Attachment and Psychosomatic Medicine: Developmental Contributions to Stress and Disease

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Objective: The object of this study was to evaluate the evidence linking attachment insecurity to illness. Attachment theory describes lifelong patterns of response to threat that are learned in the interaction between an infant and his or her primary caregiver. Despite its biopsychosocial domain, attachment theory has only recently been applied to psychosomatic medicine. Method: MEDLINE and PsychInfo databases were searched from 1966 to 2000 for English language papers with key words “attachment” and “object relations.” Papers and their cited references were reviewed if they were directly related to physical illness, symptoms, or physiology. A hypothetical causal model was developed. Results: Direct and indirect evidence from survey studies supports an association between attachment insecurity and disease. Animal studies and human experiments suggest that attachment contributes to individual differences in physiological stress response. There is also less robust support for insecure attachment leading to symptom reporting and to more frequent health risk behaviors, especially substance use and treatment nonadherence. Evidence supports the prediction from attachment theory that the benefits of social support derive more from attachment relationships than nonattachment relationships. Conclusions: Although the available data are suggestive rather than conclusive, the data can be organized into a model that describe attachment insecurity leading to disease risk through three mechanisms. These are increased susceptibility to stress, increased use of external regulators of affect, and altered help-seeking behavior. This model warrants further prospective investigation. Key words: Attachment, disease model, stress, development, biopsychosocial.

There is convincing evidence that social support (1), depression, (2, 3) and stress (4) have important effects on the progress of some diseases, although the relationship between these psychosocial factors and disease processes is complex and often inconsistent between studies (1, 2, 4). Furthermore, there is a growing body of evidence describing a high prevalence of past psychological trauma, especially sexual abuse, in persons with a variety of physical conditions including gastrointestinal disorders (5), fibromyalgia, (6, 7) and pain syndromes (8, 9). One of the central questions for any model that ascribes disease risk to relatively common events such as trauma, loss, isolation, or to the ubiquitous experience of stress is how the model accounts for individual differences in psychosocial susceptibility.

This paper explores the possibility that attachment theory could be extended to a biopsychosocial model of development and health that explains inconsistencies in the stress and social support literature as the result of predicted and measurable individual differences. The attachment model explains how repeated crucial interactions between infant and caregiver result in lifelong patterns of stress-response, receptivity to social support, and vulnerability to illness.

ATTACHMENT THEORY

When Bowlby described attachment theory (10–12), he provided a biological basis for understanding close, protective relationships. A central innovation of attachment theory was the recognition that a child’s desire for proximity to his or her mother is a biological drive which has been selected in evolution, rather than a behavior which is learned to satisfy other biological drives such as hunger. If maintaining proximity is a fundamental need, attachment behavior can be understood as a set of strategies that have been learned to achieve optimal proximity. An infant, defenseless on its own, maintains proximity to her or his mother through a complex system of communications and behaviors, which increase its chances of survival. Attachment behavior, such as smiling, vocalizing, crying, and approaching, is the normal, adaptive response of a mammal to threat. Organized patterns of attachment behavior emerge at a relatively fixed time for each species, in humans the second half of the first year (13).

Before the emergence of organized attachment behavior (ie, before learning) infant signaling of needs is innate and responsive to immediate stimuli. In this pre-attachment period appropriate proximity relies on parental anticipation of infant needs and sensitivity to infant signals. In the transition to organized attachment behavior, proximity is increasingly dependent

ACTH = adrenocorticotropic; CSF = cerebrospinal fluid; HbA1c = glycated hemoglobin; HPA = hypotha-

omic-pituitary-adrenal; NE = norepinephrine; SOC = sense of coherence; UC = ulcerative colitis.

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Attachment behaviors occur in patterns that show substantial differences between infant-parent pairs but occur in typical clusters. Infants can be reliably classified in an attachment typology based on Ainsworth’s standardized Strange Situation in which the infant is presented with stressful situations that include the presence of a stranger, separation of the infant from his or her primary caretaker, and reunion (13). Avoidant infants cry minimally during separation, treat the stranger similarly to the parent, and avoid contact on reunion. Secure infants show distress at separation and actively seek contact at the time of reunion, which is soothing. Angry-ambivalent infants similarly show separation distress and proximity-seeking on reunion, but combine proximity-seeking with angry resistance (13). The fourth category, disorganized attachment, was created later to include subjects who present no such coherent pattern.

The origin of these attachment phenotypes is thought to reside in a complicated “goodness of fit” between parent, child, and environment. Although most attention has been paid to the parental contributions to attachment style it is also recognized that parental attitudes are shaped by the temperamental needs and abilities of the infant and by environmental stresses and supports (15). Despite this complexity, some generalizations are useful. In general, parenting that is unresponsive relative to the needs of a child or intolerant of a child’s distress predisposes to avoidant attachment. The child learns that seeking proximity through crying and clinging is futile. On the other hand, independence is reinforced and valued. As a result attachment behaviors are relatively deactivated. Maintaining a degree of protective proximity depends on avoiding communications of dependence (16).

