CHAPTER 1

Understanding Psychopathology

THE ROLE OF VULNERABILITY

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We believe there is no more important goal in psychopathology research than understanding the causes of psychopathology. Although there may be multiple pathways to such an understanding, theory and research on vulnerability are indispensable to this quest for causality. In a broad sense, it is difficult to envision an effective effort to understand the causes of disorder that does not include an examination of the processes that give rise to the disorder. Even more broadly, a case can be made that efforts to understand vulnerability to psychopathology underlie virtually all efforts to understand psychopathology itself.

Theory and research related to a number of different psychopathological conditions are examined in the various chapters in this book. Each of these examinations focuses on the specific vulnerability theory and data that are relevant to particular disorders. We start here, however, with a broader examination of the idea of vulnerability that can serve as a foundation for understanding vulnerability in these more specific disorders. In this vein, we start by briefly examining what is arguably the single most important aspect of psychopathology, that is, the concept of causality in psychopathology. We follow with a discussion of the notion of vulnerability itself and then move to issues concerning the relationships among vulnerability, risk, and resilience, and then finally to issues concerning the distinction between childhood and adulthood.

WHAT IS CAUSALITY?

In the most obvious sense, "causality" refers to the processes that create or facilitate the transition from a normal state of psychological functioning into an abnormal psychological state. Although this concept of causality is accurate, it is incomplete; causality does not refer simply to this onset phase but also to other important processes in the course of psychopathology. As such, a more complete examination of causality necessitates some discussion of both onset and maintenance processes.

Onset Causality

Whether a first onset of a disorder or the occurrence of a subsequent episode of a disorder, understanding the processes involved in onset are critical to understanding the causes of psychopathology. From a vulnerability perspective, data on these processes inform researchers about the factors that place individuals at risk for experiencing a disorder. Similarly, data have also provided insights into how these risk processes in the nondisordered person are translated into a psychopathological state in that same person.

Onset can be further understood in the context of distal and proximal vulnerability. Although investigators differ to some extent in drawing the temporal lines for these different risk processes, proximal factors are generally regarded as those that become apparent right before the onset of a disorder. Distal factors, however, occur before the disorder but more distant in time from its appearance. For example, a model of a psychopathological state that specified certain psychological or physiological responses to life events would be specifying a more proximal cause, whereas a model that focused on the creation of risk factors in childhood would be focusing on more distal variables.

Maintenance Causality

Some researchers have differentiated between the onset and the maintenance of a disorder and have tacitly suggested that the onset or appearance of psychopathology is synonymous with causality. Correspondingly, maintenance processes are not viewed as causal, and hence relatively little importance is ascribed to these factors (Ingram, Miranda, & Segal, 1998). We argue, however, that causality is not synonymous solely with onset and that the factors that maintain a disorder can be legitimately seen as causal.

We thus suggest that an exclusive focus on onset is too narrow a conception of the construct of causality. Consider the case of depression. A considerable amount of data shows that depression is a persistent disorder, with symptoms lasting months and in some cases years (e.g., dysthymia). Moreover, data also show that untreated depression lasts between 6 months to 1 year or, depending on the severity of the episode, possibly up to 2 years (Goodwin & Jamison, 1990). Unless a model argues that the factors that lead to the onset of a disorder are identical to the factors that maintain the disorder (and few

even address this idea), the processes associated with the perpetuation of psychopathology can be considered to have real causal significance. Indeed, a case might be made that such factors are more meaningful than onset factors—that is, the psychological, interpersonal, occupational, academic, and perhaps biological damage associated with a disorder like depression arises not because it occurred but rather because it persists over weeks, months, and perhaps years.

Although we focus on depression in this example, virtually all psychopathological states are problematic not only because they occur but also because they are maintained over time. We can therefore legitimately ask whether the causes of this occurrence over time are any less important than the causes of its initial appearance. Thus, we argue that fully understanding vulnerability requires investigating not only the factors that bring about a disordered state but also those that result in its continuation over time.

HOW DO WE DEFINE VULNERABILITY?

