Vulnerability-Stress Models

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CHAPTER 2

Vulnerability-Stress Models

RICK E. INGRAM AND DAVID D. LUXTON

Early models of psychopathology typically identified processes operating during the course of the disorder as reflecting the key determinants of the onset of psychopathology (e.g., irrational beliefs; Ellis, 1962). Such models have led to important advances in understanding important features of psychopathology. For example, in the cognitive arena, schema models initially focused almost exclusively on understanding cognitive variables functioning in the disordered state. This conceptual approach, as well as the empirical research motivated by these models, has led to a number of significant insights into depression (Ingram, Miranda, & Segal, 1998), anxiety (e.g., McManus & Clark, 2002), personality disorders (Beck, 1999), and even problematic marital interactions (Beck, 1989). Schema models thus represent a clear example of the power of such constructs as they apply to the description of psychopathology.

Stress has also been recognized as an important contributor to the development and course of psychopathology, so much so that a variety of models have featured stress as a primary determinant of disordered functioning (Brown & Harris, 1978, 1989). Such models suggest that severe enough negative events could precipitate psychological disorders even without reference to individual psychological or biological characteristics. For example, the link between an adverse social environment and the onset of depression has long been recognized. The majority of research investigating possible links consistently finds a relationship between the experience of stressful life events and the onset of depression, with some data suggesting that approximately 50% of individuals diagnosed with depression have experienced severe stress before onset (Mazure, 1998). More recent perspectives suggest the possibility that life stress may engender a specific subtype of depression (Monroe & Hadjiyannakis, 2002).

Despite advancing understanding, the limitations of these approaches have become increasingly apparent. For instance, models that place primary emphasis on stress as a key cause of a disorder have difficulty dealing with data showing that even extreme stress is not linked to psychopathology in all individuals (Monroe & Hadjiyannakis, 2002); after all, approximately 50% of individuals do *not* show evidence of a disorder such as depression

following significant life stress. Hence, although data convincingly show that stress plays a role in depression, they just as convincingly show that other factors also play a critical role.

The fact that not all individuals who experience significant stress develop a disorder has led, in part, to the recognition that vulnerability processes are important components of psychopathology; such factors predispose some individuals to psychopathology when stress is encountered. Notions about vulnerability have also begun to address questions about whether variables operating within the disordered state are antecedents of the state, or whether they can reasonably be considered to be consequences of the state (e.g., Barnett & Gotlib, 1988). By definition, vulnerability to a disorder must serve as an antecedent of the disorder. Although vulnerability ideas have been a central part of some of the earliest models of psychopathology (e.g., Beck, 1967), the emphasis on their essential nature in the onset of psychopathology has seen a remarkable resurgence (Segal & Ingram, 1994).

Although vulnerability and stress can be reasonably considered to be conceptually distinct constructs, separately, their power to describe key aspects of psychopathology is limited. Thus, most modern models of psychopathology explicitly combine vulnerability and stress in their descriptions of the functional processes leading to disorder. This chapter focuses on the interaction of vulnerability and stress as essential for understand ing the development of psychopathology. To serve as a background for exploring their interactive role, we briefly provide definitions of *vulnerability* and *stress* and then briefly discuss the origins of these constructs. We then examine general principles that characterize most diathesis-stress models and, finally, explore different models of vulnerability-stress interactions. Finally, we comment on some issues that are pertinent to conceptualizations of stress and conceptualizations of diatheses in the context of the diathesis-stress relationship.

DEFINITIONS

Numerous discussions of vulnerability (e.g., Ingram et al., 1998) and stress (e.g., Grant & McMahon, Chapter 1 of this volume) can be found in the literature. Detailed examination of these ideas can be found in these sources. For purposes of context, in this chapter we briefly note ideas about the individual constructs that form diathesis-stress models. We start with stress.

Stress

Definitions of stress encompass a number of facets. In general, however, stress falls into a limited number of broad categories. One major category of stress is conceptualized as the occurrence of significant life events that are interpreted by the person as undesirable (Lazarus & Folkman, 1984; Luthar & Zigler, 1991; Monroe & Peterman, 1988; Monroe & Simons, 1991). The accumulation of minor events or hassles represents another kind of stress (Dohrenwend & Shrout, 1985; Lazarus, 1990). Socioeconomic factors have also been implicated in stress, in that variables such as low maternal educational status or membership in an ethnic minority group may reflect stressful living circumstances (Luthar & Zigler).

