CHAPTER 1

Introduction

Somatization is among the most puzzling phenomena that healthcare workers encounter. In somatization physical symptoms occur in the absence of any identifiable causal mechanism. The causes of somatization that we are able to implicate are neither proximate nor somatic, seeming instead to be indirect and to reside in the patient's mind or culture. Somatization appears to be universal. We find it in all present societies and in all past societies for which we have relevant records. Forty centuries ago, the physicians of Egypt were familiar with somatization; some years later so were those of ancient Greece.

For the contemporary clinician, the patient who somatizes is a pressing practical problem. Here there is distress, dysfunction, and disability of great magnitude and intransigence. Patients diagnosed with the most severe form of somatization, somatization disorder, have been shown to incur healthcare expenses that are nine times the U.S. average and consume disproportionate amounts of the time and energy of healthcare providers (Smith, Monson, & Ray, 1986a). In addition to the extensive direct costs, somatization disorder creates enormous indirect costs to the economy in the form of lost work productivity. Individuals diagnosed with somatization disorder report being bedridden for 2–7 days per month (Katon et al., 1991; Smith et al., 1986a). Somatization disorder is not only costly, but also difficult to treat successfully. In a longitudinal study following patients with somatization disorder who were receiving standard medical care, only 31% recovered after 15 years (Coryell & Norten, 1981). Typically, patients with somatization disorder are dissatisfied with the medical services they receive and repeatedly change physicians (Lin et al.,

1991). These "treatment-resistant" patients frustrate healthcare providers with their frequent complaints and dissatisfaction with treatment (Lin et al., 1991). No controlled medication trial for somatization disorder has been published. Anecdotal evidence suggests that many patients diagnosed with somatization disorder refuse to take medication and that those who do frequently report adverse medication side effects (Murphy, 1982). The story is much the same with other polysymptomatic somatoform disorders (Fallon, 2004). As of this writing, pharmacological treatment has had minimal success with somatization.

In this book we describe our efforts to alleviate the suffering of patients with somatization. Over the last decade, we have developed a dedicated psychosocial treatment for somatization that draws upon various traditions in psychotherapy, especially cognitive-behavioral therapy and emotion-focused experiential therapies, a treatment we call affective cognitive-behavioral therapy (ACBT). The principal aim of this book is to describe that treatment and to provide the training material necessary for its effective use. To frame our approach to this problem, we first provide a brief review of the history of somatization and of psychosomatic medicine. We then discuss philosophical and sociocultural underpinnings of somatization and conclude with an overview of theories of somatization.

BACKGROUND: HISTORICAL AND THEORETICAL

The history of somatization begins with hysteria. Hysteria was first described 4,000 years ago by the Egyptians. Typical cases involved pain in the absence of any injury or pathology in the location of the pain. The Egyptian theory held that a wandering uterus moved about the body and produced pain from various regions. Greek physicians described a similar set of psychosomatic symptoms and essentially retained the Egyptian theory. The Greeks gave us the word *hysteria*, from the Greek *hystera*, meaning womb. The Greco-Egyptian formulation reveals two noteworthy features: that the disorder was primarily observed in females and that there was something thought to be essentially female about the disorder. Although the diagnostic category subsumed more than somatization, the term hysteria continued to be widely used to label somatization patients until 30 years ago.

Medieval and Renaissance medicine preserved the ancient formulation of hysteria as described by the ultimate authorities, Hippocrates and Galen, until the 17th and 18th centuries, when it was first linked with the nervous system and the emotions. At the beginning of the

17th century, the French physician Charles Le Pois opposed the uterine theory of hysteria (he believed the spleen to be the culprit) and declared that hysteria could occur in men; a few years later, Thomas Sydenham declared that hysteria was the result of psychological and emotional causes and that in men hysteria was manifested as hypochondria (Boss, 1979). Foucault (1961/1965) states that by the end of the 18th century hysteria and hypochondria were beginning to be viewed as diseases of the nerves akin to such recognized mental disorders as melancholia. By the 18th century, some authorities, such as Joseph Raulin, began to question hysteria's organic basis. Raulin described hysteria as a "disease in which women invent, exaggerate, and repeat all the various absurdities of which a disordered imagination is capable" (quoted in Foucault, 1961/1965, pp. 137–138). Before the 19th century, due to the heterogeneous nature of hysterical symptoms and the hypothesized connection with the emotions, physicians had begun to allege that these symptoms were feigned or imagined. The unsympathetic attitudes of contemporary healthcare workers toward somatizers and the tendency to regard them as malingerers can be traced to this period in the history of medicine.