Angry-ambivalent attachment typically follows from interrupted or inconsistent parental care (17). Confidence in oneself to respond appropriately to threats does not develop adequately. The value of turning to the attachment figure for security is intermittently reinforced, but the need to be vigilant for the presence and loss of the other is more strongly reinforced. Proximity, once obtained, is often not soothing, resulting in persistent anxiety.

Developmental Psychobiology of Attachment

The psychobiological basis of attachment has been studied in animals, where it is possible to observe extremes of attachment system failure. The first such set of observations is of primates raised with various degrees of maternal deprivation (18). The second is a series of experiments with rats that has elegantly elucidated the “hidden regulators” of infant physiology that occur in the mother-infant interaction (19).

Brief separation of mammalian infants from their mothers results in protest behavior, such as high-intensity vocalization, agitated searching, and excessive self-grooming (19). Persistent separation results in reduced responsiveness, decreased heart rate, and lowered body temperature often referred to as despair (18, 19). Monkeys that are kept in isolation through critical periods of infancy demonstrate gross behavioral and biological consequences. The behavioral consequences of isolation rearing represent a disorganization of innate social behaviors. What these animals are missing, according to attachment theory, are the repeated experiences of parental amplification and reinforcement of innate behavioral patterns that result in social norms becoming encoded in procedural memory (14). The biological consequences of isolation rearing include abnormalities of neurotransmitter and hormone function (18). Further consequences include reductions in cerebellar (20) and motor cortical (21) dendritic branching. These deficits illustrate the degree to which normal brain development and normal endocrine and neurotransmitter regulation are dependent on the postnatal influence of crucial social interactions.

The large conceptual gap between observations of maternally deprived primates and the normal experience of parents and infants is partially bridged by the description of a continuum of rehabilitation in deprived primates. In particular, Kraemer (18) has described how the consequences of isolation rearing can be partially compensated for by substitute mothers, ranging from inanimate surrogates to peers to same-species foster mothers. In a stepwise progression, the degree of residual impaired function in isolation-reared monkeys depends on the extent to which maternal functions are replaced by a surrogate and by the age of the monkey at the time of replacement. Inanimate surrogates lead to increased attachment behavior if the surrogates bear qualities such as feeling soft and furry, warmth, mobility, and rocking motion. Inanimate surrogates will not suffice to correct the greatly decreased CSF NE levels of the isolates, but peer surrogates are partially effective. Peer surrogates rehabilitate behavior to a greater degree, such that isolated

monkeys can become indistinguishable from peers when in familiar social situations. To achieve near normal levels of CSF NE requires same-species adult female surrogates. Importantly, none of these surrogates is able to provide isolates with the ability to perceive social relationships, to respond well to social cues or to behave normally when competing for resources. Thus primate isolation rearing provides strong evidence of both biological and behavioral effects of early social deprivation and of the partial plasticity of these effects to rehabilitation by improved social relationships.

Hofer’s rat model brings us closer to the regulatory processes that occur between mother and infant under more nearly normal conditions. The infant rat depends on its mother as a regulator of a number of functions critical to survival, including nutrition and regulation of temperature and activity (19). This body of work reveals two factors that are important to understanding the association of attachment to health. First, the mother is understood to be supplying necessary regulatory functions for the infant. Understanding that the mother serves as an external regulator of the infant’s physiology allows us to see that the loss of mother at this critical stage directly constitutes a loss of regulation. More than just a stress, maternal loss constitutes an actual undoing of the system. Second, the nature of these regulatory functions is highly specific and not intuitively obvious. For example, infant activity is increased by warmth (provided by close proximity to mother) but decreased by tactile contact with mother. Furthermore the degree of contact with and stimulation by mother is regulated by infant activity, such that the mother-infant pair taken together comprises a balanced feedback loop. Hofer’s experiments demonstrate the mechanisms by which proximity-maintaining communications occur developmentally before the feelings that are recognized, in older humans, as “security.” Attachment is first and foremost a biologically based behavioral system.

The development of autonomous function requires a transition from the interdependent mother-infant regulatory system to relatively independent internal self-regulation of physiological functions. This transition depends on the unfolding of genetically programmed neurological development under the constant modification of social interactions (22). Thus, the same processes of mutual engagement and interaction that initially serve to maintain proximity as an end in itself are the crucial prerequisites of both emotional attachment security and physiological self-regulation. These intertwined developmental processes ultimately result in an organism that can independently maintain homeostasis and mount a physiological response to external threat and in a set of expectations related to threat.