It seems reasonable to assume that a volume on vulnerability to psychopathology should offer, at a minimum, some ideas on how to define the vulnerability construct. Although the vulnerability approach to psychopathology is at least several decades old, little consensus has been reached on what constitutes an adequate definition of "vulnerability" (Ingram et al., 1998). This definitional shortcoming persists even though ideas about vulnerability have generated a significant body of theory and data. Such a corpus of knowledge is possible because we arguably know vulnerability when we see it and because researchers can identify demonstrably vulnerable groups to study. For example, data show that people who have experienced a disorder are at greater likelihood of experiencing another disorder, and hence investigators can assemble such groups to study vulnerability. Yet, these operational definitions leave aside the broader question about what constitutes the vulnerability construct itself.

Of course, simple definitions of "vulnerability" abound, and for the public at large such terms are quite appropriate. For example, those who are vulnerable are liable to, or susceptible to, psychopathology. From a scientific standpoint, however, exchanging one poorly defined term for another is an unsatisfactory means of appreciating the nature and complexity of the vulnerability construct. Although truly comprehensive definitions of vulnerability are rare, one can derive a conceptual understanding of this construct by examining its core features, that is, those features that have been examined in theory and research and that can therefore offer important clues about its nature (Ingram et al., 1998).

Core Features of Vulnerability

Examination of the literature suggests that several themes appear repeatedly in discussions of vulnerability, which focus on vulnerability as a stable trait,

the endogenous and latent nature of vulnerability, and the role of stress in actualizing vulnerability. We now turn to a brief examination of each theme.

Vulnerability as a Stable Trait

Researchers frequently discuss vulnerability as an enduring trait. Zubin and Spring (1977) pioneered many of the ideas about vulnerability in their work on understanding causal processes in schizophrenia. Zubin and Spring were also the most specific about what they regarded as the trait-like nature of vulnerability: "We regard [vulnerability] as a relatively permanent, enduring trait" (p. 109); "the one feature that all schizophrenics have … is the everpresence of their vulnerability" (p. 122). They leave little doubt that "vulnerability" refers to processes that endure over time.

Other investigators have tended to be not quite as specific, but the trait-like nature of vulnerability is nevertheless implicit in many of their discussions of vulnerability. Such assumptions of permanence are likely rooted in the genetic level of analysis employed by researchers who pioneered this concept. For example, many schizophrenia researchers emphasize the genetic endowment of individuals who are at risk for this disorder. Meehl's (1962) pioneering concept of schizotaxia represents an inherited neural deficit, whereas other influential researchers such as Zubin and Spring (1977) and Nicholson and Neufeld (1992) are quite explicit that genetic factors determine the relative level of vulnerability (at least for schizophrenia).

Permanence, however, need not be rooted in genetic factors. An example from the schizophrenia literature is again illustrative. Researchers have suggested that prenatal stress or trauma may lead to vulnerability to schizophrenia (Brennan & Walker, Chapter 14, this volume). For example, both maternal influenza and significant famine have been linked to a rise in the rate of schizophrenia. In regard to the latter example, a two-fold increase in schizophrenia was subsequently reported following a massive famine in China between 1959 and 1961 (St. Clair et al., 2005). Postnatal factors, such as exposure to environmental toxins, have also been implicated (Brown, 2007), which may interact with genetic liabilities to render the vulnerability even more permanent.

Such conceptualizations tend to posit that no decrease in absolute vulnerability levels is possible. This is not to suggest, however, that functional vulnerability levels cannot be attenuated by several factors, such as those that affect neurochemistry. For instance, medications such as lithium carbonate, which alters the likelihood of developing the symptoms of a bipolar episode by presumably controlling the neurochemistry of the underlying vulnerability, may prove helpful. Similar diminishment of functional vulnerability may be seen in the actions of psychopharmacological treatments for depression with medications such as the various generations of tricyclic agents and the more recent selective serotonin reuptake inhibitors (Potter, Padich, Rudorfer, & Krishnan, 2006; Shelton & Lester, 2006). Even though functional vulner-

ability may be altered and individuals are less likely to develop the disorder, the vulnerability persists; for example, in many cases the probability of experiencing a psychiatric episode is increased if the medication is discontinued. Thus, even though the vulnerability may be controlled, the vulnerability trait itself remains.