Paul Briquet's (1859) seminal monograph, *Traité Clinique et Thérapeutique de L'hystérie*, was a landmark in the descriptive psychopathology of somatization. Our current conception of somatization disorder derives directly from this paper. Briquet's meticulous and exhaustive listing of the symptomatology of hysteria remains unsurpassed. In fact, he described three related syndromes: conversion phenomena, hysterical personality, and multiple chronic unexplained somatic symptoms (Dongier, 1983; Mai & Merskey, 1980), all overlapping in symptomatology somewhat and often observed to co-occur. Briquet's perspicuous work was revived by Purtell, Robins, and Cohen (1951) and developed further by members of the illustrious Washington University Department of Psychiatry. Perley and Guze (1962) published a list of 57 symptoms commonly reported by women diagnosed with hysteria, symptoms that were clustered in 10 different areas. These investigators were the first to suggest specific criteria for the diagnosis of hysteria: the presence of 25 symptoms from at least 9 of the 10 symptom areas (Guze, 1967). Later, this list of 57 symptoms was expanded to 59 symptoms and the term "Briquet's syndrome" was adopted (Guze, Woodruff, & Clayton, 1972). The criteria for Briquet's syndrome were incorporated into the Feighner criteria (Feighner et al., 1972), the precursor to the symptom set that appeared in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III; American Psychiatric Association, 1980). In that volume the theoretically neutral term "somatization" was preferred over the traditional terminology.

Although some of the traditional language remains in the fourth edition of the DSM (e.g., "conversion disorder"), the word "hysteria" no longer appears (DSM-IV; American Psychiatric Association, 1994). The ninth edition of the World Health Organization's (1979) *International Classification of Diseases* (ICD-9), a more cosmopolitan nosology of somatic and mental disorders published a year earlier than DSM-III, retains much of the perennial terminology, including not only hysteria but also "neurasthenia."[1] ICD-10 (World Health Organization, 1993) has shifted in the direction of the DSM, though without banishing every bit of the classical vocabulary.

The history of somatization also is interconnected with two important and historically related developments in the history of psychiatry: (1) psychosomatic medicine and (2) psychoanalysis. Although eventually absorbed by psychoanalysis and subsumed within a psychoanalytic theoretical framework, psychosomatic medicine had an established history before Freud. From antiquity, the interaction between mind and body and its effects upon health had been alluded to by many writers. An early systematic account was William Falconer's (1788) *A Dissertation on the Influence of the Passions upon the Disorders of the Body*. The term "psychosomatic" was used first by Heinroth in 1818 as "describing the interplay between mind and body in health and disease" (West, 1982, p. xvi). By the end of the 19th century "nervous conditions," including psychosomatic ailments such as neurasthenia, and the "nerve doctors" who treated them had proliferated, so much so that during the Victorian era "bad nerves" was thought of as something of an epidemic (Shorter, 1997).

It was about this time that Sigmund Freud entered upon the scene. As a young man Freud spent the winter of 1885–86 as a student of Jean-Martin Charcot at the Salpêtrière hospital in Paris. There he observed the world's leading authority, Charcot, use hypnosis to remove hysterical symptoms. Upon his return to Vienna, Freud began a close collaboration with Joseph Breuer. The product of this collaboration was the book *Studies in Hysteria* (1895/1974), in which Breuer and Freud developed the concept of "conversion," a process whereby intrapsychic activity putatively brings about somatic symptoms. Although Freud was later to break with Breuer and go on to create the substan-

[1]Neurasthenia is defined in ICD-10 as persistent and distressing feelings of exhaustion after minor mental or physical effort, accompanied by one or more of the following symptoms: muscular aches or pains, dizziness, tension headache, sleep disturbance, inability to relax, and irritability. The term was coined in 1856 by Robert Mayne and popularized by the American neurologist George Beard during the second half of the 19th century (Gijswijt-Hofstra & Porter, 2001).

tial edifice of psychoanalysis, his work on hysteria was a blueprint for and harbinger of later theoretical efforts. Here the ideas of early emotional trauma or intrapsychic conflict as the cause of physical symptoms began to take shape. This work also introduced the notion of a physical symptom as an unconscious form of communication, a device for securing secondary gain, or a means for avoiding emotional pain.