Although it is clear from these descriptions that the definitions of *stress* are many, we can view *stress* in the context of this chapter as the life events (major or minor) that disrupt those mechanisms that maintain the stability of individuals' physiology, emotion, and cognition. Indeed, Selye's (1963) classic description of stress notes that such events represent a strain on the person's adaptive capability that cause an interruption of the person's routine or habitual functioning. Stress thus reflects those factors that interfere with the system's physiological and psychological homeostasis.

Even though stress is frequently conceptualized as the occurrence of "externally"

ordained processes, two sets of factors suggest an important role for "internal" forces in the occurrence of stress. First, although some stressful events may simply befall people, several researchers have persuasively argued, and empirically demonstrated, that other events are the results of individuals' own actions (Depue & Monroe, 1986; Hammen, 1991; Monroe & Simons, 1991; Rutter, 1986). For instance, a person with social skills deficits (e.g., inappropriately critical of others) may engender tumultuous relationships with acquaintances, coworkers, and romantic partners that result in the generation of significant stress. Vulnerable individuals, or those in a disordered state, may therefore play a role in creating their own stresses (Ingram et al., 1998). Later in this chapter, we expand on the implications of this idea as it pertains to diathesis-stress models.

A second factor is the influence of appraisal processes on what is perceived to be stressful (Monroe, 1989; Monroe & Simons, 1991). That is, stress is not independent of the individual's appraisals of events. Even though there are a number of events that are undoubtedly universally appraised as stressful (e.g., the death of a loved one), even in these cases individual differences may determine the degree of stress that is perceived and experienced. In other cases, events that are perceived as stressful by some individuals may be perceived and experienced as not stressful, or at the least as minimally stressful, by other individuals. Indeed, a multitude of other factors can affect the determination and degree of stress.

Diatheses

We employ the terms *diathesis* and *vulnerability* interchangeably. A diathesis, or vulnerability, is typically conceptualized as a predispositional factor, or set of factors, that makes possible a disordered state. The earliest psychopathology models featuring vulnerability suggested that these redispositional factors constituted genetic or biological factors. In more recent years, the term has been broadened to include psychological factors, such as cognitive and interpersonal variables, that make a person susceptible to psychopathology (Monroe & Simons, 1991).

Intuitive ideas about vulnerability imply an increased susceptibility to emotional pain and to the occurrence of psychopathology of some type. Yet, as intuitively straightforward as this concept has been, and despite extensive discussion in the literature about vulnerability, few precise definitions are available in the scientific literature. Ingram et al. (1998) noted several core features of vulnerability that appear to constitute the common themes that emerge in discussions of vulnerability and that can thus help establish a working definition of the construct. These ideas suggest that vulnerability is a trait, is stable but can change, is endogenous to individuals, and is usually latent.

Most discussions regard vulnerability as an enduring trait. For example, Zubin and Spring (1977) argued that "we regard [vulnerability] as a relatively permanent, enduring trait" (p. 109). They continue, "The one feature that all schizophrenics have . . . is the everpresence of their vulnerability" (p. 122). Such assumptions of permanence seem likely to be rooted in the genetic level of analysis employed by researchers who pioneered this concept, as can be seen among schizophrenia researchers who point to the genetic endowment of individuals who are at risk for this disorder. Meehl's (1962) idea of schizotaxia represents an inherited neural deficit, whereas other researchers, such as Zubin and Spring, Nicholson and Neufeld (1992), and McGue and Gottesman (1989), explicitly argue that genetic endowment determines one's level of vulnerability (at least to schizophrenia). Hence, little change is theoretically possible.

Although vulnerability may in many cases be permanent and enduring, this need not

always be true. For example, when the level of vulnerability analysis is psychological rather than genetic in nature, change may be possible. Even though assumptions of genetic vulnerability offer little possibility for modification of vulnerability, most psychological approaches rely on assumptions of dysfunctional learning as the genesis of vulnerability. Given such assumptions, vulnerability levels may fluctuate as a function of new learning experiences.

The traitlike nature of vulnerability suggests that vulnerability tends to be, at the least, stable. It is important to note, however, that stability does not necessarily mean permanence. That is, even though the idea of stability suggests a resistance to change, it does not presume that change is never possible. Under some circumstances, positive changes in an otherwise stable variable may very well occur. Indeed, the notion of therapy is based on just this premise. It is also the case, however, that some experiences (e.g., trauma) might serve to strengthen vulnerability. It thus seems reasonable to conceptualize vulnerability as stable but not immutable.

