Section I

Domains of Concepts, Theories, and Treatments

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Depression in Chronic Physical Illness: A Behavioral Medicine Approach

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In this chapter, we address the prevalence, consequences, and presumed causes of unipolar depression in chronic, non-lethal, physical illness. We begin by outlining the central role of chronic physical illness (CPI) in medicine. Next, we consider the various manifestations of depression in CPI, focusing on the distinction between categorical approaches to depression (i.e., a diagnosable condition such as major depressive episode) and dimensional approaches (i.e., elevated levels of depressive symptoms in the absence of a diagnosable episode) to depression. From a behavioral medicine point of view, we argue, it makes more sense to address depression as a continuous dimension rather than to distinguish between diagnosable and "subsyndromal" depression. Next, we present a tentative model accounting for depression in CPI, which focuses on *person–context exchanges.* We conclude by discussing the implications of this view for assessment/screening, treatment, and prevention.

Chronic Physical Illness: Definition, Prevalence, Costs

The World Health Organization (WHO) defines chronic diseases as diseases of long duration and generally slow progression. According to the US National Center for Health Statistics, a disease lasting 3 months or more is considered chronic (http://www.who.int/topics/chronic_diseases/en). Chronic diseases refer to a variety of medical and functional somatic conditions (i.e., cancer, chronic kidney disease, chronic obstructive pulmonary disease, chronic pelvic pain, chronic pain, chronic

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respiratory diseases, diabetes, epilepsy, fibromyalgia, headache, heart disease, rheumatoid arthritis, systemic lupus erythematosus, etc.) characterized by different levels of life-threatening conditions (terminal vs. non-terminal conditions), symptom severity and duration (constant, episodic flare-ups or exacerbations, or remission with an absence of symptoms for long periods of time), illness appearance (suddenly or through an insidious process), etc. As opposed to an acute disease, which is typically characterized by an abrupt onset of symptoms that terminates in a relatively short period, either with recovery or with death, chronic illness might continue endlessly, and often becomes a defining part of the individual's life experience.

Chronic illnesses constitute a growing proportion of the total global disease burden (Katon & Ciechanowski, 2002; Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; Welch, Czerwinski, Ghimire, &Bertsimas, 2009). Over the last 50 years, chronic illnesses have steadily overtaken acute medical conditions as the primary cause of disability and use of health services in the United States. Studies show that 45% of the US population is afflicted with CPI, which accounts for 78% of health expenditure (Anderson & Horvath, 2004; Holman, 2004). Chronic illness is predicted to become ever more prevalent as populations age across developed countries and effective treatment is found for acute conditions. In fact, it is predicted that, by the year 2020, CPI will account for 60% of the global disease burden (Murray & Lopez, 1997; Welch et al., 2009).

A distinction between "disease" and "illness" is made in (behavioral) medicine in general, and particularly with respect to CPI. While the term *disease* pertains to the pathophysiology underlying the syndrome/symptoms, (e.g., an alteration in structure and function), the term *illness* pertains, more broadly, to the *human experience* of symptoms and suffering, and pertains to how a disease is perceived, lived with, and responded to by an individual and his family (Larsen & Lubkin, 2009, p. 4). Related to this distinction, Curtin and Lubkin (1995, pp. 6–7), define chronic illness as the irreversible presence, accumulation, or latency of disease states or impairments that involve *the total human environment* for supportive care and self-care, maintenance of function, and prevention of further disability.

In accord with the aforementioned definition, *chronic illness* may be considered a *chronic stressor*. As explained in more detail in the following text, stress may be defined as a threat (or perceived threat) to the organisms' allostasis or dynamic equilibrium (McEwen, 2007), caused by a physical and/or psychosocial burden (Van Houdenhove, Egle, & Luyten, 2005). Stress is thought to influence health both indirectly by promoting behavioral coping responses detrimental to health and by activating physiological systems (i.e., the sympathetic nervous system and directly by its effects on the hypothalamic–pituitary–adrenal (HPA) axis; Cohen, 2004; Lupien, McEwen, Gunnar, & Heim, 2009). Prolonged or repeated activation of these systems is thought to place persons at risk for the development of a range of physical and psychiatric disorders (Anda et al., 2006; Luyten, Van Houdenhove, Lemma, Target, & Fonagy, in press), and depression in particular (Monroe & Reid, 2009; Pae et al., 2008).