The notion of the transduction of psychological conflict into bodily symptoms was widely disseminated as psychoanalysis began to dominate psychiatry. Stekel (1924) coined the term "somatization" (*somatisieren*) during the early 1920s and defined it as "the conversion of emotional states into physical symptoms" (p. 341). That is, Stekel regarded somatization as equivalent to the mechanism of conversion that Breuer and Freud had used to explain the development of sensory or voluntary motor symptoms in hysteria. A strident and eccentric proponent of the mind–body interaction was Georg Groddeck (1977), who believed that psychic processes are etiological factors in all diseases (Avila & Winston, 2003). Groddeck contended that the symptoms of any somatic disease might be interpreted as symbolic expressions of unconscious motives and caused via the same mechanisms believed to underlie hysteria.

The father of modern American psychosomatic medicine, Franz Alexander, attempted to minimize the excesses of indiscriminate psychoanalytic approaches such as Groddeck's. Alexander (1950) took great pains to distinguish between two types of psychosomatic symptoms: (1) those cases in which psychological conflict was "converted" and communicated symbolically through physical symptoms, and (2) those cases in which the somatic symptoms resulted from the direct and indirect physiological effects of emotional arousal. This second kind of psychosomatic mechanism required few, if any, psychoanalytic assumptions and was quite compatible with mainstream scientific research, especially the work of Cannon, Seyle, and others on psychosocial stress. As psychoanalysis declined in influence, psychosomatic medicine declined also. Today, the term "psychosomatic," which was faddish in the 1950s, is no longer in vogue. Many of the problems once treated within the context of psychosomatic medicine now fall under the purview of what is, in some sense, its successor discipline: behavioral medicine.

SOCIOCULTURAL AND PHILOSOPHICAL ISSUES

The biopsychosocial concept of illness, proposed by George Engel (1977) and to which we subscribe, suggests that illness is a complex

entity involving the interplay of physical, psychological, and cultural factors. In particular, many illnesses cannot be adequately comprehended without taking into account the social contexts in which they develop and are manifested, diagnosed, and treated. What phenomena societies come to label illness or how human suffering is expressed and presented to healers are complicated matters that can be conceptualized at several different levels, levels that involve variables that interact causally. The advent of such disciplines as psychoneuroimmunology and behavioral medicine has brought us evidence, in many domains of medicine, of the close connections and complex concurrent interactions among mental, behavioral, and somatic variables. When we examine many somatic illnesses (e.g., hypertension) from the various standpoints of etiology, symptomatology, and treatment, they emerge as complex entities with multifaceted interacting components, with biological, psychological, and social causes (Baum & Posluszny, 1999; Cohen & Herbert, 1996). In the case of mental disorders or psychiatric syndromes the situation is even more complex and the levels of explanation more deeply intertwined.

The historical record shows that Western categories of psychopathology have been influenced strongly by sociocultural factors and that what gets labeled a mental illness is, to some degree, a reflection of cultural values. Drapetomania, the desire of slaves to escape captivity, was in the early 19th century considered a mental illness (Cartwright, 1851/1981; Szasz, 1987). Victorian physicians regularly performed "therapeutic" clitorectomies on masturbators, who were thought to be mentally ill. As recently as 1938, listed among the 40 psychiatric disorders in a leading textbook (Rosanoff, 1938) were moral deficiency, masturbation, misanthropy, and vagabondage. Homosexuality, which Western psychiatry regarded as a manifestation of mental illness, was "officially" depathologized, after a contentious political struggle, by a referendum of the American Psychiatric Association membership in 1974 (Kutchins & Kirk, 1997). Not so long ago, psychiatrists in the former Soviet Union performed the Orwellian maneuver of medicalizing opposition to the state when they employed the diagnosis of "sluggish schizophrenia" to effect the incarceration of many political dissidents (Bloch & Reddaway, 1977). Symptoms that indicate pathology in one society (e.g., regularly hearing the voice of a dead relative) are normal and customary in others.

Cultural variation in psychopathology results not only from differences in how psychiatric labels are applied, but from the fact that different societies seem to produce different forms of psychopathology. Many specific syndromes are unique to particular cultural contexts,

such as *ataque de nervios*, *koro*, or *taijin kyofusho*.[2] The epidemics of anorexia nervosa and bulimia in the contemporary West are unprecedented but are spreading to middle and upper classes around the world along with Westernization and its current aesthetic ideal of a slender female body (Ung & Lee, 1999). Writers such as Ian Hacking (1995, 1999) have argued persuasively that some mental disorders (e.g., multiple personality disorder) consist of roles that are created by the theories and practices of the mental health professions and subsequently enacted by patients. The articulation and dissemination of information about psychopathology through professional activities and by the media provide a symptom set and patient profile that can be assimilated by disturbed individuals who possess sufficient psychic malleability (Woolfolk, 1998).