Following from the traitlike characteristics of vulnerability, another core feature of the construct is that vulnerability is an endogenous process. In particular, whether stemming from genetically or biologically acquired characteristics or acquired through psychological or learning processes, vulnerability resides within the person. This serves to explicitly distinguish vulnerability from "external" stress or life events. Finally, because diatheses are often not easily recognized, they are frequently considered to be latent, requiring activation in some fashion before psychopathology can occur. Although not all researchers agree with this position (e.g., Just, Abramson, & Alloy, 2001), there is widespread consensus among many researchers concerning the latent nature of many vulnerability characteristics. This is particularly the case with "unseen" genetic or biological factors that may predispose to disorder, but it also includes more psychologically based vulnerability processes.

Risk and Diatheses/Vulnerability

Terms such as *risk* and *vulnerability* (or *diatheses*) are often used interchangeably, and in fact there is little doubt that these constructs overlap substantially. However, it is important to note that although we use the terms *diathesis* and *vulnerability* interchangeably, we do not view vulnerability and risk as interchangeable. As several investigators have argued (e.g., Ingram et al., 1998; Luthar & Zigler, 1991; Rutter, 1987), *risk* describes factors that are associated, or correlated, with an increased likelihood of experiencing a disorder. Nevertheless, the presence of risk suggests only an increased probability of the occurrence of a disorder; it does not specify what causes the disorder. Risk factors are thus not informative about the actual mechanisms that bring about a state of psychopathology. For example, female gender is a well-established risk factor for many disorders, but this knowledge alone is uninformative about *why* women are more likely to experience a range of disorders. Alternatively, *vulnerability* is usually defined in such a way that it reflects statements about causal mechanisms.[1] Risk is certainly an important predictive variable that can be seen as acting in concert with vulnerability (Rutter, 1988), but these constructs are not synonymous.

DIATHESIS-STRESS ORIGINS CONSIDERED

To understand fully diathesis-stress interactions, it is useful to briefly consider the historical context in which these ideas emerged. Monroe and Simons (1991) note that the diathesis concept has a long history in medical terminology. The concept dates back to

the ancient Greeks; the word *diathesis* derives from the ancient Greek idea of disposition, which is related to the humoral (body fluids) theory of temperament and disease (Zuckerman, 1999). By the 1800s, the term had become part of the psychiatric language of the day (e.g., Beard, 1881). Likewise, although the role of stress had long been considered an important factor in the development of mental disorders, it was theories of schizophrenia proposed during the 1960s (e.g., Meehl, 1962) that highlighted stress and brought the diathesis and stress concepts together. More specifically, the particular terminology of the diathesis-stress interactions was developed by Bleuler (1963) and Rosenthal (1963).

Beyond these pioneering approaches, somewhat more contemporary and detailed conceptualizations of the nature of vulnerability and the role of stress have been proposed that specify under what circumstances a disorder will ensue. For example, Audy (1971) suggested that the preservation of health requires the maintenance of a dynamic equilibrium against insults coming from chemical, physical, infectious, psychological, and social environment factors. A disorder occurs when the equilibrium is disturbed by an inability to maintain homeostasis. Vulnerability factors influence the ease and frequency with which these factors will challenge homeostasis; such factors therefore determine the probability that the disorder will occur. Thus, the highly vulnerable person is one in whom numerous circumstances can elicit an episode.

GENERAL PRINCIPLES OF DIATHESIS-STRESS MODELS

According to Monroe and Simons (1991) and Monroe and Hadjiyannakis (2002), most diathesis-stress models of psychopathology suggest that all people have some level of predisposing factors (diatheses) for any given mental disorder. However, individuals have their own point at which they will develop a given disorder, a point that depends on the interaction between the degree to which these risk factors exist and the degree of stress experienced by the individual. Because diathesis-stress models address the interactions between premorbid risk factors and situational stressors, they are useful for describing who will develop a disorder and who will not. Many—perhaps most—psychopathologists have recognized the conceptual and empirical utility of combining diathesis and stress constructs, and accordingly, models of psychopathology tend to be explicit diathesis-stress models.

A variety of diathesis-stress models have been proposed for various types of psychopathology (see Ingram & Price, 2001). Depending upon the particular theory, these models suggest specific variables that combine in some fashion to produce the disorder. Beyond the description of particular variables in particular disorders, however, these ideas about psychopathology also illustrate different ways that the structure of a diathesis-stress interaction can be conceptualized. Examination of these models suggests several general principles that characterize hypothesized diathesis-stress interactions.