Prevalence of Depression in Chronic Physical Illness

It is therefore of no surprise that unipolar depression is highly prevalent in CPI. Depression ranks fourth for disability-adjusted life-years worldwide (Insel & Charney, 2003), and is estimated to rise to second by 2020 (Murray & Lopez, 1997). Every year, 6% of adults will suffer from depression, and more than 15% of the population will experience a depressive episode during their lifetime (Pilling, Anderson, Goldberg, Meader, & Taylor, 2009). Recently, the 1-year prevalence of a depressive episode among the chronically physically ill, based on ICD-10 criteria, has been shown to range between 9.3% and 23% and to be significantly higher than the likelihood of having depression in the absence of a CPI (Moussavi et al., 2007).

Extensive research has demonstrated the association between depression and high utilization of medical services, indicating significantly higher medical costs among patients with either depressive symptoms or major depression in comparison to non-depressed patients (Katon & Ciechanowski, 2002; Welch et al., 2009). This increase in costs is seen in a variety of categories, including primary care visits, medical specialty visits, lab tests, pharmacy costs, inpatient medical costs, and mental health visits. This was also found after adjusting for chronic medical illness. Additionally, depressed patients were found to have higher costs than non-depressed elderly (Katon & Ciechanowski, 2002).

Welch et al. (2009) have shown that depressed patients had higher non-mental-health costs than non-depressed patients in 11 comorbid illnesses studied, while the per-patient difference in non-mental-health cost between depressed and non-depressed patients ranged from US$1,570 in obesity to US$15,240 in congestive heart failure. The ratio of cost between non-depressed and depressed patients ranged from 1.5 in obesity to 2.9 in epilepsy. Depression was associated with significantly higher cost of non-mental healthcare in 10 of 11 chronic comorbid diseases studied. Even while controlling for number of chronic comorbid diseases, depressed patients had significantly higher costs than non-depressed patients. Though the magnitude of the cost difference was similar to that reported in previous studies, the consistency of the magnitude across 11 chronic comorbid diseases has not been previously reported. Also, the largest components of higher cost in depression were higher pharmaceutical and outpatient costs. Further research is necessary to determine the underlying reasons for these variations. However, these differences in utilization do indicate that depressed patients not only saw doctors more often, but also were prescribed non-mental-health drugs at higher cost or in greater quantity than non-depressed patients.

Though the increased cost of general medical services associated with depression has been established in several different medical settings, using cost of services as a measure of utilization of care, the cause of higher cost in depressed patients remains unclear. Thus, it is unclear whether these individuals suffer chronic comorbid illnesses of greater severity, or if they are seeking more medical care than non-depressed individuals, independently of illnesses severity, or whether they have poor

compliance with medical care or even alterations in pathophysiology triggered by depression.

Depression: Categorical and Dimensional Approaches

However, what exactly *is* depression? For the last two and a half decades, depression researchers have been discussing whether depression is a binary, all-or-none, clinical entity such as the ones described in leading psychiatric diagnostic manuals such as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR, published by the American Psychiatric Association, 2000) or the *International Statistical Classification of Diseases and Related Health Problems–10* (ICD-10; World Health Organization, 2008), as opposed to a continuous variable denoting various levels of severity that might be ordered along a known and measurable continuum, such as scores on the Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977). Two scientific approaches were taken in order to resolve the debate: employment of new taxometric procedures to examine the extent to which depression is a continuum or a taxon on the one hand and the systematic examination of impairment associated with "subsyndromal depression" on the other, namely, whether elevated levels of depressive symptoms, in the absence of a formal DSM/ICD diagnosis, are associated with impairment. Evidence pertaining to both approaches is briefly reviewed.

Developed by Waller and Meehl (1998), taxometics is a statistical procedure aimed at identifying categories from dimensions, so as to establish the optimal indicators and base rates of the putative category (taxon). Simply put, taxometrics purports to provide an answer to the question "is a certain variable (e.g., depression) binary or continuous?" The idea behind taxometrics is that valid indicators of a potential taxon intercorrelate in which taxon members and non-members are mixed, but do not correlate in "pure" samples. Thus, the size of the correlation between key indicators of depression (e.g., self-report measures such as the CESD or the Beck depression inventory–II [BDI-II]; Beck, Steer, & Brown, 1996), computed for samples comprised of either depressed, non-depressed, and mixed individuals, should indicate the existence – or not – of a depression taxon. The problem, however, is that there are few studies that rely on this sophisticated procedure in depression research, and, thus far, results have been mixed, some consistent with the presence of a depressive taxon while some are not (e.g., Beach & Amir, 2003, Franklin, Strong, & Greene, 2002; Grove et al., 1987; Hankin, Fraley, Lahey, & Waldman, 2005; Ruscio & Ruscio, 2000; Whisman & Pinto, 1997; for a review, see Pettit & Joiner, 2006, p. 15).