Expectations learned after the development of declarative memory form a cognitive schema that defines what sorts of events are threatening, how one should react to threats, and what one can expect of others under these circumstances. On the other hand, earlier learning encoded in procedural memory does not form a cognitive “expectation” but a nonverbal working description of what counts as a threat and what is done when one is detected. Together, this set of expectations relating to threat, proximity, and separation are called the internal working model.

Adult Attachment Patterns and the Internal Working Model

Any relationship in which proximity to the other affects felt security is an attachment relationship (23). The primary example of adult attachment is between committed sexual partners, but many other relationships may include a degree of attachment, including the relationship of patient and clinician.

In Bowlby’s theory, the internal working model provides the continuity of attachment style over time. Models of self and other vary from very positive expectations (resiliency of self, responsiveness of other) to very negative expectations (fragile or incompetent self, unavailable or unreliable other). Since expectations of self and other are relatively independent (24), an individual’s attachment status can be described by their position on each of these dimensions, which has led to a four-category, two-dimension model of attachment (25).

In the four-category model a person who has positive expectations of both self and other is secure. Secure attachment is associated with an internalized sense of being worthy of care, of being effective in eliciting care when required, and a sense of personal efficacy in dealing with most stressors independently (23). Secure individuals are described as adaptable, capable, trusting, and understanding (26). In this description, we are choosing to emphasize aspects of attachment that relate to security, threat, and stress and to de-emphasize feelings related to loveability and comfort. This is consistent with Bowlby’s psychobiological model.

Insecure attachment can be categorized as preoccupied, dismissing, and fearful. A person who expects to cope with stress inadequately but whose expectations of others are more positive is preoccupied. Preoccupied attachment, the adult correlate of infant angry-ambivalent attachment, is associated with excessive care-seeking, separation protest, and fear of loss (23).
Although the preoccupied individual seeks care, the soothing that results from contact is partial and transient. Preoccupied individuals are described as anxious, dependent, emotional, impulsive, and approval-seeking (26).

People who distrust the effectiveness of social supports but have a positive view of themselves emphasize independence and are classified as dismissing. A self-sufficient and undemanding attitude is often highly valued, but the associated distrust and avoidance of intimacy communicates the underlying insecurity. Situations that demand relinquishing control and depending on others, such as hospitalization for acute illness, may result in crisis. Dismissing attachment is characterized by coldness to others and competitiveness (25).

Finally, a person whose expectations of both self and other are negative is fearful. Fearful attachment is the least-studied category, having been introduced by Bartholomew in 1991 (25). Fearful individuals are described as cautious, doubting, self-conscious, shy, and suspicious (26).

Dismissing and fearful attachment taken together are the adult correlate of infant avoidant attachment. Most studies do not distinguish between dismissing and fearful attachment, often labeling the composite category “avoidant.” To maintain consistency, the term avoidant will be used to describe dismissing and avoidant styles (except where studies specifically distinguish dismissing and fearful styles), whereas “preoccupied” will be used to designate the attachment type that is variously referred to as preoccupied, anxious, ambivalent, and resistant.

Studies of attachment types in different age groups have demonstrated that the prevalence of avoidant attachment is fairly constant in all age cohorts (about 20% to 25%) whereas the prevalence of preoccupied attachment is less in older cohorts (from about 20% at college age to 5% to 8% in middle age) (26, 27). Recently, a 31-year longitudinal study in women supported the view that these changes represent trends in individuals over time, rather than cohort effects, by showing a modest decline in preoccupied attachment, a similar increase in secure attachment, and stable avoidance attachment scores over adult life (26).

The importance of the internal working model for understanding attachment and health is that it clarifies the dilemma of whether attachment insecurity should be understood as an emotional state or a personality trait. An attachment type is the result of an internal working model that guides affects and behavior when a threat is perceived. Presumably the neurological substrate for this internal working model has developed through the effects of highly salient conditioning in the first attachment. Attachment type is then understood as a disposition toward certain perceptions of others, certain perceptions of self, and certain preferred strategies that will be triggered by the presence of a perceived threat. The emergence of attachment behavior is largely context dependent, a state phenomenon, but the internal working model that provides consistency to the pattern of behavior that emerges is a trait.

**METHODS**

MEDLINE and PsychInfo databases were searched from 1966 to 2000 for English language papers with key words “attachment” and “object relations.” Peer-reviewed papers and their cited references were included for review if they were directly related to physical illness, symptoms, or physiology, and not included if they addressed only psychopathology. This literature was first reviewed for evidence for and against an overall association of insecure attachment and disease. Possible causal mechanisms linking insecure attachment to disease were then reviewed. A causal model was developed and the evidence for and against hypotheses contained in the model was reviewed.

**ATTACHMENT INSECURITY AND DISEASE**

To assess whether there is an overall association of insecure attachment and disease there are four relevant aspects of attachment literature. The evidence for an association of attachment and disease in childhood, an association in adulthood, an association of childhood precursors of insecure attachment and adult disease, and the association of secure attachment and later health will be reviewed.