Additivity

On the surface, diathesis-stress models represent straightforward, linear, dose-response–type relationships, or additive relationships. Hence, at the most basic level, many models suggest that whether or not a disorder will develop depends on the combined effects of stress and the loading of the diathesis. One model, for example, may suggest that relatively minor stressors may precipitate the onset of the disorder for a person who is highly vulnerable, whereas another model might suggest that a major stressful

event might cause a similar reaction in a person low in vulnerability. Although various models may accord a stronger role for one component over the other, this idea presupposes *additivity,* that is, the idea that diatheses and stress add together in some way to produce the disorder.

Ipsative Models

Monroe and Hadjiyannakis (2002) note that many diathesis-stress models reflect an ipsative approach to the relationship between the constructs. Ipsative models posit an inverse relationship between factors such that the greater the presence of one factor, the less of the other factor is needed to bring about the disorder. Ipsative models are not necessarily distinct from additive approaches and can thus be considered an additional quality of many diathesis-stress models of psychopathology. More specifically, these models suggest that the diathesis and stress sum together to cause psychopathology, and that whatever this sum is, it reflects an inverse relationship. Thus, the degree of effect of diathesis or stress can be offset or compensated by the other in the summation that is needed for psychopathology.

Mega Diathesis-Stress Models

Although ipsative (and additive) models are prevalent, Monroe and Hadjiyannakis (2002) also note that other possibilities exist. One such possibility is a model that suggests that disorder results from the combination of significant life stress *and* a heightened vulnerability. For the sake of simplicity, we refer to this as a *mega* diathesis-stress model to denote that both the diathesis and the stress must be considerable before a disorder occurs. Thus, cognitive models of depression that conceptually rely on diathesis-stress interactions would suggest that not only is the presence of a depressogenic schema needed, but substantial life stress must *also* occur before the process eventuates in depression. This differs from an ipsative model, which suggests that minimal stress is needed for depression to occur in individuals with a strongly depressogenic schema.

Static Versus Dynamic Diathesis-Stress Relationships

Comparison of ipsative and mega models reveals a neglected aspect of many diathesis-stress models of psychopathology, specifically the idea that the relationship between the diathesis and stress can change over time. This changing interaction can be illustrated by reference to the idea of kindling. In response to data showing that repeated episodes of depression within some individuals begin to appear with decreasing stress, Post (1992) proposed the idea of kindling. Kindling suggests that repeated instances of a disorder cause neuronal changes that result in more sensitivity to stress. With heightened sensitivity, less stress becomes necessary to activate the requisite processes that lead to psychopathology. Applying these ideas to diathesis-stress models suggests that the precise relationship between these constructs is not necessarily static. More specifically, this also suggests that as the relationship changes with recurrence or relapse, mega processes may become more ipsative. That is, whereas the mega model suggests that high levels of both stress and diatheses are needed, the kindling theory suggests that at some point diatheses are changed (and presumably strengthened) so that less stress becomes necessary to activate the vulnerability factors. Of course, other changes are also possible. Recall that we noted that in at least some models, diatheses are viewed as stable although not necessarily immutable. It is therefore possible that the relationship between diatheses and stress may change if the diathesis becomes weaker.

We believe that consideration of the static versus dynamic relationship between diatheses and stress has potentially important implications for the conceptualization of diathesis-stress ideas. In general, varying relations between diatheses and stress models over time may affect the accuracy of the model at any given moment, but they may also have considerable implications for models that seek to understand the function of these processes in remission, recovery, relapse, and recurrence. As we have noted, few contemporary models, at least explicitly, take into theoretical account potential changes over time of the relationship between diatheses and stress, but clearly the nature of the relationship over time is an important factor to consider.

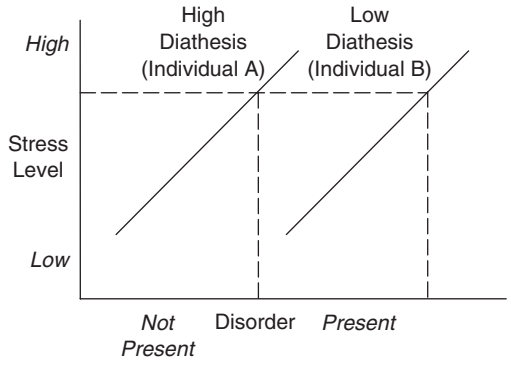

Figure 2.1 Additive Model of Diathesis-Stress Interaction With a Dichotomous Diathesis for Disorder

DIATHESIS-STRESS MODELS

With the caveat that dynamic relationships may be quite significant, our focus is on more "static" models of the diathesis-stress processes. At present, most diathesis-stress models are ipsative, although several permutations are possible. Hence, different investigators have described these models in somewhat varying terms. The models we describe here illustrate these different terms. In particular, we discuss the *interactive model with dichotomous diatheses,* the *quasi-continuous diathesis models, threshold models,* and *risk-resilience continuum models*. Before doing so, it is important to acknowledge that these models tend to vary in emphasis rather than in basic structure. Thus, there is considerable overlap in how these approaches view the relationship between diatheses and stress.