The trait-like nature of vulnerability is perhaps most clearly seen in contrasting it to the episodic nature of psychological disorders. For instance, Zubin and Spring (1977) clearly distinguish between an enduring vulnerability trait and episodes of schizophrenia that "are waxing and waning states" (p. 109). Hollon, Evans, and DeRubeis (1990) and Hollon and Cobb (1993) also distinguish between (1) stable vulnerability traits that predispose individuals to the disorder but do not constitute the disorder and (2) state variables that represent the occurrence of the symptoms that reflect the onset of the disorder. Thus, while predisposing factors are enduring traits, virtually all investigators characterize the disorder itself as a state. Disordered states can therefore emerge and decline as episodes cycle between occurrence and remission, but the traits that give rise to vulnerability for the disordered state are typically thought to remain constant.

Although vulnerability is assumed by many theorists, particularly those working within a genetic framework, to be permanent and enduring, this need not be the case. This is especially true when the level of vulnerability analysis is psychological rather than genetic or prenatal in nature. As we have noted, assumptions of genetic vulnerability offer little possibility for the modification of vulnerability characteristics. Many psychological approaches, however, rely on assumptions of dysfunctional learning as the genesis of vulnerability. Given these assumptions, not only functional but actual vulnerability levels may fluctuate as a function of new learning experiences that influence the particular vulnerability factor. For instance, Hollon, Stewart, and Strunk (2006) have summarized data showing that, compared to pharmacotherapy for depression, cognitive therapy is more effective in preventing relapse and recurrence, presumably because the underlying vulnerability has been at least partially altered. In this vein, Hollon et al. (1990) and Hollon and Cobb (1993) argue that the effects of pharmacological treatments may be largely symptom-suppressive but that psychological interventions such as cognitive therapy are designed to alter dysfunctional cognitive structures and, to the extent that genuine vulnerability is rooted in such structures, may lessen susceptibility to psychopathology. Fewer recurrences of the disorder over time may reflect decreased vulnerability. It is certainly possible that factors other than vulnerability reduction may be at the heart of cognitive therapy's prophylactic effects, but this example does illustrate how, theoretically at least, actual vulnerability levels might be altered.

Of course, from the viewpoint of a psychological level of analysis, vulnerability may decrease with certain corrective experiences, or, alternatively, it may increase over time. This latter possibility would be the case if continued exposure to aversive experiences and stressful life events served the function

of enhancing the factors that contribute to vulnerability. Some perspectives have suggested that frequent experiences with the disorder itself may increase vulnerability for future onsets. For example, in describing the idea of kindling, Post (1992, 2007) has proposed such a process in the area of affective disorders. Post suggests that each episode of an affective disorder leaves a residual neurobiological trace that leads to the development of pathways by which increasingly minimal stress becomes sufficient to activate the mechanisms that result in a disorder. Such a process thus leads to increased vulnerability.

The possibility that psychological vulnerability levels can be altered (up or down) suggests a subtle but potentially important distinction between stability and permanence. Stability and permanence are likely to be viewed as synonymous. However, even though the concept of stability clearly suggests a resistance to change, it does not presume that change is never possible. Under the right circumstances, changes in an otherwise stable variable may very well occur. Indeed, the entire concept of psychotherapy is based on precisely this premise. Without intervention or the introduction of other significant life experiences, however, little change in stable psychological variables should occur. On the other hand, variables that are considered to be enduring, particularly biological processes emanating from genetic or prenatal processes, imply a permanence or immutability that is not only resistant to change under ordinary circumstances but is assumed to offer virtually no possibility of change.

Vulnerability as Endogenous and Latent

Another core feature that is possible to glean from vulnerability research is that vulnerability represents an endogenous variable. This is perhaps most clearly seen in genetic conceptualizations of vulnerability, but it is equally relevant for psychological conceptualizations. That is, whether stemming from inborn characteristics or acquired through learning processes, the vulnerability resides within the person. This aspect can be contrasted to other levels of analysis that might, for example, focus on environmental or external sources of stress that initiate a disorder, or perhaps a focus on interpersonal styles that may lead to aversive interactions (see Joiner & Coyne, 1999). We discuss this distinction more fully in the section differentiating vulnerability from risk. For now it is important to note that, although these "external" variables are clearly important, the locus of vulnerability processes is within the person.

