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INTRODUCTION: EMOTION AND HEALTH

John T. Cacioppo

The proposition that emotion is a consequence of ill health is uncontroversial. A voluminous literature confirms that one of the best predictors of overall happiness in adults of all ages is health and physical fitness (Myers, 1992). This body of research further shows that emotions are especially stirred by ill health, chronic pain, fatigue, or threat of death. The underlying mechanisms for the effect of ill health on emotion are less straightforward and more fascinating than the effect itself, however. Maier and Watkins (1998), for instance, review evidence that an infection triggers a centrally orchestrated sickness response, which is mediated by vagal afferents and includes behavioral (e.g., lethargy), autonomic (e.g., fever), and neuroendocrine (e.g., cytokine) components. One's energy and affect are down when one becomes ill in part because one's cytokines (e.g., interleukin-1) are up.

The mechanisms by which cytokines affect the brain and emotion are also becoming clearer. Cytokines signal through the vagus nerve; they trigger small signal molecules that then diffuse into brain tissue, and cytokines themselves cross the blood-brain barrier. Once in the brain cytokines influence various neurotransmitters that impact moods and emotion, such as serotonin, dopamine, and norepinephrine (e.g., Dantzer, Bluthe, Laye, Bret-Dibat, Parnet, & Kelley, 1998; Weiss, Sundar, Becker, & Cierpial, 1989; cf. Sternberg, 2000).

The ill health of an individual with whom one is connected can also affect one's own emotions, which calls forth an entirely different class of mechanisms ranging from cognitive appraisals of the demands of caring for the individual to sympathic and empathic responses to emotional contagion (Hatfield, Cacioppo, & Rapson, 1994). Additional details about the various mechanisms by which emotion is a consequence of health can be expected in the coming decades. What they all have in common thus far is the notion that the emotions that result from health or ill health foster adaptive actions, self-regulation, and social coordination.

Emotions as Determinants of Health

The proposition that emotion can influence health or disease has been more controversial in recent history. Socrates (496–399 B.C.) and Hippocrates (466–375 B.C.), however, regarded emotion as a determinant of health and disease. In the 3rd century B.C., the Greek physician Erasistratos diagnosed a young man's debilitating attacks as caused by an emotional affliction—lovesickness (Mesulam & Perry, 1972)—and to this day, across the doorway of the building housing one of the first pharmacies in Switzerland, is an inscription that proclaims cures for all but lovesickness. In contrast, for most of the 20th century, "medical" maladies of the ilk suffered by Erasistratos's patient have been viewed as falling in the realm of mental (see Part 9 this volume) rather than physical health, a view fostered ironically by Sigmund Freud's seminal work on the role of "psychic determinism" in somatic conversion hysteria.

Bodily symptoms thought to be caused by mental or

emotional disturbances came to be labeled as psychosomatic disorders (Alexander, 1950), to distinguish them from disorders that were more amenable to the extant medical knowledge and technology. Included in psychosomatic disorders was an assortment of disorders that shared the absence of a coherent biological etiology (e.g., essential hypertension, gastric and duodenal ulcers, migraine headaches; eating disorders, asthma, arthritis).

"Stress" seemed to have something to do with these disorders, but the concept of stress itself was vaguely (or circularly) defined. Operationalizations and measures across studies, especially across animal and human studies, were regularly so different (e.g., restraint, hypoglycemic, orthostatic, mathematic stressors) that results were difficult to compare or reconcile (Lovallo, 1997). Stressors were not always negative since positive as well as negative events were considered stressors in studies focused on predicting ill health (Holmes & Rahe, 1967). Further complicating matters, the measurements of stress within a given study were often so weakly correlated that they provided poor convergent validity for the construct of stress (e.g., Lacey, 1959). In short, neither emotion nor health was a simple, unitary concept, and the search for a singular universal mechanism relating emotion to health was doomed to failure.