Interactive Model With Dichotomous Diatheses

As noted, vulnerability-stress models originated from schizophrenia theory and research, starting with Meehl's (1962) groundbreaking ideas. In his first model of schizophrenia, Meehl described the diathesis as a single dominant "schizogene," which produces a schizotaxic brain pathology (e.g., neural integrative defect) that eventuates in a schizotypic personality. According to Meehl, however, only some people with schizotypic personality will develop clinical schizophrenia. Most at-risk individuals will not, because the schizotypic personality, although necessary for the development of schizophrenia, is not sufficient in and of itself for the development of schizophrenia. Instead, an environmental stressor is required to produce schizophrenia. Meehl suggested that the stress produced by a schizophrenogenic-type mother who is "ambivalent and inconsistently aversive to the schizotypic" is the most important type of stress that may produce the disorder. Alternatively, if the schizogene is absent, no amount of stress or type of rearing will produce schizophrenia. In sum, Meehl's theory suggested that the onset of schizophrenia is a joint function of both biological and psychological factors.

Meehl's (1962) first model for schizophrenia, which arguably launched the idea of diatheses and stress, can thus be described as an interactive model with dichotomous diatheses (see Figure 2.1). *Dichotomous diathesis* suggests that one either has the diathesis or

does not; if the diathesis is absent, there is no effect for stress. Hence, even severe stress will not lead to the development of the disorder. On the other hand, when the diathesis is present, the expression of disorder will be conditional on the degree of stress. That is, as stress increases, so does the risk for the disorder in those who possess the diathesis.

We note Meehl's (1962) original model for historical context, but it is also important to note that this model has been updated to the extent that it no longer resembles the earlier model. Hence, to better clarify the interaction between diathetic characteristics and environmental stressors, Meehl (1989, 1990) revised his original model to describe another pathway that could lead to schizophrenia, called the SHAITU genophenocopy (Meehl, 1989, 1990). SHAI stands for personality trait extremes—submissive, hypohedonic, anxious, and introverted—of polygenic origins, which may increase the potential for schizotaxia to develop into schizotypic personality and subsequently lead to clinical schizophrenia. TU stands for environmental risk factors; T stands for major or frequent minor traumas during development, whereas U stands for unlucky events in adult life, which also increase the risk for schizophrenia. In Meehl's original 1962 model, the dominant schizogene and the resulting schizotaxic brain pathology were necessary but not sufficient causes of schizophrenia. In contrast, the SHAITU genophenocopy not only plays a role in the schizotaxic type of schizophrenia, but it can produce a schizophrenic disorder even in the absence of the schizogene. As such, however, the revised diathesis-stress model is no longer an example of an interactive model with dichotomous diathesis conceptualization.

Although Meehl's (1962) original model of schizophrenia illustrates the idea of an interactive model with dichotomous diathesis, a more contemporary example can be seen in the posttraumatic stress disorder (PTSD) theory proposed by McKeever and Huff (2003). They suggest two types of diatheses: One type consists of ecological variables and revolves around factors such as child abuse and cognitive distortions. Another type is biological and includes variables such as neurophysiological dysregulation. Individuals with higher degrees of these premorbid vulnerability factors (diatheses) would not need to experience as severe a stressor to reach the threshold and develop PTSD symptomatology. In contrast, individuals without the diatheses might not display any signs or symptoms of PTSD even after experiencing a traumatic event. Even to the extent that signs or symptoms are experienced, however, they would not be indicative of a clinical disorder.