In line with the idea that vulnerability is an endogenous process, and that vulnerability remains stable even though observable states of psychopathology arise and then (in many cases) diminish, some investigators have suggested that vulnerability is not easily observable and can thus best be conceptualized as a latent process. From a research perspective, this feature can perhaps be seen most clearly in the empirical search for observable markers of vulnerability; numerous investigators have sought to find reliable empirical indicators of the presence of the vulnerability. There are a variety of research strategies for identifying markers, but in each case they operate with the assumptions

that (1) vulnerability processes are present in individuals who have few or no outward signs of the disorder, (2) these processes are causally linked to the appearance of symptoms, and (3) they are not readily observable and are therefore difficult to assess. This sense of latency is particularly the case in investigations that rely on some kind of stressful or challenging event that makes detection of the vulnerability factor possible (see Shelton, Hollon, Purdon, & Loosen, 1991, for a discussion of the challenge paradigm as it pertains to the conceptualization of vulnerability and dysregulation). The search for vulnerability indicators is thus the search for predictors of the disorder in the absence of symptoms of the disorder, an empirical strategy reflecting a conceptual judgment that vulnerability is present and stable but not easily observable—that is, latent.

The Role of Stress

We have alluded to the importance of stress in fully defining vulnerability, and it is therefore important to note that although stress may not be a core feature of vulnerability in the sense that it is stable and endogenous, it nevertheless is an important enough variable to be included within any discussion of the core features of vulnerability.

To comprehensively examine definitions of "stress" would require an entire volume, and indeed while volumes have been devoted to this topic (for classic examples, see Brown & Harris, 1989, and Cohen, 1988). In general, however, stress can be understood as falling into several broad categories. A number of investigators (e.g., Luthar & Zigler, 1991; Monroe & Simons, 1991) note that a major category of stress is conceptualized as the occurrence of significant life events that, in the case of psychopathology, are interpreted by the person as undesirable or aversive. Another kind of stress can be seen as the accumulation of minor events, hassles, or challenges (Lazarus, 1990).

Although the definitions of "stress" may be numerous, we can view stress generally as the life events (major or minor) that disrupt the mechanisms maintaining the stability of an individual's physiology, emotions, and cognitions. Classic descriptions of stress suggest that such events represent a strain on the person's adaptive capability that initiates an interruption of the person's routine or habitual functioning. As such, stress interferes with the system's physiological and psychological homeostasis and is thus seen as a critical variable in a multitude of models of psychopathology (Monroe & Harkness, 2005; Monroe & Simons, 1991), regardless of whether these models focus explicitly on (endogenous) vulnerability factors.

The problems with conceptualizing and adequately assessing stress have been well documented, as have concerns about separating concepts of stress from concepts of psychopathology (e.g., Hammen, 1991; Monroe & Harkness, 2005; Monroe & Simons, 1991). Nevertheless, we argue that at a conceptual level it makes sense to separate stress from vulnerability and psychological disorder. Such a conceptual separation recognizes the possibility that

stress can exist independently of appraisal processes and can be consensually defined and objectively measured; everyone would agree, for instance, that a car accident resulting in permanent confinement to a wheelchair will be stressful for everyone regardless of his or her appraisal processes. Moreover, separation of the stress and vulnerability constructs facilitates communication about the variables potentially operating in psychopathology; that is, it is possible to talk about stress without frequent qualifications attributable to appraisal processes.

The Diathesis–Stress Relationship

By conceptually separating stress and vulnerability, we are better able to examine the diathesis–stress relationship. The diathesis concept has a long history in medical terminology. In tracing this history, Monroe and Simons (1991) note that the concept dates back to the ancient Greeks and as early as the late 1800s was well lodged in the psychiatric vernacular of the day. "Diathesis" signifies a predisposition to illness and has evolved from its original focus on constitutional, biological factors to presently also encompass psychological variables such as cognitive and interpersonal susceptibilities. Moreover, such diatheses are typically considered to be latent and, as we have noted, must be activated in some fashion before psychopathology can occur. In line with this concept, many models of psychopathology are explicitly diathesis–stress models. Thus, although there is general agreement that vulnerability constitutes an endogenous process, most models also recognize that events perceived as stressful act to trigger vulnerability processes that are linked to the onset of the disordered state. In many cases, psychopathology is therefore the interactive effect of the (latent and endogenous) diatheses and events perceived as stressful. Framed within the context of a diathesis–stress conceptualization, stress is integral to virtually all current conceptualizations of vulnerability.