Is Childhood Attachment Insecurity Associated With Childhood Disease?

The clinical observation that parents of ill infants experience anxiety that might interfere with reliable and consistent parenting has led to the study of attachment security in several diagnostic groups. Remarkably, no excess of attachment insecurity has been found in premature infants (28, 29), in preschoolers with developmental delays (29), or in infants with cleft lip and palate, (30) compared with either healthy controls or community norms.

On the other hand, toddlers with certain chronic illnesses have been found to have a higher prevalence of insecure attachment. Children with recurrent otitis media over six months exhibit more attachment insecurity than children without recurrent otitis media (31). Twenty-six children aged 12 to 22 months with failure to thrive exhibited less secure attachment and more anxious, disorganized attachment in the Strange Situation, regardless of organic or nonorganic etiology of failure to thrive (32). Cystic fibrosis is associated with a higher prevalence of disorganized insecurity than found in healthy controls (33). Epilepsy is associated with a higher prevalence of disorganized insecure attachment than found in healthy controls (33). Epilepsy is associated with a higher prevalence of disorganized insecure attachment than found in healthy controls (33). Epilepsy is associated with a higher prevalence of disorganized insecure attachment than found in healthy controls (33). Epilepsy is associated with a higher prevalence of disorganized insecure attachment than found in healthy controls (33). Epilepsy is associated with a higher prevalence of disorganized insecure attachment than found in healthy controls (33).
Is Adult Attachment Insecurity Associated With Adult Disease?

Adult attachment pattern can be reliably classified using the Adult Attachment Interview, while dimensions of attachment security are measured with a number of self-report instruments (37). While adult attachment has been extensively studied in relationship to psychopathology (38) there has been much less investigation of the relationship between adult attachment and physical illness. In idiopathic spasmotic torticollics, avoidant attachment, measured by the Attachment Q-sort, is over-represented compared with normal controls (39). In UC, avoidant attachment (compulsive self-reliance) measured by the Reciprocal Attachment Questionnaire is more prevalent in a subgroup of patients who lack an antibody marker than in a clinically similar subgroup of UC patients with the marker. This finding is taken as tentative confirmation of a hypothesis of distinct etiological paths in these two groups (40).

SOC is a construct that describes a psychological predisposition to life stress response (41). SOC describes a person’s confidence in their ability to understand and manage stimuli from their internal and external environments and, thereby, cope effectively with stressors. SOC is very similar in description to the secure end of the two-dimension, four-category model of attachment. An association of low SOC and coronary heart disease risk has been found in white-collar men, but not in blue-collar men (42).

Are Childhood Risk Factors for Attachment Insecurity Associated With Adult Disease?

The best prospective test of the influence of insecure attachment on adult disease would be to determine attachment security in children and follow them until adulthood. Such studies are not yet available in humans, despite the fact that some cohorts of children observed in the Strange Situation are now old enough that their adult health status could be investigated. There are animal studies and less direct human studies, however, which address this question.

In animal studies, premature maternal separation is an experimental analogue of the naturally occurring parental behaviors that contribute to attachment insecurity. In a strain of rats that is genetically susceptible to stress-induced gastrointestinal ulceration, early weaning results in significantly more stress-induced ulceration after the pups have grown to maturity (43). Inconsistent results have been reported in cancer-prone rats. In one study separation of rat pups from their mothers for 20 hours per day for 18 days was associated with longer survival time after inoculation of Erlich carcinoma (44). In another study of the same animal model, rats that were completely weaned from their dams prematurely showed longer survival after implantation of Erlich carcinoma if they were paired with a littermate after weaning (45).

In humans, one useful strategy to examine this relationship is to look at childhood events that are often precursors of insecure attachment. Evidence supports the clinical observation that experiences of trauma, neglect, parental loss, and separation increase a child’s risk of insecure attachment (46). While attachment security is the result of a complex interaction between a child and his or her caretakers and environment, and not all traumatized children become insecurely attached, trauma can be understood as the most provocative test of the resiliency of the infant-caretaker system. Study of the potential precursors of attachment insecurity is important because events like trauma and parental death can be reported retrospectively with more reliability than reports of the subjective quality of parent-child attachment. Studying the precursors of insecurity allows causal inferences to be made about attachment in the absence of prospective studies. A very large study of primary practice medical patients provides strong evidence for an association between precursors of attachment insecurity and adult diseases. The risk factors assessed were psychological, physical, or sexual abuse, violence against mother, and living with family members who were substance abusers, mentally ill, suicidal, or imprisoned. The disease outcomes studied were ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease. In 9,508 patients, a graded relationship was found between the number of retrospectively reported childhood family risk factors and adult disease (47).

Is Secure Attachment Associated With Adult Health?