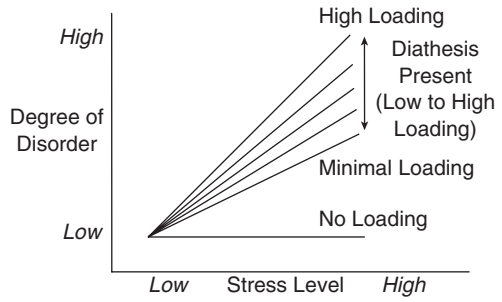

Figure 2.2 Interactive Model of Diathesis-Stress Interaction With Quasi-Continuous Diathesis

Quasi-Continuous Diathesis Models

Dichotomous models suggest that when the diathesis is absent, there is no effect for stress. That is, regardless of the amount of stress experienced by the individual, the disorder will not occur if the individual does not have the diathesis. However, many disorders suggest polygenic models that allow for varying degrees of diatheses (such as the level of a particular neurotransmitter) (Zuckerman, 1999). Thus, instead of being dichotomous, the diathesis is "quasi-continuous" (Monroe & Simons, 1991). As illustrated in Figure 2.2, in the quasi-continuous model there is a point beyond which a disorder

will occur, but there is also a continuous effect of the diathesis once the threshold is passed. In other words, a very minimal level of diathesis may be insufficient to produce the disorder even under high stress, but the probability of disorder increases as a function of both level of stress and strength of the diathesis beyond a minimal level (Zuckerman).

Few models of psychopathology are explicitly framed in terms of a continuous or quasi-continuous vulnerability model, but it is easy to see how this diathesis-stress conceptualization could be applied to psychopathology models. Moreover, this idea could also help clarify or refine these models. For example, schema models of depression are typically conceptually stated as dichotomous models; if the individual possesses a depressogenic schema, then he or she is at risk for depression when events occur that activate this schema (see Beck, 1967, for the original description of the role of depressogenic schemata in depression). However, various discussions of the properties of schemata suggest how schemata could be conceptualized in more continuous terms (e.g., the relative density and strength of negative connections; see Segal, 1988). Some descriptions of these processes have been implicitly, but rarely explicitly, suggested (see Ingram et al., 1998). To the extent that schemata could explicitly be considered to represent a more continuous variable, such that some individuals may possess schemata that are "strongly" depressogenic, whereas others may possess only "weak" or mild depressive schemata, then a more continuous diathesis-stress model may not only be applicable to depression, but may also suggest refinement of key elements of the theory that were not previously considered.

Threshold Models

Some models suggest that the synergism between the diathesis and stress yields an effect beyond their combined separate effects (Monroe & Simons, 1991; Rothman, 1976). Moreover, complex diathesis-stress models that represent additive and interactional relationships between variables, as well as threshold effects for the diathesis, have also been proposed (Monroe & Simons). These ideas can be illustrated by what we would term a *threshold model.*

To illustrate a threshold model, consider the integrative model of schizophrenia proposed by Zubin and Spring (1977). Zubin and Spring suggest that *every* person has a degree of vulnerability that represents a threshold for the development of schizophrenia. At the most basic level, this model suggests that as the intensity of the trauma (stressor) increases, so too do the risks for psychopathology. The diathetic threshold is the point at which the people who fall below the threshold will not develop the disorder, whereas those above this level cross the threshold into disorder (see Bebbington, 1987; Monroe & Simons, 1991). Thus, the threshold for triggering schizophrenia may vary from one person to the next depending on the degree of vulnerability and the level of stress experienced. For a person who is highly vulnerable, relatively minor stressors may cause the threshold to be crossed. On the other hand, a major stressful event might cause a similar reaction even for a person low in vulnerability.

Risk-Resilience Continuum Models

Invulnerability, competence, protective factors, and *resilience* are terms often used to describe the opposite of vulnerability (Ingram & Price, 2001). Resilience can be thought of as factors that make a person resistant to the deleterious effects of stressors. Examples of resilience features could include particular personality traits, social skills, and coping responses. Resilience and vulnerability represent, therefore, opposite ends of a vulnerability continuum, although models typically do not specify if resilience simply reflects the lack of vulnerability

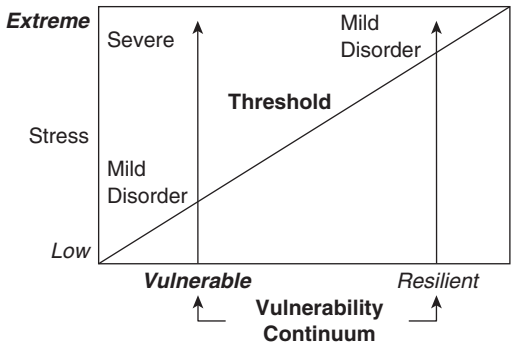

Figure 2.3 The Vulnerability-Resilience Model

factors or instead encompasses specific factors that confer resilience.