Historically in Western psychiatry, a mental disorder has been posited in one of two instances. The first of these occurs when there exists a theory of psychogenesis, such as psychoanalysis, that hypothesizes mental entities to be the underlying causes of the symptoms of a disorder. The second instance involves the presence of symptoms in the absence of a physicalistic explanation. In this second instance, psychogenic etiology may be inferred solely from the absence of a known underlying physical mechanism, thus revealing a tacit dualism that originated even before Paracelsus and that continues to underlie Western medicine: Disease entities, whether they be causes or symptoms, belong to one of two categories, either the physical or the mental, these two categories being mutually exclusive (Robinson, 1996). Symptoms of almost any variety that cannot be linked to a scientifically explained physical pathology are assumed to be psychogenic. Individuals afflicted with multiple sclerosis, Wilson's disease, temporal lobe epilepsy, and numerous other maladies currently within the purview of somatic medicine were once regarded as mentally ill. Through the course of medical progress, mental illness has served as a residual category wherein poorly understood or refractory illness has been placed, often temporarily, only to be removed when medical science established the physical mechanisms underlying the disorder (Grob, 1991).

Dualistic assumptions operate not so subtly within DSM-IV. In DSM-IV two principal classes of disease entities are posited: (1) general medical conditions and (2) primary mental disorders. Contradistinct from the most paradigmatic mental disorders contained in DSM-IV are mental symptoms resulting from a "general medical con-

[2]Each of these "culture-specific syndromes" comprises somatic and psychological symptoms.

dition" (read: physical illness). Symptoms arising from such causes, indeed, imply the absence of a mental disorder. Of course, DSM-IV's authors claim that the distinction between primary mental disorders and those stemming from a general medical condition should not be taken to imply that there are fundamental differences between mental disorders and general medical conditions (American Psychiatric Association, 1994, p. 165). But the volume is careful to distinguish symptoms deriving from a general medical disorder from those that emanate from a primary mental disorder. This distinction is drawn so sharply that symptoms of organic origin are exclusionary for the diagnosis of such paradigmatic disorders as schizophrenia. The locution of the volume instantiates not only dualism, but also logical circularity, in that a general medical condition is defined as a medical condition other than a primary mental disorder, and a primary mental disorder is defined as something other than the result of a general medical condition. The distinction drawn here, whether nominal or substantive, is old Cartesian wine in a new bottle, that venerable distinction between the functional and the organic. In its language DSM-IV also stipulates, as a kind of axiom, the historical role of psychiatry as a processor of aberrations within the category of illness. Mental illnesses are abnormal, poorly understood illnesses that normal physicians do not treat.

Some have argued that the concept of somatization is unintelligible in medical traditions without a dualistic ontology. In many societies the concept of somatization has no meaning, since distinctions between mental and physical illness are not prevalent (Fabrega, 1991). For example, within the medical traditions of China and India, illness is conceived holistically in terms of various imbalances. The mind–body distinction is neither fundamental nor sharply drawn.

Studies of mental illness in non-Western societies reveal that somatic, rather than psychological symptomatology, often is the primary indication of a psychiatric disorder. For example, research in China has found that symptoms of psychiatric patients were predominantly somatic. For years the most commonly diagnosed mental disorder in China has been *shenjing shuairuo*, an indigenous diagnostic category signifying a "weakness of nerves" (Parker, Gladstone, & Chee, 2001). This disorder is described in the *Chinese Classification of Mental Diseases, 2nd Edition, Revised* (CCMD-2-R; Chinese Medical Association & Nanjing Medical University, 1995) and is accepted as a commonplace and legitimate illness by both medical practitioners and the general public. The disorder, oddly enough, is characterized largely by somatic symptoms, many of the same symptoms treated by Euroamerican "nerve doctors" in the 19th century, such as fatigue (Shorter, 1997).