Another approach is to examine whether elevated levels of depressive symptoms, assessed as a continuous variable, are associated with psychosocial and medical impairment, regardless of the presence or absence of a depressive taxon. This approach might be extended by comparing individuals with a formal diagnosis of major depressive disorder (MDD), with those who do not meet criteria for such a

diagnosis, but who have elevated scores on a depression measure. Here, the pattern is clear: elevated scores of depressive symptoms on the CESD, BDI-II, or other measures are associated with substantial psychosocial impairment, which is sometimes equivalent to the impairment experienced by people with MDD (e.g., Judd, Akiskal, & Paulus, 1997).

Taking into consideration the two lines of evidence, we conclude that, for practical purposes, unipolar depression in chronic illness should be considered as serious even when it is subsyndromal, namely, when patients reported elevated levels of depression on a self-report measure, but do not meet criteria for an MDD diagnosis. Accordingly, henceforth, we use the term "depression" in its wider use, denoting elevated levels of depressive symptoms.

Role of Depression in CPI

Various models of the relationship between depression and CPI have been proposed, and evidence seems to support in part each of these approaches.

First, CPI might *lead to* depression. For instance, in a prospective study of nondepressed elderly living in the community, the most common stressful life event that was associated with the onset of major depression was the development of a life-threatening medical illness in the respondent or his/her spouse (Wells, Golding, & Burnham, 1988). Studies addressing the onset of depression as a secondary consequence of the existence of a chronic illness were conducted in the context of a variety of chronic medical conditions, consistently pointing to higher levels of depression associated with chronic illnesses (Harpole et al., 2005; Katon & Schulberg, 1992; Moussavi et al., 2007). For example, while controlling for sociodemographic differences between groups, depression was found to be more common among patients with rheumatoid arthritis than in healthy individuals (Dickens, McGowan, Clark-Carter, & Creed, 2002). In a large, nationally representative sample, very high 12-month prevalence and age/sex-adjusted odds of major depression were found in individuals with common chronic medical conditions (i.e., hypertension, diabetes mellitus, coronary artery disease, congestive heart failure, stroke or cerebrovascular accident, chronic obstructive pulmonary disease, and end-stage renal disease). Specifically, the 12-month prevalence estimates of major depression ranged from 7.9% to 17%, and the age/sex-adjusted odds of major depression ranged from 1.96 to 3.56 (Egede, 2007).

Second, depression might be *a causal factor* in CPI, such as ischemic heart disease (IHD), stroke, cancer, and epilepsy (e.g., Evans et al., 2005; Frasure-Smith, Lespérance, & Talajic, 1993). In a large, population-based sample, based on the US Health and Retirement Survey (HRS), depression was significantly associated with the onset of diabetes, heart problems, and arthritis, but not cancer, as examined during a 12-year follow-up study among older working-age adults (ages 50–62), even while controlling for demographics, health risk indicators (body mass index [BMI] and smoking), income, and other relevant variables. Hence, working-age

older people with depression at baseline were at significantly higher risk of developing chronic illnesses (Karakus & Patton, 2011).

Another very large population-based worldwide study demonstrated that comorbid state of depression incrementally worsens health as compared with depression alone, with any of the chronic illnesses alone, and with any combination of chronic illnesses without depression. Depression comorbid with other chronic illnesses produced significantly greater decrements in health than from one or more chronic illnesses, even after adjustment for socio-demographic, country of origin, and economic factors (Moussavi et al., 2007).

Studies have also associated depression with increased morbidity and mortality (Bair, Robinson, Katon, & Kroenke, 2003; Katon, 2003; Moussavi et al., 2007). Moreover, another review showed that the impact of depression on chronic diseases (i.e., asthma, arthritis, cardiovascular disease, cancer, diabetes, and obesity), as well as the tendency of chronic disease to worsen symptoms of depression, both accounted for the significant associations found between depression and chronic diseases (Chapman, Perry, & Strine, 2005).