The Changing Landscape of Health and Disease

The beginning of the 20th century was a period when antibiotics were nonexistent, public health was underdeveloped, and germ-based diseases were among the major causes of adult morbidity and mortality. Only 4% of the U.S. population lived to be over 65 years of age, compared to over 17% today. Medical scientists at the turn of the 20th century focused on the major health problems of the day about which they could do something, with a remarkable record of success. By the end of the 20th century, public health improvements, widespread vaccinations, and advances in medical and pharmacological treatments had greatly diminished the ravages of infectious diseases. The fastest growing segment of the population is now older adults, with the number of persons under the age 65 in the United States tripling during the 20th century while the number of persons 65 and over increasing by a factor of 11 (U.S. Department of Health and Human Services, 1990).

While threats from infectious diseases demand continued vigilance (Garrett, 2000) and research and advances in molecular biology provide powerful new weapons with which to combat the devastation of genetic diseases (Kandel & Squire, 2000), entry into the 21st century has brought into prominence a new and looming set of health problems. The leading causes of death in industrialized nations are heart disease, cancer, cerebrovascular disease, accidents, chronic lung disease, pneumonia and influenza, diabetes, suicide, HIV infection, and chronic liver disease/cirrhosis (e.g., Blumenthal, Matthews, & Weiss, 1994). Chronic diseases are now the most frequent sources of complaints and the largest causes of morbidity and mortality in older adults. According to estimates by Luskin and Newell (1997), by the early 1990s individuals 65 and older accounted for 36% of all hospital stays and 48% of total days of doctor care in the United States. These percentages and the absolute costs are expected to increase as the elderly increase in numbers and in percent of the total population. Neither the paradigm of germ-based diseases nor the paradigm of simple genetic defects provides the best platform from which to attack chronic disorders with complex social, psychological, environmental, and behavioral determinants.

The Emergence of a New Paradigm

The dramatic theoretical and methodological developments in the affective sciences enjoyed over the last two decades coincided propitiously with the increasingly apparent need for a better paradigm for health and disease in the 21st century. This convergence was not announced by a single individual or event but rather was realized independently by researchers worldwide from multiple disciplines. The cumulative growth and potential synergisms are astounding. Figure 55.1 depicts the number of published articles uncovered by Medline searches on the terms "emotion" and "health" of the National Library of Medicine database from 1966 to 2000. As can be seen, the increase in scientific interest in emotion and health coincides nicely with the changing landscape of health problems. From this work, a paradigm shift can be identified in which environmental stimuli and events affect health not by a direct effect on the body (e.g., germ theory) but through their effects on affect (e.g., "stress") and behavior.

Within this new paradigm, one of the most powerful

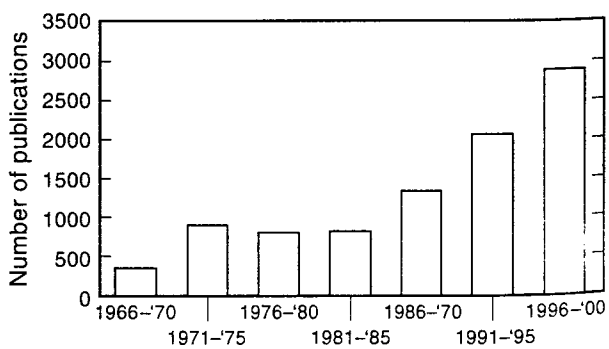

Figure 55.1 Number of publications per 5-year period from 1966 to 2000 on the topic of "emotion and health."

conceptual ideas to emerge is that the physiological systems activated by quotidian events can not only protect and restore but also damage the body. Claude Bernard (1878/1974) observed that the extracellular fluid constitutes the immediate environment—the internalized sea—for plants and animals. He noted the relative constancy of this internal milieu and regarded this constancy, and the physiological mechanisms that served to maintain it, as providing protection against the powerful entropic forces that threaten to disrupt the biological order essential for life. Without mechanisms to stabilize the cellular environment, organisms to survive would be confined to a limited ecological niche. In Claude Bernard's (1878/1974) terms, therefore, the existence of these mechanisms permitted warm-blooded creatures to live a free and independent existence.