Summary of the Core Features of Vulnerability

In sum, a review of the existing literature suggests a number of essential features that characterize the construct of vulnerability. Perhaps its most fundamental core feature is that vulnerability is considered a trait (rooted in biological and/or psychological processes) as opposed to the kind of a state that more accurately characterizes the actual appearance of the disorder. Despite its trait-like qualities, vulnerability is not necessarily permanent or unalterable (though psychological vulnerability is relatively stable and resistant to change). Corrective experiences can occur that may attenuate the vulnerability, or, alternatively, certain experiences may increase vulnerability factors. In addition, vulnerability is viewed as an endogenous process that is typically conceptualized as latent. Finally, although conceptually distinct from vulnerability, stress is a critical "feature" of vulnerability in that many models postulate that vulnerability cannot be realized without stress. This last feature

of vulnerability represents the essence of the diathesis–stress approach that is common among many current models of psychopathology.

THE RELATIONSHIPS AMONG VULNERABILITY, RISK, AND RESILIENCE

Terms such as "vulnerability" and "risk" (and to a lesser degree "resilience") tend to be used interchangeably. Such usage is understandable; clearly the ideas of vulnerability and risk refer to similar phenomena and share a number of features. Nevertheless, we believe that these terms, and the constructs they represent, are not interchangeable and should be clearly distinguished in any discussion of vulnerability. We therefore examine the associations between vulnerability and risk and vulnerability and resilience, respectively.

Vulnerability and Risk

Vulnerability and risk are not synonymous. We argue, as have others (e.g., Cicchetti & Valentino, 2006), that the concept of risk refers to variables that are empirically associated with a greater likelihood of experiencing a disorder (e.g., poverty and stress as they relate to social injustice). Risk thus serves broadly to predict the likelihood of dysfunction. However, it is not informative about the actual mechanisms of a disorder. That is, risk tells us that someone may develop a disorder but not specifically how or why the disorder occurs. Thus, risk refers to descriptive variables rather than causal ones.

Because risk factors are generally uninformative about the actual mechanisms that bring about a state of psychopathology, knowledge about risk factors is not particularly helpful with regard to specific psychosocial intervention strategies. Presumably the most effective treatment for a disorder targets not only the symptoms of the disorder but also the mechanisms that helped to bring it about, although it should be noted that some authors have argued that the only effective treatment is one that alters broadly defined risk factors. For example, Albee (2000) makes the case that until risk factors such as poverty and social injustice are changed, individual treatment is likely to be ineffectual—akin to using a band-aid to try to stop a hemorrhage.

It should also be noted that, although risk and vulnerability are conceptually separate, these concepts are not necessarily empirically unrelated. In pointing out a similar distinction between vulnerability and risk, Rutter (1987) and Luthar and Zigler (1991) have argued that these variables interact with one another to produce the onset of a disorder. Thus, the person who is "at risk" because he or she lives, for example, in a particularly stressful environment is apt to see this risk realized in disorder if he or she also possesses the vulnerability mechanisms. This is the essence of the diathesis–stress interaction that characterizes numerous models of psychopathology.

We have argued that risk represents a descriptive factor rather than a

causal one, and that as an endogenous factor, only vulnerability can play a causal role. Yet, data have shown that risk variables both can predict the onset of psychopathology and are correlated with vulnerability. Such findings may make it tempting to suggest that risk variables might in fact have causal significance. Rutter et al. (1988), however, cautions against drawing causal inferences solely from risk variables that appear linked to a disorder. To illustrate this point, Rutter (1988) notes findings indicating that test results on a national examination were superior for schools where the children's work was exhibited on the walls (Rutter, Maughan, Mortimore, & Ouston, 1979). This positive association constituted an empirical predictor of better test performance, but few would argue that putting children's work on the wall "caused" the improvement in their test grades. Rather, such behavior was indicative of an enhanced school atmosphere that perhaps had some causal link to better performance. In sum, then, risk is an important predictive variable that tends to operate in concert with vulnerability, but it is uninformative, either theoretically or empirically, about mechanisms. "Vulnerability," on the other hand, is a term that should be reserved for discussion of the mechanisms implicated in the onset or maintenance of a disorder.

Vulnerability and Resilience

"Invulnerability," "competence," "protective factors," and "resilience" are terms used by various investigators to describe the opposite of vulnerability. Each of these terms suggests some level of invulnerability to psychopathology in the face of stress, and although they may reasonably be used interchangeably, some subtle distinctions do exist. For example, invulnerability suggests an absolute level of protection from psychopathology; to the extent that individuals are characterized as invulnerable, the implication is that they will never experience a disorder. "Resilience," on the other hand, suggests that it is difficult but not impossible to experience psychopathology. We thus prefer the concept of resilience over others because it implies a diminished, but not zero, possibility of psychopathology.

