to it in crucially important ways that would not have emerged at all readily out of other approaches. The fields of competence, positive psychology, risk and protection and resilience all have importance, but it is a mistake to want to group them together.
- Costello, E. J., Compton, S. N., Keeler, S. N., & Angold, A. (2003). Relationships between poverty and psychopathology: A natural experiment. *Journal of the American Medical Association*, 290, 2023–2029.
- Davey Smith, G., & Ebrahim, S. (2003). “Mendelian randomization”: Can genetic epidemiology contribute to understanding environmental determinants of disease? *International Journal of Epidemiology*, 32, 1–22.
- Davey Smith, G., & Ebrahim, S. (2005). What can Mendelian randomization tell us about modifiable behavioural and environmental exposures. *British Medical Journal*, 330, 1076–1079.
- D’Onofrio, B. M., Turkheimer, E., Eaves, L. J., Corey, L. A., Berg, K., Soles, M. H., et al. (2003). The role of the children of Twins design in elucidating causal relations between parent characteristics and child outcomes. *Journal of Child Psychology and Psychiatry*, 44, 1130–1144.
- D’Onofrio, B. M., Van Hulle, C. A., Waldman, I. D., Rodgers, J. L., Harden, K. P., Rathouz, P. J., et al. (2008). Smoking during pregnancy and offspring externalizing problems: An exploration of genetic and environmental confounds. *Development and Psychopathology*, 20, 139–164.
- Eisenberg, L. (1977). Development as a unifying concept in psychiatry. *British Journal of Psychiatry*, 131, 225–237.
- Elder, G. H. (1974). *Children of the Great Depression*. Chicago: University of Chicago Press.
- Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology*, 23, 7–28.
- Fergusson, D. M., & Horwood, L. J. (2003). Resilience to childhood adversity: Results of a 21-year study. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 130–155). Cambridge: Cambridge University Press.
- Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1992). Family change, parental discord and early offending. *Journal of Child Psychology and Psychiatry*, 33, 1059–1075.
- Garnezy, N. (1974). The study of competence in children at risk for severe psychopathology. In E. J. Anthony & C. Koupernik (Eds.), *The child in his family: Children at psychiatric risk* (Vol. 3, pp. 77–97). New York: Wiley.
- Garnezy, N. (1985). Stress-resistant children: The search for protective factors. In A. Davids (Ed.), *Recent research in developmental psychopathology* (pp. 213–233). Elmsford, NY: Pergamon Press.

- Garnezy, N., Masten, A. S., & Tellegen, A. (1984). The study of stress and competence in children: A building block for developmental psychopathology. *Child Development, 55*, 97–111.
- Glueck, S., & Glueck, E. (1950). *Unraveling juvenile delinquency*. New York: Commonwealth Fund.
- Harris, T., Brown, G. W., & Bifulco, A. (1986). Loss of parent in childhood and adult psychiatric disorder: The role of lack of adequate parental care. *Psychological Medicine, 16*, 641–659.
- Hauser, S., Allen, J., & Golden, E. (2006). *Out of the woods: Tales of resilient teens*. Cambridge, MA: Harvard University Press.
- Hariri, A. R. (2011). Neurobiological mechanisms supporting gene–environment interaction effects. In K. A. Dodge & M. Rutter (Eds.), *Gene–environment interactions in developmental psychopathology* (pp. 59–70). New York: Guilford Press.
- Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297*, 400–403.
- Hoek, H. W., Brown, A. S., & Susser, E. (1998). The Dutch famine and schizophrenia spectrum disorders. *Social Psychiatry and Psychiatric Epidemiology, 33*, 373–379.
- Honda, H., Shimizu, Y., & Rutter, M. (2005). No effect of MMR withdrawal on the incidence of autism: A total population study. *Journal of Child Psychology and Psychiatry, 46*, 572–579.
- Jahoda, M. (1959). *Current concepts of positive mental health*. New York: Basic Books.
- Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry, 68*, 444–454.
- Keating D. P. (Ed.). (2011). *Nature and nurture in early child development*. New York: Cambridge University Press.
- Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders*. New York: Guilford Press.
- Keyes, C. L. M. (2007). Promoting and protecting mental health as flourishing: A complementary strategy for improving national mental health. *American Psychologist, 62*, 95–108.
- Laub, J. H., Nagin, D. S., & Sampson, R. J. (1998). Trajectories of change in criminal offending: Good marriages and the desistance process. *American Sociological Review, 63*, 225–238.
- Laub, J. H., & Sampson, R. J. (2003). *Shared beginnings, divergent lives: Delinquent boys to age 70*. Cambridge, MA: Harvard University Press.
- Layard, R. (2005). *Happiness*. New York: Penguin Press.
- Levine, S., & Mody, T. (2003) The long-term psychobiological consequences of intermittent postnatal separation in the squirrel monkey. *Neuroscience & Biobehavioral Reviews, 27*, 83–89.