Hence, the disorder is most often translated for Westerners as neurasthenia. Medical anthropologist Arthur Kleinman (1982) evaluated a sample of Chinese neurasthenics and determined that the majority manifested significant depressive symptomatology, albeit not sufficient nor in the requisite configuration to meet DSM criteria for a diagnosis of major depression. He concluded that many Chinese given the neurasthenia diagnosis could be suffering from depression, though not the Euroamerican form of depression that is characterized by despondence and patterns of thinking described by cognitive theorists such as Aaron Beck (Beck, 1976; Beck, Rush, Shaw, & Emery, 1979). In fact, studies of depression in China have found that relatively few Chinese manifest the DSM-IV syndrome of depressed mood, self-criticism, guilt, and pessimism. Chinese epidemiological research also suggests that patients with anxiety, like patients with depression, present a greater ratio of somatic to psychological symptoms than that found in the West (Parker, Cheah, & Roy, 2001; Tsoi, 1985; Zhang, Shen, & Li, 1998).

Research on psychopathology in China frequently is used to argue that cultural factors are crucial in determining the manner in which human suffering is experienced and, more specifically, to support the view that non-Western societies are prone to generate somatic expressions of distress. One distinction that is made in cross-cultural theory is that between somatization and "psychologization." The former refers to the experience of bodily aspects of distress whereas the latter refers to the experience of the psychic, social, and mental aspects of distress (Kirmayer, 1984; White, 1982). According to this formulation, either somatization or psychologization could serve as alternative modalities through which a negative emotional reaction is experienced and as alternative "idioms of distress" through which emotional pain is communicated. It has been suggested that psychologization is compatible with Western, Euroamerican concepts of selfhood and with an individualistic, psychologically minded worldview that emphasizes causal explanations implicating individuals and their traits as sources of events (Kihlstrom & Canter Kihlstrom, 1999; Kirmayer, Young, & Robbins, 1994). Somatization, in contrast, has been associated with the more sociocentric cultural views of selfhood where self-reflection and self-examination are deemphasized or disvalued; here, behavior is more often viewed as caused by the external environment, rather than by qualities of the person such as psychological traits or willpower. Other factors that might influence the ratio of psychologization to somatization are the stigma attached to psychological symptoms and the degree to which a desired treatment is obtained through either a psychological or a somatic presentation (Kirmayer, 2001).

The twin, reciprocal processes of somatization and psychologization are at first glance somewhat obscure and inaccessible. The workings are most often illustrated by examples of how emotional distress might be somatized. But let us first take the example of how a physical illness might be psychologized. Suppose you are a person who has contracted a mild viral infection and you are beginning to become symptomatic. Typically, if you conduct an examination of your initial symptoms, you may tend to emphasize elevated core body temperature or gastrointestinal motility, which you or your healthcare provider can readily link to the effect of a viral infection. The most familiar bodily discomforts of an infection constitute an incomplete inventory of all of its adverse accompaniments. There may be feelings of lethargy and dysphoria and difficulty concentrating. Why emphasize the former somatic symptomatology rather than the latter "psychological" effects of an infection? One could argue that the practice depends upon the patient's frame of reference. We report our physical symptoms, it could be argued, because we think we have an illness, which we conceive as a disorder stemming from physical causes, the important effects of and treatments for we believe to be somatic. Yet suppose you have the aforementioned symptoms but do not know that you have an infection and do not, therefore, privilege and adopt a physicalistic frame of reference. Suppose, also, that you are in the midst of a psychological crisis, such as job loss or divorce. Perhaps you have recently seen a daytime television talk show on which people report the various adverse emotional effects of psychological stress. Under these circumstances, with an attributional bias primed by salient psychosocial perturbations, you might be less inclined to measure your body temperature and, instead, might be disposed to focus on your mental state and conclude that you are depressed or "stressed out."

The theory we have been describing assumes that the background assumptions or the "idiom of distress" is crucial to determining whether a disruption of homeostasis is experienced as physical or mental. An individual can learn to attend to and express physical discomfort, rather than psychological distress, especially if an idiom of affect is not available. Thus, the theory has it that somatization conditions can be shaped through processes of selective attention to physical symptoms and by learning a vocabulary of somatic symptomatology.

Part of the allure of cross-cultural research on somatization symptoms is the possibility that cultural differences in the experience of illness might hold the key to understanding the mechanisms that underlie somatization. At this juncture, however, cross-cultural research on

unexplained physical symptoms must be regarded as inconclusive and fraught with methodological problems, including confounds involving socioeconomic factors and cross-cultural differences in healthcare systems.