If insecure attachment is associated with disease it should also be true that secure attachment is associated with health. Support for this association can be inferred from the 50-year prospective study of a cohort of men recruited at university age (48). Adult attachment was not explicitly measured at the onset of this study because the concept had not been described in 1942. However, childhood strengths were measured in several ways that taken together suggest that secure attachment may be a predictor of physical health. Specifically, the cohesiveness of the home before age 20 and whether or not the boy’s relationships with his mother and father were conducive to trust, autonomy, and initiative were predictors of the man’s health at age 50 (49). At age 60, early adult closeness to siblings became the more significant predictor. Maturity of defenses at age 20 to 47 also predicts physical health at 60 (48).

MODEL OF INSECURE ATTACHMENT CONTRIBUTING TO DISEASE

Because there is suggestive evidence from a range of studies favoring an overall association of attachment insecurity and illness, we wish to examine the possibility that insecure attachment contributes to disease processes. In reviewing the literature, we have developed a model describing paths by which insecure attachment could affect the course of disease. Figure 1 describes potential psychosomatic mechanisms that could account for a correlation of insecure attachment and disease. The model describes how insecure attachment may be associated with disturbances of stress regulation (Path 1), use of external regulators of affect (Path 2), and nonuse of protective behaviors (Path 3). These possible paths organize the literature review that follows. The theoretical reasoning behind each path is followed by an empirical review.

Path 1: Insecure Attachment May Affect Stress Regulation

Of the three proposed paths by which insecure attachment could affect disease, the role of stress is of particular interest from a physiological point of view. Attachment and stress are related developmentally because, on the one hand, the stress response has been selected in evolution to be triggered by environmental threat while, on the other hand, the function of the attachment system is to increase security in the face of environmental threat. We propose three ways by which attachment may determine individual differences in the stress response.

Attachment insecurity may increase perceived stress. The internal working model describes the evaluative processes by which an individual deals with threat by negotiating proximity to trusted others. Included in this evaluation is a determination of what con-
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Fig 1. Model of hypothesized mechanisms by which attachment security could contribute to disease.

attachment constitutes a threat, which may involve both qualitative experiential features of the environmental situation and a threshold for triggering attachment behavior. Preoccupied attachment involves a self-perception of vulnerability, which may lead to a lower threshold for activating attachment behavior. In one model of hypochondriasis and somatization, preoccupied persons have acquired a sense of personal vulnerability and vigilance so intense that interoceptive sensation (normal perception of physiological operations) is perceived as a potential threat (50). Avoidant attachment, on the other hand, includes an attitude of interpersonal distrust that may result in situations that require intimacy or interdependence (including a situation of apparent "social support") being perceived as threatening.

Mikulincer and Florian (51) have studied the relationship of adult attachment to perceived threat and distress under two conditions of real-life stress. In 92 male military recruits undergoing four months of combat training appraisal of the training in terms of threat was more pronounced in ambivalent (preoccupied) persons than in avoidant or secure persons. In a retrospective study of students who had experienced missile attacks in the Gulf War, attachment type had a significant effect on the intensity of perceived distress. All insecure subjects reported higher levels of hostility and somatization than secure subjects, while ambivalently attached (preoccupied) subjects also reported more anxiety and depression distress (52).

Attachment insecurity may affect the intensity or duration of the physiological stress response. The stress response is highly regulated at multiple physiological levels. It is reasonable to assume that this complexity allows that central factors (such as insecure attachment) may modulate the system partially, allowing not only for individual differences in the initiation or noninitiation of the stress cascade but also for modification of its intensity or duration.

In rats, an increase in maternal care elicited by separating an infant mouse from the mother ("handling") for a few minutes each day in the first two weeks of life results in lifelong reduction in the HPA response to stressors. If the separation is longer, mothers ignore the pups on their return and the pups suffer a lifelong increase in the HPA response to stressors. If the separation is longer, mothers ignore the pups on their return and the pups suffer a lifelong increase in the HPA response to stressors.

In university students, a history of childhood bereavement and a self-report of disturbed family environment each exert a main effect on the cardiovascular response to a laboratory stressor. In the same protocol, family disturbance also predicted an enhanced cortisol response to the stress of viewing a movie depicting the death of a boy's mother (58). Similarly, women with a history of childhood sexual abuse exhibit increase ACTH, cortisol, and heart rate responses to a standardized stress compared with women without such a history (59). While these studies allow inferences to be drawn about attachment insecurity, one study of stress response has directly measured adult attachment. In idiopathic spasmodic torticollis (but not in healthy controls), avoidant attachment measured by the Attachment Q-sort is associated with elevated salivary cortisol response to the stress of discussing attachment relationships (39).