A working assumption is that resilience and vulnerability represent different ends of a vulnerability continuum. Such a continuum is seen as interacting with stress to produce the possibility that a disordered state will occur. Thus, at the most extreme vulnerability end of the range, little life stress is necessary to result in a disorder. At the resilient end of the range a great deal of stress will be needed before psychopathology develops. Figure 1.1 represents the vulnerability–resilience relationship. As this figure illustrates, with enough stress even the most resilient people will be at significant risk for the development of symptomatology, although these symptoms will probably be milder than those of the vulnerable person who experiences low to moderate stress and will almost certainly be milder than those of the vulnerable person under significant stress. Resilience thus suggests the opposite of vulnerability and implies a resistance to disorder but not an immunity.

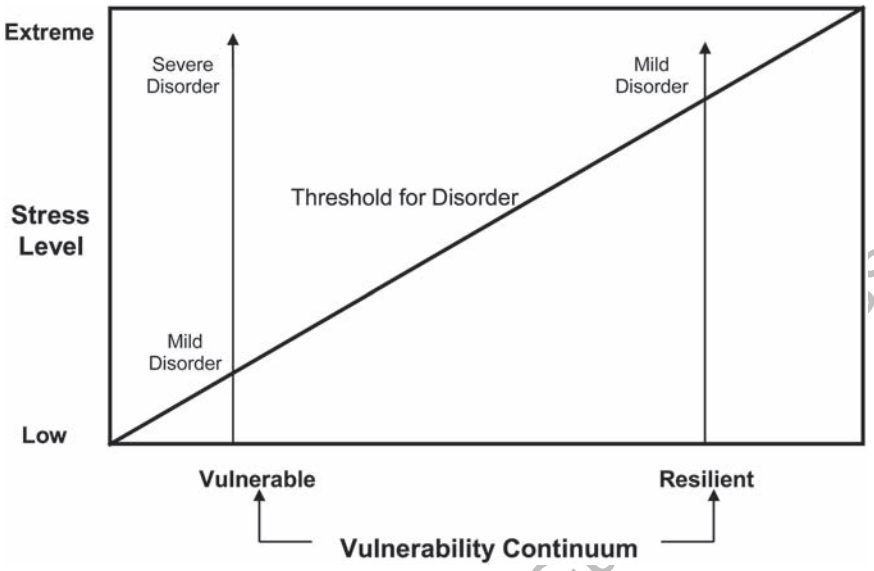

FIGURE 1.1. Representation of a diathesis–stress continuum. When vulnerability is at its highest level, less stress is required to activate psychopathology.

DEFINING CHILDHOOD AND ADULTHOOD: WHERE DO WE DRAW THE LINE?

An examination of vulnerability across the lifespan obviously encompasses vulnerability theory and research on both children/adolescents and adults. This begs the question, however, as to where to draw that line between childhood and adulthood. Although the distinction is clear in many cases (e.g., comparing a 5-year-old child to a 50-year-old adult), differentiating the actual line is significantly more difficult.

There are several approaches to differentiating childhood from adulthood. Perhaps the most simple relies on legal definitions, although even here the distinction is not always clear. For example, in the United States adulthood is legally defined for most behaviors as beginning at age 18. At this age, individuals have virtually all the legal rights and responsibilities of all other adults. Yet, there is at least one notable exception—in most states the right to drink alcohol and to work in settings where alcohol is served is not granted until age 21. Legal definitions within the United States thus view adulthood as commencing for the most part at age 18 and completely by age 21.

Another way to demarcate between childhood and adulthood, which is particularly relevant for any discussion of vulnerability to psychopathology, is to rely on the current North American psychiatric classification system, DSM-IV-TR (American Psychiatric Association, 2000). In terms of explicit defini-

tions, however, DSM-IV-TR generally sidesteps this issue and states that the provision of a separate section for disorders that are usually first diagnosed in childhood or adolescence is not meant to suggest that "there is any clear distinction between 'childhood' and 'adult' disorders" (p. 39). Nevertheless, when age is mentioned at all in diagnosing "Disorders Usually First Evident in Infancy, Childhood, or Adolescence" (the general category that covers all these disorders), the maximum age is 18 for disorders appearing in adolescence. Likewise, most assessment methods for children and adolescence (e.g., the Diagnostic Interview for Children and Adolescents) define their top range at around age 17, and corresponding assessment methods for adults typically define a minimum age of 18 (e.g., the Structured Clinical Interview for DSM-IV Disorders [SCID]). The current diagnostic approach thus appears to have adopted an age demarcation between adolescence and adulthood similar to the U.S. legal standard, although for different reasons.