Whether there are cultures that foster somatization is still a complex and controversial question. The World Health Organization's international collaborative study of Psychological Problems in General Health Care (Gureje, Simon, Ustun, & Goldberg, 1997) did not find the disparity in somatization disorders between East and West that might have been predicted from the early formulations of medical anthropologists. Nor did the ratio of somatic to psychological symptoms of depression vary across cultures in a systematic or expected fashion. The data were, however, to some degree consistent with the cultural hypothesis. For example, somatization rates were significantly higher in Latin America than in the rest of the world, and rates of somatization were higher in China than in the United States (Simon, Von Korff, Piccinelli, Fullerton, & Ormel, 1999). This study failed to include indigenous, culture-specific syndromes or to analyze single-symptom presentations, nor was there adequate assessment of the ratio of somatization to psychologization in the more frequently diagnosed forms of psychopathology, such as depression. A study that provides probative evidence on relative somatizing versus psychologizing tendencies across cultures has yet to be conducted.

MODELS OF SOMATIZATION

Not since the psychoanalytic era has somatization been viewed as a well-understood phenomenon. In some sense we have not progressed very far beyond Breuer and Freud's psychodynamic theory of conversion hysteria. Currently, there is widespread admission among authorities that no adequate theory of somatization exists. Indeed, one might argue that, with the exception of the oft-criticized and scientifically beleaguered psychoanalytic theory, there are no well-developed theories of somatization, only some fragmentary models or speculation. In Chapter 3, we describe and analyze some models of somatization and examine the relevant empirical evidence supporting each.

In evaluating models of somatization one needs to be mindful of the logical pitfalls that abound in the territory of mind–body relationships. As we have seen in conjecture about the cause of somatization, such phrases as "emotional distress expressed as physical symptoms" or a "somatic idiom of distress" or a tendency to "somatize rather than

psychologize" are invoked. Such locutions are problematic and bespeak the poverty of our theories as they invariably risk the possibility of emerging as either pseudoexplanatory or tautologous. Such formulations often fail to explain because they leave key terms undefined and unexplicated. We have seen this kind of fallacious logic before in psychiatric discourse that utilizes the notion of "chemical imbalance" in explanations of the treatment of depression—that is, a prior chemical imbalance is inferred from a presumptive "balancing" of neurochemistry by antidepressants. Key concepts that are unexplained can result in circular reasoning, illustrated by Moliere's physician who attributed the effect of a soporific to its "dormative powers." A cogent explanation of somatization must spell out exactly what is denoted by the "emotional distress" that is putatively "expressed" somatically and also include valid and reliable methods for measuring it. Can the emotional distress thought to underlie somatization be identified independently of its somatic expression? If not, what is the epistemological status of our model or theory? Are we simply assuming a priori that there is some emotional basis for any unexplained physical symptom? If so, we have engaged in question begging rather than explanation.

Fortunately for scientists, healthcare providers, and patients, various diagnoses and the forms of psychotherapy and pharmacotherapy applied are warranted, for the most part, by empirical findings that validate clinical practices rather than confirm underlying theory. There are few cases in psychiatry in which a treatment can be shown to produce clinical benefits because it affects a well-understood mechanism that is implicated conclusively in pathogenesis. We simply do not have theories or models of mental disorders that have been validated in the manner of our theories of, for example, infectious diseases. Our technologies of healing are legitimized not by the verification of the underlying theory, but by the efficacies of these technologies. Research on treatments for mental disorders is much more akin to industrial practices of product testing than to theory-based applied science. Fortunately, effective clinical interventions need not wait upon validated scientific theories of psychiatric disorder. We and our colleagues, as did the empirics of old, put our money on pragmatic observable results of interventions, as opposed to armchair speculation about the true nature of things.

This is not to say that we practice "dust-bowl" empiricism or are unguided or undisciplined by a priori assumptions, models, and theories. Our approach to somatization draws heavily on several sources: (1) stress research and the stress-management and self-regulation literature; (2) the contemporary psychology of emotion and those

experiential approaches to psychotherapy that emphasize emotional processing; (3) social learning theory and the cognitive-behavioral interventions that are predicated on cognitive-appraisal and conditioning models of behavior; (4) role theory as it derives from sociocultural analyses of illness behavior by sociologists and medical anthropologists. In Chapter 3, we elaborate the rationale for our treatment, but first, in Chapter 2, we define more precisely the problem we wish to treat.

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