[Attachment pattern may determine the success of social support in buffering stress.] Social support has been widely studied as a mediator of illness and is considered to be beneficial to a range of health outcomes (60), despite controversy about the details of the association and its mechanism (61). Attachment theory may be required for a coherent reading of the complexities of the relationship of social support and health. Attachment theory, for example, predicts that attachment relationships will provide "felt security" in the face of threat or loss whereas other (nonattachment) relationships will not. Given the close developmental relationship between attachment style and stress response, it can be hypothesized that relationships that provide "felt security" are also more effective in buffering stress. West (62) has used this prediction to explain why close, intimate, and confiding relationships buffered women against...
the psychiatric sequelae of stressful life events in Brown’s Camberwell study, when no such effect was found with other close affiliate relationships, even to first degree relatives (63). Furthermore, attachment theory predicts that the degree of buffering provided by an attachment figure would be greater for secure individuals than insecure ones and greater in preoccupied insecurity than in avoidant insecurity.

It is expected that attachment style will also influence the degree to which social contact is sought at times of stress. Secure attachment is thought to be associated with a greater willingness to seek support and a related expectation that support will be available and effective. Avoidant attachment is associated with a desire for interpersonal distance and self-reliance (23), whereas preoccupied attachment may be associated with less support-seeking because of a fear of unavailability or rejection.

A longitudinal study of 60 recently widowed and 60 married men and women supports the prediction that attachment relationships are special, in that the emotional loneliness resulting from the loss of an attachment partner cannot be compensated by nonattachment social support (64). With respect to stress and health variables, the ability of the proximity of an attachment figure to reduce physiological stress responses has received support from both animal and human studies. In guinea pigs the presence of a bonded partner is more effective than the presence of another same species animal in reducing cortisol response (65). In humans, partner support results in lower cortisol reactivity to a laboratory stress in men, compared with stronger support or no support (66). Family emotional involvement has a stronger association to cardiovascular health behavior than other forms of social support (67). Support from a spouse or family member is more effective in long-term reduction of diastolic blood pressure than other social support (68).

The hypothesis that different patterns of attachment are associated with different patterns of using social support has also been studied. In students, both the perception of available support and the extent to which support is sought at times of stress were greater in secure subjects than in avoidant or ambivalent (preoccupied) subjects (69). In 92 military recruits avoidant attachment was associated with less support-seeking than secure attachment (51). Secure women are more likely than avoidant women to seek emotional support from partners in an anxiety-provoking laboratory experience (70). In 81 healthy young adults dismissing and fearful attachment styles were associated with much less support seeking than secure and preoccupied styles. Support-seeking in this study was significantly mediated by the perception of available support (71).

Path 2: Insecure Attachment May Result in Altered Use of External Regulators of Affect

The next proposed etiological pathway concerns regulation of affect. Since insecure attachment results in deficits in internal affect regulation (72, 73), it is expected that insecurity will be associated with greater use of external regulators. A number of behavioral strategies that are used to regulate dysphoric affect (to soothe, to distract, or to excite) are also risk factors for disease. Included among these are smoking tobacco, drinking alcohol, using other psychoactive drugs, over-eating, under-eating, and engaging in risky sexual activity. This path focuses on regulatory behavioral strategies, somewhat arbitrarily distinguishing these from the relationship factors (social support) that have been described above.

 Substance use: The relationship between adolescent coping, adolescent-parent relationship, and substance use is fairly well established, although the inferences that can be drawn regarding attachment are generally indirect. Childhood abuse and trauma predict adolescent smoking and alcohol abuse (74) and adult smoking, alcohol consumption, alcoholism, and drug abuse (47, 75). Direct studies of attachment reveal that adults with avoidant attachment drink alcohol to enhance positive affect (76). Adolescent substance users value social conformity less than nonusers (74, 77, 78), which is closely related to parental support (77) and strength of emotional bonds to family and friends (74). Drug use in young adults is also related to parent-child attachment (79).

 Eating behavior. Patterns of food consumption are thought to be related to developmental relationships, and food consumption has physiological and affective regulatory effects. It is predicted, therefore, that insecure attachment will be a risk factor for disturbances of eating behavior. Insecure attachment is associated with weight concern and lower self-esteem in adolescents (80). Parental overprotection is associated with oral control and anorectic behavior in adolescent girls (81). Eating behavior is also associated with smoking and alcohol consumption (81). External regulation through food intake has also been suggested as a mechanism responsible for obesity. One study has provided confirmatory evidence (82), although this has not yet been replicated with better-validated measures of attachment insecurity.

 Sexual behavior. It is expected that attachment style will have a strong influence on sexual behavior (83). Unfortunately, the literature currently available is quite modest. As with substance use, social conformity and peer bonding have been associated with lower sexual activity in teenagers (81). Unsafe sexual behavior has been associated with impaired object relations (84).