Extending this perspective, Walter Cannon (1939) equated adaptive reactions with homeostatic processes that maintained the constancy of the fluid matrix (cf. Berntson & Cacioppo, 2000). Cannon argued that the variations from basal physiological levels ("set points") do not reach the dangerous extremes that impair the functions of the cells or threaten the existence of the organism because adaptive reactions are automatically triggered to return the affected physiological system to a basal state. In the past couple decades, the mechanisms by which such adaptive reactions are achieved have been elaborated (e.g., Berntson, Cacioppo, & Quigley, 1991), and the concept of homeostasis has been extended to the notion of allostasis—the ability to achieve stability in the internal milieu through change (McEwen & Stellar, 1993; Sterling & Eyer, 1988).

Cannon focused primarily on the physiological basis of homeostasis, but he also studied the influence of emotional disturbances on various physiological processes. This latter work focused on what he termed the emergency reaction. In Cannon's formulation, autonomic and neuroendocrine activation associated with emotional disturbances serve to mobilize metabolic resources to support the requirements of fight or flight, thereby promoting the protection and survival of the organism.

Many of the powerful elicitors of emotion in contemporary society—personal affronts, traffic congestion, pressing deadlines, computer viruses, perceived injustices—do not require or even allow behavioral fight or flight, and the reactions in response to these events can substantially exceed metabolic requirements. Thus, a design for the brain and stress physiology that worked well in human evolution in terms of maintaining a constant internal milieu may have maladaptive aspects that have become discernible as civilized societies developed and life expectancy increased well beyond the reproductive years (Lithgow & Kirkwood, 1996). Hans Selye's (1956) pullulating work on the general adaptation syndrome provided early support for the notion that physiological activation in response to stressors is beneficial up to a point but excessive or prolonged activation may indeed have hidden costs.

Selye studied physical stressors in animals, but subsequent work underscored the importance of idiosyncratic construals (i.e., cognitive appraisals) of an event for the feelings, autonomic adjustments, and behavioral responses to the event (e.g., Lazarus & Folkman, 1984; Mason, 1975; cf. Scherer, Schorr, & Johnstone, 2001) as well as the temporal dynamics of the elicited emotions (Davidson, 1998). Acute (e.g., major life events) and chronic (e.g., daily threats and challenges) psychological stressors can have short-term protective and long-term detrimental consequences. The latter costs have been termed allostatic load, which represents an overarching mechanism by which emotions may influence health. Berntson and colleagues (in chapter 58) review the neurobehavioral processes underlying the idiosyncratic construal of acute and chronic events, and McEwen and Seeman (in chapter 59) review evidence of the value of the concepts of allostasis and allostatic load in research on emotions and health.

Ceteris paribus, negative emotions have a larger impact on the brain (Ito, Larsen, Smith, & Cacioppo, 1998), the viscera (Cannon, 1939; cf. Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000), and social cognition and behavior (Cacioppo, Gardner, & Bernston 1997; Skowronski & Carlston, 1989; Taylor, 1991). With positive and negative emotions and their underlying substrates conceptualized as falling at the opposite ends of a single bipolar evaluative mechanism, focusing on the relatively potent and disruptive negative emotional processes to the exclusion of positive emotional processes made some sense. It followed from the putative reciprocity of positive and negative hedonic processes that reducing negative feelings was equivalent to increasing positive feelings, that what was learned from the study of negative emotional processes transferred directly and completely to positive emotional processes (but see Cacioppo, Gardner, & Berntson, 1999).