- Luthar, S. S., & Brown, P. J. (2007). Maximizing resilience through diverse levels of inquiry: Prevailing paradigms, possibilities, and priorities for the future. *Development and Psychopathology, 19*, 931–955.
- Lyons, D. M., Buckmaster, P. S., Lee, A. G., Wu, C., Mitra, R., Duffey, L. M., et al. (2010). Stress coping stimulates hippocampal neurogenesis in adult monkeys. *Proceedings of the National Academy of Sciences of the United States of America, 107*, 14823–14827.
- Lyons, D. M., & Parker, K. J. (2007). Stress inoculation-induced indications of resilience in monkeys. *Journal of Trauma Stress, 20*, 423–433.
- Lyons, D. M., Parker, K. J., Katz, M., & Schatzberg, A. F. (2009). Developmental cascades linking stress inoculation, arousal regulation, and resilience. *Frontiers in Behavioral Neuroscience, 3*, 32.
- Maier, S. F., Amat, J., Baratta, M. V., Paul, E., & Watkins, L. R. (2006). Behavioral control, the medial prefrontal cortex, and resilience. *Dialogues in Clinical Neuroscience, 8*, 397–406.
- Masten, A. S., Burt, K. B., Roisman, G. I., Obradović, J., Long, J. D., & Tellegen, A. (2004). Resources and resilience in the transition to adulthood: Continuity and change. *Developmental Psychopathology, 6*, 1071–1094.
- Masten, A. S., Hubbard, J. J., Gest, S. D., Tellegen, A., Garmezy, N., & Raimarz, M. (1999). Competence in the context of adversity: Pathways to resilience and maladaptation from childhood to late adolescence. *Development and Psychopathology, 11*, 143–169.
- Masten, A. S., & Powell, J. L. (2003). A resilience framework for research, policy and practice. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 1–28). Cambridge: Cambridge University Press.
- Masten, A. S., & Tellegen, A. (2012). Resilience in developmental psychopathology: Contributions of the Project Competence Longitudinal Study. *Development and Psychopathology, 24*, 345–361.
- Moffitt, T. E., Caspi, A., & Rutter, M. (2005). Strategy for investigating interactions between measured genes and measured environments. *Archives of General Psychiatry, 62*, 473–481.
- Mohaupt, S. (2008). Review article: Resilience and social exclusion. *Social Policy & Society, 8*, 63–71.
- Obradović, J., Bush, N. R., & Boyce, T. (2011). The interactive effect of marital conflict and stress reactivity on externalizing and internalizing symptoms: The role of laboratory stressors. *Development and Psychopathology, 23*, 101–114.
- Parker, K. J., Buckmaster, C. L., Schatzberg, A. F., & Lyons, D. M. (2004). Prospective investigation of stress inoculation in young monkeys. *Archives of General Psychiatry, 61*, 933–941.
- Parker, K. J., & Maestripieri, D. (2011). Identifying the key features of early stressful experiences that produce stress vulnerability and resilience in primates. *Neuroscience & Biobehavioral Reviews, 35*, 1466–1483.
- Pluess, M., & Belsky, J. (2009). Differential susceptibility to rearing experience: The case of childcare. *Journal of Child Psychology and Psychiatry, 50*, 396–404.
- Pluess, M., & Belsky, J. (2011). Prenatal programming of postnatal plasticity? *Development and Psychopathology, 23*, 29–38.
- Poulton, R., & Moffitt, T. E. (2008). Is it important to prevent early exposure to drugs and alcohol among adolescents? *Psychological Science, 19*, 1037–1044.
- Prescott, C. A., & Kendler, K. S. (1999). Age at first drink and risk for alcoholism: A noncausal association. *Alcoholism: Clinical and Experimental Research, 23*, 101–107.
- Quinton, D., & Rutter, M. (1988). *Parenting breakdown: The making and breaking of intergenerational links*. Aldershot: Avebury.
- Rice, F., Harold, G., Boivin, J., Hay, D., van den Bree, M., & Thapar, A. (2009). Disentangling prenatal and inherited influences in humans with an experimental design. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 2464–2467.
- Risch, N., Herrell, R., Lehner, T., Liang, K. Y., Eaves, L., Hoh, J., et al. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *Journal of the American Medical Association, 301*, 2462–2471.
- Rutter, M. (1986). Child psychiatry: The interface between clinical and developmental research. *Psychological Medicine, 16*, 151–169.
- Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry, 57*, 316–331.
- Rutter, M. (1989). Psychiatric disorder in parents as a risk factor in children. In D. Shaffer, I. Philips, N. Enver, M. Silverman, & V. Anthony (Eds.), *Prevention of psychiatric disorders in child and adolescent: The project of the American Academy of Child & Adolescent Psychiatry. OSAP Prevention Monograph 2* (pp. 157–189). Rockville, MD: US Department of Health and Human Services, Office of Substance Abuse Prevention.