Path 3: Insecure Attachment May Alter Use of Protective Factors

In addition to an excess of health risk behaviors, insecure attachment may also contribute to illness through the failure or nonuse of protective factors. One of the most important protective factors, social support, has been described above. Additionally insecure attachment may affect treatment adherence.

 Treatment adherence. Diabetes is an example of a disease in which treatment adherence is crucial. Vierdeman and Hymowitz (85) proposed a model of diabetic control in which “the effectiveness of [the patient’s] engagement and participation in treatment” is determined by early relationship experience. The experience of developing autonomy, mastery, and reliable warm support during the toddler phase provides a positive body image and sense of self-control that persists through life and allows for sensitivity to bodily needs and for appropriate self-control at times of health crisis. In the absence of such a sense of autonomy and flexibility, health crises produce defensiveness, especially denial. The mobilization of maladaptive defenses during a health crisis results in an inability to autonomously monitor blood sugar and respond adaptively by adjusting insulin and diet, and the inability to benefit from the availability of supportive resources. Recently this proposition has been empirically studied using HbA1c as a measure of diabetic control in 15 type 1 diabetics. HbA1c is an established biological index that is elevated in poorly controlled diabetes. Subjects with avoidant attachment had significantly higher levels of HbA1c than those with secure or preoccupied attachment (86). While it is presumed that this worse control is due to nonadherence, it is also possible that increased glucocorticoids in avoidantly attached persons contribute to insulin resistance.

Symptom reporting. Two studies directly support the link between attachment insecurity and symptom reporting. In students who were making the transition from home to living at university, a relationship of avoidant attachment and physical symptoms was found, mediated by emotional self-control and emotion-focused coping (87). In another study an excess of medically unexplained

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symptoms was found in fearful and preoccupied Hepatitis C patients compared with securely attached individuals with the same disease (88).

There is also indirect support for this association. In university students who report a low SOC there is a positive correlation between negative life events and physical symptoms (89). In addition, low SOC was found to predict sickness-absences from work in women, but not in men, over a four-year longitudinal study of municipal workers (90). Further indirect evidence comes from studies of adults who were sexually abused in childhood who have more pain complaints, at more sites and greater health care utilization than nonabused controls (9).

DISCUSSION

This review finds supportive evidence for the hypothesis that attachment insecurity contributes to physical illness. A model is presented that describes how attachment insecurity may contribute to three mechanisms that increase disease risk: altered stress physiology, increased use of external regulators of affect, and altered use of health-protective behaviors. Thus attachment style may be a predictor of stress-vulnerability and disease risk for a number of diseases.

A rationale for differentiating between individuals who are highly susceptible to stress, physiologically and behaviorally, and those whose response to stress is well-regulated is crucial for etiological research. Since heterogeneity in stress susceptibility is likely to dilute true stress relationships in population-based research, this rationale should provide new research approaches to the several diseases in which stress is thought to be a risk factor, but for which research on this association has resulted in inconsistent results.

The attachment paradigm provides a truly biopsychosocial model of disease. The model describes how repeated, critical interactions occur at key developmental periods between a child and his or her caretaker (each of whom come to the interaction with their own set of more or less determined temperamental needs and vulnerabilities). While the need for such interactions and the basic “vocabulary” of interaction is determined for a species over an evolutionary timescale, within this domain there is much flexibility allowing an individual to develop patterns of behavior and physiological response which provide the best fit between the individual, his or her caretaker, and their environment. The consequences of this developmental setup are both physiological and behavioral, and help to explain the wide range of expression of distress and coping behavior that is found in humans.

This review has focused on the possibility that patterns of attachment influence disease risk. It must be recognized, however, that the relationship of attachment to illness is very likely to be bidirectional. Illness events are near-perfect triggers for the mobilization of attachment behavior. Because illness events can represent loss, threat, isolation, and dependency, they are precisely the types of stress that will initiate attachment behaviors. The concept of the internal working model helps to clarify the distinction between attachment behaviors, which will often be elicited by illness stresses, and attachment patterns, which are more enduring. Nonetheless attitudes that indicate attachment insecurity may well be easier to elicit when the attachment system is mobilized during illness stress. Clearly there is a need for prospective clinical studies and controlled experimental studies to test the causal processes that link correlated states.

We propose that the deleterious health effects of attachment insecurity are not universal. Given the ubiquity of stress and the high prevalence of insecure attachment (35% to 40% of the general population (27)), attachment insecurity cannot logically be a sufficient condition for any moderately uncommon disease. Furthermore, it would not make evolutionary sense for a highly conserved behavioral pattern (91) to be a sufficient condition for major illness. It is more persuasive that attachment insecurity contributes to disease by adding risk as a “second hit” on a preexisting vulnerability, such as would be provided by expression of a gene or by an infectious agent, which provides the disease specificity.