Third, there is evidence for *reciprocal relationships* between chronic illness and depression. For instance, reciprocal relationships have been found between chronic pain and depression (for a review, see Bair, Robinson, Katon, & Kroenke, 2003), and between depression and neurological diseases such as Parkinson's disease, epilepsy, and Alzheimer's disease (Evans et al., 2005). Evidence suggests that depression might develop either as a consequence of the psychological stress associated with neurological diseases, or as a result of underlying neurodegenerative process (Zubenko et al., 2003). Yet, a history of depression has also been depicted as a risk factor for the development of Alzheimer's disease (Green et al., 2003) and epilepsy (Forsgren & Nystrom, 1990; Hesdorffer, Hauser, Annegers, & Cascino, 2000), suggesting common pathogenic mechanisms. Additionally, Evans et al. (2005) suggested that biological mechanisms (i.e., increased serum glucocorticoids, catecholamines, and growth hormone which counter the effects of insulin, insulin resistance, secretion of inflammatory cytokines, HPA axis hyperactivity, and increases in plasma cortisol) might link depression with chronic illnesses such as diabetes and congestive heart failure. Similarly, pain and depression might occur simultaneously because of their respective neurochemical associations with serotonin and norepinephrine, and altered levels of these neurotransmitters might affect changes that precipitate the occurrence of pain and depression (Evans et al., 2005). Congruent with this assumption, there is increasing evidence that depression and several chronic pain disorders are part of a spectrum of disorders with high familial aggregation (Hudson et al., 2003).

Diverse behavioral factors might account for the effect of depression on chronic illness and its adverse course. These are listed in the following text:

Poor self-care and non-adherence (i.e., non-compliance with treatment recommendations). Self-management of chronic illness includes working effectively with healthcare providers, self-monitoring (i.e., checking blood glucose),

implementing medication regimens, following prescribed diet and exercise regimens, quitting smoking, and minimizing drinking as well as minimizing the impact of medical illness on social role functioning. A concerning large percentage of medical patients do not consistently adhere to the recommendations of their physicians for prevention or treatment of acute or chronic conditions (i.e., by not taking their medication correctly, persist in lifestyles that endanger their health, etc.). Non-adherence can result in exacerbation of illness, incorrect diagnoses, as well as patient and physician frustration, and generally has a consistently negative effect on treatment outcomes. Medical patients may be non-compliant for many reasons, including their disbelief in the efficacy of treatment, barriers such as adverse effects and financial constraints, and lack of help and support from family members (Katon & Ciechanowski, 2002). Based on a large meta-analysis, DiMatteo, Lepper, and Croghan (2000) found that depression, but not anxiety, was strongly associated with the risk of being non-compliant with medical treatment recommendations.

There might be a variety of reasons as to why depression is significantly associated with non-adherence. Depression may hamper self-management of chronic illnesses, including impairments in cognitive functioning, cognitive focus, energy, and motivation caused by depression that in turn might have an effect on the ability and willingness of patients to follow through with treatment. Also, depression involves a sense of hopelessness and even lack of self-efficacy and optimism that any action will be worthwhile that might also hamper the ability and motivation to adhere to treatment. Finally, social support, which is eroded by depression, might complicate the self-management of chronic physical illness (Gallant, 2003), and lead to poor adherence (DiMatteo, 2004).

Amplification of symptoms and illness intrusiveness

Substantial evidence demonstrates that patients with both mental disorders and subsyndromal psychological distress report significantly more physical symptoms than non-distressed populations, even while controlling for the severity of medical illness (Katon & Ciechanowski, 2002). One of the challenges patients with chronic medical illness are dealing with is managing to live and adapt to the chronic medical symptoms caused by their medical illness, and most patients habituate to these chronic aversive symptoms quite well (at least until they reach severe levels that might markedly affect functioning). However, an extensive data suggest that having comorbid depressive illnesses in patients with chronic medical illness interferes with this adaptation process and is associated with amplification of both symptoms from the CPI as well as physical symptoms associated with other body organ systems. Several studies have shown that, when major depression was effectively treated among chronically ill patients, they reported being able to cope more effectively with their illness, and that their symptoms of medical illness were less distressing despite little change in objective measures of their medical disorder (Borson et al., 1992; Sullivan, Katon, Russo, Dobie, & Sakai, 1993).