- Rutter, M. (1999). Social context: Meanings, measures and mechanisms. *European Review, 7*, 139–149.
- Rutter, M. (2006). Implications of resilience concepts for scientific understanding. *Annals of the New York Academy of Sciences, 1094*, 1–12.
- Rutter, M. (2007). Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on Psychological Science, 2*, 377–395.
- Rutter, M. (2008). Developing concepts in developmental psychopathology. In J. J. Hudziak (Ed.), *Developmental psychopathology and wellness: Genetic and environmental influences* (pp. 3–22). New York: American Psychiatric Publications.
- Rutter, M. (2009). Understanding and testing risk mechanisms for mental disorders. *Journal of Child Psychology and Psychiatry, 50*, 44–52.
- Rutter, M. (2010). Gene–environment interplay. *Depression & Anxiety, 27*, 1–4.
- Rutter, M. (2011). Resilience: Causal pathways and social ecology. In M. Ungar (Ed.), *The social ecology of resilience*. New York: Springer.
- Rutter, M. (in press-a). The role of science in understanding family troubles. In J. McCarthy (Ed.), *Family troubles?* London: Policy Press.
- Rutter, M. (in press-b). “Natural experiments” as a means of testing causal inferences. In C. Barzini, P. Dawid, & L. Bernardinelli (Eds.), *Statistical methods in causal inference*. New York: Guilford Press.
- Rutter, M. (in press-c). Resilience: Clinical implications. *Journal of Child Psychology and Psychiatry*.

- Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In E. M. Hetherington (Ed.), *Mussen's handbook of child psychology: Vol. 4. Socialization, personality, and social development* (4th ed., pp. 775–911). New York: Wiley.
- Rutter, M., & Rutter, M. (1992). *Developing minds: Challenges and continuity across the life span*. New York: Basic Books.
- Rutter, M., & Silberg, J. (2002). Gene–environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology*, 53, 463–490.
- Rutter, M., & Sonuga-Barke, E. J. (Eds). (2010). Deprivation-specific psychological patterns: Effects of institutional deprivation. *Monographs of the Society for Research in Child Development*, 75, 1–252.
- Rutter, M., Thapar, A., & Pickles, A. (2009). From JAMA: Commentary on paper by Risch et al. (2009). Gene–environment interactions: Biologically valid pathway or artefact? *Archives of General Psychiatry*, 66, 1287–1289.
- Sameroff, A., Gutman, L. M., & Peck, S. C. (2003). Adaptation among youth facing multiple risks: Prospective research findings. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 364–391). Cambridge: Cambridge University Press.
- Sampson, R. J., & Laub, J. H. (1993). *Crime in the making: Pathways and turning points through life*. Cambridge, MA: Harvard University Press.
- Sampson, R. J., & Laub, J. H. (1996). Socioeconomic achievement in the life course of disadvantaged men: Military service as a turning point, circa 1940–1965. *American Sociological Review*, 61, 347–367.
- Sampson, R. J., Laub, J. H., & Wimer, C. (2006). Does marriage reduce crime? A counterfactual approach to within-individual causal effects. *Criminology*, 44, 465–508.
- Seery, M. D. (2011). Resilience: A silver lining to experiencing adverse life events? *Current Directions in Psychological Science*, 20, 390–394.
- Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychology*, 55, 5–14.
- Silberg, J. L., & Eaves, L. J. (2004). Analysing the contributions of genes and parent–child interaction to childhood behavioural and emotional problems: A model for the children of twins. *Psychological Medicine*, 34, 347–356.
- Silberg, J. L., Maes, H., & Eaves, L. J. (2010). Genetic and environmental influences on the transmission of parental depression to children's depression and conduct disturbance: An extended children of twins study. *Journal of Child Psychology and Psychiatry*, 51, 734–744.
- Stacey, M., Dearden, R., Pill, R., & Robinson, D. (1970). *Hospitals, children and their families: The report of a pilot study*. London: Routledge & Kegan Paul.
- Stein, Z. A., Susser, M., Saenger, G., & Marolla, F. (1975). *Famine and human development: The Dutch hunger winter of 1944–1945*. New York: Oxford University Press.
- Thapar, A., Rice, F., Hay, D., Bolvin, J., Langley, K., Van den Bree, M., et al. (2009). Prenatal smoking may not cause ADHD: Evidence from a novel design. *Biological Psychiatry*, 66, 722–727.
- Uher, R., Caspi, A., Houts, R., Sugden, K., Williams, B., Poulton, R., et al. (2011). Serotonin transporter gene moderates childhood maltreatment's effects on persistent but not single-episode depression: Replications and implications for resolving inconsistent results. *Journal of Affective Disorders*. Advance on-line publication.
- Uher, R., & McGuffin, P. (2008). The moderation by the serotonin transporter gene of environmental adversity in the aetiology of mental illness: Review and methodological analysis. *Molecular Psychiatry*, 13, 131–146.
- Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry*, 15, 18–22.