The evidence reviewed here provides modest support for each of the hypotheses examined. Considered together, the direct and indirect evidence that favors a role for attachment in disease forms a coherent group of literature that calls for careful evaluation. The model that has been described and reviewed is complex, but each of the several hypotheses that it generates seems to be open to be operationalized and tested.

The association of attachment insecurity (and its precursors) and the physiological stress response has been more thoroughly studied than the association of attachment security and the behavioral risk factors that we were able to identify. As a result there is more robust evidence for the physiological impact of attachment insecurity than for its behavioral impact currently, which may run counter to intuition. Investigation of the role of attachment insecurity in treatment nonadherence and other health risk behaviors should be a priority.

The model has significant implications. If attachment insecurity contributes to disease course or onset, then this may lead to treatment opportunities. One can speculate that physiologic dysregulation which accompanies insecure attachment could lead, for example, to testing of benzodiazepines, or modulators of corticotrophin-releasing factors to reduce disease risk in persons with a specific vulnerability to a particular
illness (for example those at genetic risk) and/or those of a specific attachment type.

An attachment perspective might modify behavioral interventions for external regulators of affect that are also disease risk factors. Increasing treatment adherence in avoidantly attached individuals, for example, might be facilitated by strategies that enhance perceived personal control and respect interpersonal distance, whereas interventions to reduce excessive help-seeking in individuals with preoccupied attachment would take a very different approach.

Several caveats must be emphasized. One is that the association of attachment insecurity with major depression (92) has been given insufficient attention in the literature reviewed here. This association is important to the study of physical disease for a number of reasons. First, depression is often associated with a worse course of physical illness (2, 3). Second, depression may, in some cases, be a direct consequence of severe attachment disruption. Third, there is an important overlap in the physiological features of attachment insecurity and depression. Depression, chronic stress, and insecure attachment share an association to HPA dysregulation (54, 58, 93) in which it is conceptually unclear which entity is primary. Thus, the biopsychosocial concepts of depression and insecure attachment cannot be cleanly separated. At a minimum, the inter-relationship needs to be addressed by measuring and controlling for depressive symptoms in studies of attachment insecurity and disease.

It is also worth noting the differences between attachment and two other psychological constructs that have been invoked to explain the relationship between distressing experience and physical symptoms. SOC is descriptively similar to the secure end of the self-appraisal dimension of attachment in the four-category model. Although this observation has not been directly tested, it is possible that there is considerable overlap between these constructs. It remains problematic that SOC captures only half of the attachment construct. The “appraisal of other” dimension appears not to overlap with SOC. This is important because the appraisal of other dimension is crucial to understanding the avoidant stance which appears to be important in treatment adherence (86), in symptom reporting (87), and in the ability to benefit from the buffering potential of social supports. Neuroticism has also been postulated to mediate between the experience of childhood abuse and adult disease (94). Neuroticism, which describes a perceived inability to process or cope with intense affect, is not identical with insecure attachment. The neuroticism construct does not distinguish between attachment and nonattachment relationships in their ability to provoke and buffer stress. The differing predictions of a neuroticism model and an attachment model in this regard are empirically testable.

Finally, methodological concerns limit confidence in the original research reviewed in this paper. At this early stage of development of this literature most of the studies that have been published are positive studies, which introduces a potential bias. In addition, with the exception of one large general practice cohort (47), two prospective studies (36, 48), and the experiments (18, 19, 58, 65), these studies report on relatively small samples and are cross-sectional in design. Thus no conclusions can be drawn about the validity of the model.

In an effort to promote rigorous testing of a series of hypotheses that are important to psychosomatic medicine, the methodology of this review has been inclusive and synthetic. It is hoped that this exercise in hypothesis generation will encourage the development of a literature robust enough to allow for review that is exclusive and analytic in the future. In particular, prospective and longitudinal research is required to control for bidirectional influences between (context-dependent) attachment behavior and illness events and to determine if attachment types which are construed to be traits are, in fact, present as predisposing factors before disease onset.

REFERENCES

ATTACHMENT AND DISEASE


49. Vaillant GE. Natural history of male psychological health, II: some antecedents of healthy adult adjustment. Arch Gen Psychiatry 1974;31:15–22.


ANNOUNCEMENT

Minority Research Training in Psychiatry

The Program for Minority Research Training in Psychiatry (PMRTP) is funded by the National Institute of Mental Health. Through it, the American Psychiatric Institute for Research and Education sponsors training of minority medical students, Psychiatric residents, and fellows who are interested in research by providing advice, placement assistance, tuition, stipends, travel and other expenses. The director of the Program is James Thompson, M.D., M.P.H.; the project manager is Ernesto Guerra. For more information call the toll free number for the PMRTP, 1-800-852-1390, or 202-682-6225, email eguerra@psych.org, or write to PMRTP at the American Psychiatric Institute for Research and Education, 1400 K Street, NW, Washington, DC 20005.