Another and more comprehensive approach focuses on theory and research on developmental processes. Rather than relying on arbitrary ages and legal standards, a developmental approach considers the physiological, emotional, and psychological maturation processes that occur as individuals progress from childhood to adulthood. Not surprisingly, from this perspective no single age best represents when an individual transitions into adulthood. In terms of physical maturation (e.g., maximum height and the development of secondary sexual characteristics) girls typically reach maturity by roughly age 16, whereas boys typically do so by age 18. However, some physical change will continue (e.g., brain maturation and adding strength and muscle mass through the mid- to late 20s). But physical maturation alone is not sufficient to differentiate between adolescence and adulthood in a psychological or emotional sense.

The determination of adulthood is also strongly influenced by the social context in which development occurs. For example, cultural differences can vary widely. In most non-Western cultures the transition from childhood to adulthood is socially defined and marked by a significant social event, such as marriage. In contrast, in the contemporary West, where there is a strong emphasis on individualism and independence and fewer and less well defined rituals of passage, the transition to adulthood is often determined by individual cognitive, emotional, and behavioral changes (Arnett & Taber, 1994). Nevertheless, from a developmental perspective, by approximately 18 years of age most individuals have experienced physiological, psychological, behavioral, and social changes that propel them to at least begin the transition into adulthood.

It is clear that any approach to defining adulthood must take into account a wide range of psychological, physiological, and cultural factors. Certainly the boundary between adolescence and adulthood is best represented as a gradual transition rather than an abrupt change. In general, however, at least in Western societies, it seems safe to suggest that the ages of 18–20 are a

reasonable time to begin to differentiate between adolescence and adulthood, recognizing that some individuals will not developmentally "fit" their age category. In fact, each of the approaches we briefly examined to determine the boundary between adolescence and adulthood (legal, psychiatric, and developmental) seems to converge on a similar time frame. Thus, somewhere between the ages of 18 and 20 appears to be a convenient place to mark the transition from adolescence to adulthood. Indeed, even a cursory review of the published research on child and adult psychopathology supports this age period as the typical line of demarcation between adolescence and adulthood.

PSYCHOPATHOLOGY AND VULNERABILITY FOUNDATIONS: A BRIEF SUMMARY

In this chapter we have noted two different facets of causality: causes of disorder and causes of the maintenance of disorder. We also examined certain core features that appear to characterize vulnerability constructs. In particular, we argued that vulnerability refers to the relatively stable causal mechanisms of psychopathology that are endogenous to the individual but that for many psychopathological states are actualized through a diathesis–stress relationship. We also noted distinctions between the concepts of vulnerability and risk and further suggested a preference for conceptualizing vulnerability and resilience as different ends of the vulnerability continuum, noting that resilience implies a resistance but not an immunity to disorder. We briefly reviewed some different perspectives on differentiating adolescence and adulthood and noted that they tend to converge on the ages of 18 to 20 as defining the transitional phase into adulthood.

As should be at least implicit in this discussion, we believe that vulnerability research represents not only the current cutting edge of psychopathology research but also the future for psychopathology research. Not that psychopathology research that focuses on describing the operation of various processes in the disordered state will be unimportant, but rather the clearest advances in understanding the causes of psychopathology will come from research that focuses explicitly on vulnerability. Not only will this approach bring us closer to understanding causal processes, but also in so doing it will bring us closer to understanding the mechanisms that must be therapeutically addressed once a disorder has occurred. An adequate understanding of vulnerability can also aid in preventing the onset of psychopathology, or at the least attenuating the duration and intensity of disorders along with their damaging effects (e.g., deficits in interpersonal functioning). Moreover, although still separated by a gulf between childhood/adolescence and adulthood theory and research, we believe that the clearest road to understanding vulnerability and prevention will come from research that considers vulnerability from a lifespan perspective.

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