As in other models, the diathesis continuum interacts with a continuum of stress to produce the possibility that a disordered state will occur. At the most extreme vulnerability end of the spectrum, little life stress is necessary to trigger disorder. At the resilient end, however, a great deal of stress is needed before psychopathology develops. A vulnerability-resilience relationship is presented in Figure 2.3. As this figure illustrates, with decreasing resilience, and hence increasing vulnerability, the probability that stress will result in a disorder increases. Conversely, when resilience increases, the risk of disorder goes down but does not vanish entirely. That is, resilience may be the opposite of vulnerability, suggesting a resistance to disorder but not immunity from it entirely (Ingram et al., 1998). Of course, although not specified by most models, the idea of resilience can easily be incorporated into diathesis-stress interactions.

Like a threshold model, a risk-resilience model also notes a threshold at which a particular disorder will be encountered. However, this model can also take into account the severity of psychopathology that is experienced. Hence, even the most resilient people can be at risk for developing significant symptomatology with enough stress, although the symptomatology will likely be less severe than that experienced by individuals who experience stress and who are vulnerable. On the other hand, highly vulnerable people who encounter significant stress are proposed to experience a more severely disordered state. This model therefore takes into account not only the continuum of vulnerability, ranging from vulnerable to resilient, but also a continuum of disorder severity.

SOME ISSUES FOR CONSIDERATION IN DIATHESIS-STRESS MODELS

While maintaining the same basic structure, the models we have described reflect the different approaches that investigators have taken to understanding psychopathology. Beyond these basic models, there are, however, a number of issues that need to be considered as efforts continue to more fully understand how diatheses and stress interact to produce psychopathology. Although artificial in some respects, for discussion purposes we divide these into diathesis issues and stress issues.

Diathesis Issues in Diathesis-Stress Models: Single Versus Multiple Diatheses Factors

For models suggesting genetic diatheses, evidence of the polygenic aspect of psychopathological disorders suggests a combination of genes that may be required for disorder. Thus, individuals inheriting any particular gene defect will be normal if they do not possess the other gene defects needed to produce a disorder such as schizophrenia. Of course, genetic diatheses are not the only approach to understanding psychopathology. Hence, models featuring psychosocial factors may also need to highlight more than one diathetic factor. For example, an interpersonal and cognitive model of depression (Gotlib & Hammen, 1992) extended to vulnerability would need to specify the multiple diathesis factors that fall into their respective cognitive and interpersonal categories, and the link

between them, to provide a more complete model. Likewise, models that highlight biological and psychological factors (e.g., Goodman & Gotlib, 1999) would also need to specify the link between various processes and how they would work in concert to produce the disorder when life stress is encountered.

Development and Stress Issues in Diathesis-Stress Models

The nature of the diathesis-stress interaction described in various models is often ambiguous (Monroe & Hadjiyannakis, 2002). As we have noted in our description of schema models of depression, diatheses are often portrayed as discontinuous and categorical (people either have a given diathesis or they do not). Alternatively, stress is frequently portrayed as nonspecific and continuous, varying only in degree but not in type. However, diatheses can often become continuous once a certain threshold has been reached. Moreover, because diatheses and stress are rarely completely independent of each other, the interactions between the diathesis and stress can be quite complex (Monroe & Simons, 1991).

Early formulations of the diathesis-stress model were based on biological factors (e.g., Meehl, 1962) that inferred temporal precedence and assumed that the diathesis was inactive in the developmental scheme of things. Thus, the interpretation of a significant interaction seemed relatively clear-cut: Stress activated the diathesis, which in turn brought about the onset of disorder. This interpretation suggests that until the diathesis is activated by stress, the diathesis is inconsequential. The complementary influence of the diathesis on stress was typically disregarded in these early models (Monroe & Simons, 1991).

The influence of diatheses on stress also received little attention in early formulations of this relationship, but there are several ways in which the constructs may not be as independent as they seem at first glance. For example, it may be that likelihood of incurring a stressor increases with the loading of the diathesis. To the extent that the diathesis influences the incidence of the requisite forms of stress, the more likely it is that highly predisposed people will develop a disorder. For a young person in the early stages of schizophrenia onset, for example, abnormal or socially withdrawn behavior that results from diathesis may create tension in the young person's interpersonal life at home and at school. The additional interpersonal stress, which is directly influenced by the diathesis, may exacerbate stress and subsequently increase the likelihood of the onset of full-blown schizophrenia.