Understandably, given this historical context, the vast majority of research on health and emotion has studied the impact of negative emotional experiences on disease processes, with such studies outnumbering those studying positive, health-promoting factors 11 to 1 (Mayne, 1999). A long-standing area of interest has focused on the impact of psychological stress on cardiovascular activity (e.g., Tomaka, Blascovich, Kibler, & Ernst, 1997), neuroendocrine response (e.g., Cacioppo et al., 1995), and immune function (Glaser & Kiecolt-Glaser, 1994) as well as on mental (Folkman, Lazarus, Gruen, & DeLongis, 1986) and physical health (Cohen, Tyrrell, & Smith, 1991). A second major area of research has focused on the effects on physical health of negative emotions such as depression, anxiety, loneliness, hostility, and anger, as well as on emotional personality traits (e.g., neuroticism; e.g., Friedman &

Booth-Kewley, 1987; Miller, Smith, Turner, Guijarro, & Hallet, 1996). These areas of study have been productive in identifying negative emotional factors that act as health risk factors, and for developing models explicating how such factors may contribute to disease outcomes.

This research has tended to focus on the impact of negative emotional experiences (e.g., "stress") on disease processes. Accordingly, one view to emerge from this work is that negative emotions are something to be avoided or, when aroused, to be diminished rather than to be made a kernel for brown study. It is not that negative emotions were thought to have no value in everyday life; it is that human fight or flight responses evolved in such a different ecological niche that the daily events in contemporary society were triggering maladaptive negative affective (stress) reactions. Indeed, many of the contemporary health problems have affective bases ranging from anxiety, anger, and depression to unrealistic or drug-induced feelings of euphoria and invulnerability.

The early stages of emotional processing involve attentional deployment, cognitive appraisals, and reappraisals (Gross & Levenson, 1997). As an emotion unfolds, action tendencies emerge along with supportive peripheral physiological adjustments. At this point, emotional regulation becomes response focused, largely through the suppression of the expression of the response tendencies that have been activated. Gross and colleagues have shown that suppressing one's natural expressions of an emotion results in more autonomic activation than either changing the meaning of the emotionally evocative event or simply expressing one's disgust naturally. Because suppressing one's emotional expression requires self-monitoring and self-adjustments, it also requires cognitive resources that could otherwise be allocated to other tasks such as solving the immediate problem or encoding associated events for future reference. Geise-Davis and Spiegel (in chapter 56) review evidence that the expression of emotions, even negative emotions, is also healthier than the denial or suppression of emotional expression.

In addition, emotions can foster health and well-being. Emotions are not disembodied abstractions without purpose or import, but rather they motivate and guide attention, thought, and action. What is it about the circumstances in which emotions enhance rather than diminish health? Factors such as self-esteem, self-efficacy, and resilience predict positive mental and physical health (O'Leary, 1990, 1992), dispositional optimism speeds recovery from breast cancer and heart surgery (Scheier et al., 1989), social support and hardiness facilitate well-being even in times of stress (Cohen & Wills, 1985; Wiebe & Williams, 1992), and greater social integration predicts longer mortality (House, Landis, & Umberson, 1988). Ryff and Singer (in chapter 57) note that emotions that foster health contribute to life meaning and purpose, quality social relationships, self-esteem, or mastery. They build on this observation to examine the various roles in which emotion can promote health and well-being.

The past two decades have seen a dramatic rise in research on emotion and health. Investigations have moved from a paradigm in which environmental stimuli and events have a direct damaging effect on the body (e.g., germ theory) to one in which stimuli and events act on physical health through effects on emotion and behavior. Understanding the ways in which emotion influences health requires an integrative multilevel approach. Interest in integrative systems analyses fell out of vogue in the latter half of the 20th century, however, because it sits at the other end of the scientific spectrum from reductionistic molecular biology. The confluence of a changing landscape of health problems worldwide and the emergence of powerful multidisciplinary studies of emotion and health portend a return to integrative analyses spanning the cellular to the cultural levels of organization (Anderson, 1998; Cacioppo & Berntson, 1992). In this sense, the chapters in this part of the *Handbook* represent the wave of the future.

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