This idea is similar to proposals regarding stress generation. As we have previously noted, stress is typically seen as operating externally to the individual, although it does appear that at least some people may also play a part in creating the stressful environment that acts to trigger pathology. That is, the diathesis may influence the manner in which a person deals with life and thus the nature of the stressors to which he or she is exposed. Indeed, a number of researchers have argued that many stressors may constitute the results of one's own actions (Depue & Monroe, 1986; Hammen, 1991, 1992; Monroe & Simons, 1991; Rutter, 1986). For example, people who have doubts about their self-worth may seek reassurance in an effort to counter these doubts (Luxton & Wenzlaff, in press), but repeated efforts may result in rejection from others, therefore precipitating a depressive disorder. Beyond the exacerbation of stress that may occur as the result of the emergent activation of diatheses, vulnerable individuals may thus play a role in creating their own stresses, which may then activate the diatheses and precipitate disorder.

Some models have proposed that the vulnerability factor itself affects the perception of stress (e.g., Zubin & Spring, 1977), suggesting that stress is not independent from vulnerability. In this sense, the vulnerability

does not "cause" the stress in this case, but rather the vulnerability is part of the stress. In other cases, stress may affect the development of the diathesis. For example, there is evidence to suggest that stress may play a role in the etiology of schizophrenia as early as the prenatal period, when the fetus is exposed to a possible range of developmental insults that in turn produces the diathesis (Brennan & Walker, 2001). In depression theory and research, the "scar" hypothesis (Rohde, Lewinsohn, & Seeley, 1990) suggests that a first episode of depression may leave cognitive scars in the form of negative thinking patterns that may not have been previously present. If such scars subsequently serve as a diathesis for additional episodes of depression, then this may be understood as a stress-induced diathesis.

Not only may a disorder be the result of both diathesis and stress, but the diatheses may precipitate stress that combines with stress not related to the diatheses (Monroe & Simons, 1991). For example, "external" stressors (e.g., a death in the family, alcoholic parent, socioeconomic strife) may or may not be substantial enough to trigger the disorder. However, if the diathesis plays a direct role in creating "other" kinds of stress by, for example, increasing tension in the person's interpersonal life, the combined state of affairs may subsequently increase the likelihood of the onset of a full-blown disorder.

Typically, diathesis-stress models refer to stressful events that are proximal to the onset of disorder. However, it should be noted that stressors earlier in life may also influence how later stressful events are responded to and thus increase future susceptibility to disorder. For example, maladaptive methods of coping with stress in childhood and throughout development may be detrimental to the development of effective coping competencies; lacking effective coping skills, in turn, can compromise resilience and encourage vulnerability (Hammen, 1992). Thus, maladaptive cognitions about the self and others and ineffective coping competencies may contribute to the occurrence of stressful events and circumstances—and these in turn may trigger depressive reactions.

SUMMARY AND CONCLUSIONS

Individually, vulnerability and stress are important concepts, but their real power lies in their interaction. Diathesis-stress models thus describe the interactions between these constructs and are useful for understanding the development of psychopathology. In this chapter, we described some basic principles that characterize diatheses-stress models, such as the idea that models tend to be additive and ipsative. We also noted that mega diathesis-stress models are also possible, although uncommon, and emphasized the importance of considering varying relationships between diatheses and stress over time. We also described the interactive model with dichotomous diatheses approach to diathesis-stress conceptualizations, the quasi-continuous diathesis model, threshold models, and risk-resilience continuum models. In describing these different models, however, we also noted that these models tend to vary not in basic structure but rater in the emphasis that different investigators give to different components. Finally, within the context of diathesis-stress interactions, we noted some outstanding issues that reflect on conceptualizations of diatheses and conceptualizations of stress.

The development of psychopathology is obviously complex and involves numerous vulnerability factors and interactions between those factors and stress. Diathesis-stress models are excellent heuristic devices (Monroe & Simons, 1991) that enable us to potentially understand how predispositional factors from various domains may increase susceptibility to psychopathology and subsequently create the sufficient conditions for the onset of disorder. Furthermore, diathesis-stress models

help describe how diatheses and stressors can be better conceptualized and more precisely measured empirically with respect to specific forms of psychopathology. Such models are necessary if psychopathologists ever hope to be able to understand the multifactoral complexity of psychopathology, including developmental experiences, biological vulnerabilities, psychological susceptibilities, and socioenvironmental variables.

NOTE

1. Because possessing vulnerability places one at higher risk for developing a disorder, vulnerability is probably most accurately seen as a subcategory of risk.

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