Related to the issue of symptom amplification is *illness intrusiveness.* Illness intrusiveness pertains to encompassing illness- and treatment-induced

disruptions to the individual's lifestyles, valued activities, and interests. Conceptualized as a facet of the chronic illness experience that is common across conditions, illness intrusiveness is a fundamental determinant of subjective well-being (Devins, Edworthy, & ARAMIS Lupus State Models Research Group, 2000; Devins et al., 2001). The concept's central assumption is that chronic illness and its treatment affect subjective experience through illness intrusiveness, an intervening variable, which is also affected by moderating psychological and social factors (Devins et al., 2000, 2001). The applicability of the illness intrusiveness concept to CPI has been demonstrated in various illnesses (bone marrow transplants, irritable bowel syndrome, rheumatic diseases, SLE), cardiac events, and others (Devins et al., 2000, 2001). Evidence for a negative reciprocal longitudinal relationship between chronic illness and depression was also found in systemic lupus erythematosus (SLE) patients (Schattner, Shahar, Lerman, & Abu Shakra, 2010). While increased levels of depression were predicted by illness intrusiveness and symptom concealment, initial (baseline) levels of depression were associated with an increase in illness intrusiveness over time, thus suggesting a reciprocal relationship between depression and illness intrusiveness in SLE.

Effect on the social context. As noted, depression is known for having a deleterious effect on the social context (Pettit & Joiner, 2006), which, in turn, is likely to exacerbate the course of chronic illness (Ranjan, 2001). According to Wells et al. (1989), patients with major depression perceive their social and occupational functioning, as well as their general health, as more impaired than patients with several other medical disorders. Also, when major depression was comorbid with a medical disorder, there was an additive functional impairment. Subsyndromal depressive symptoms were also correlated with additive disability among patients with chronic medical illness (Wells et al., 1989).

A Heuristic, Behavioral-Medicine Perspective on Depression in Chronic Illness: Depressive Vulnerability and Person–Context Exchanges

Based on the literature reviewed, we propose a heuristic perspective on depression in CPI. Faithful to the mission of this book, this perspective is behavioral, namely, it is based on psychosocial research identifying key variables and processes in depression, and – more recently – in depression in CPI. Central to this perspective are two constructs: depressive vulnerability and person–context exchanges.

Depressive vulnerability pertains to individuals' *proclivity* to develop depressive (and related, anxious, and other) symptoms, particularly in the face of life stress (Shahar, 2001). Individuals' cognitive–affective (i.e., the ways in which they think about the world) or personality (Blatt, 2004, 2008) style plays a key role in explaining depressive vulnerability. There are numerous dimensions of psychological

vulnerability "out there." These include, but are not limited to, personality features such as dependency, sociotropy, autonomy, perfectionism, rejection sensitivity, dysfunctional attitudes, harm avoidance, neuroticism, anxiety sensitivity, looming maladaptive style, and self-criticism). The last four decades, however, have brought about an appreciable amount of research, implicating two dimensions as "arch-villains" in explaining vulnerability to depression: neuroticism and self-criticism (Kotov, Gamez, Schmidt, & Watson, 2010; Steel, Schmidt, & Shultz, 2008; Blatt, 2004, 2008; Shahar, 2001, respectively).

Neuroticism pertains to a broad, higher-order personality factor referring to an enduring proclivity for experiencing negative affect. Neuroticism is generally considered to be a major risk factor for psychopathology, and depression in particular (Costa & McCrae, 1992; Goldberg, 1993; Lahey, 2009). However, important criticism has been leveled against this construct (e.g., Spitzer, First, Shedler, Westen, & Skodol, 2008). For instance, it has been suggested that, because this dimension is defined from the outset as the proclivity to experience negative affect, the use of this construct to predict emotional disorder is circular (Farmer et al., 2002). On the other hand, the predictive power of neuroticism vis-à-vis various forms of psychopathology, not just emotional disorders, is so impressive that it should not be dismissed out of hand. Neuroticism appears to tap into the temperamental underpinning of psychophysiological vulnerability to depression, challenging other putative vulnerability dimensions to demonstrate their predictive power over and above neuroticism. Of all the present dimensions, self-criticism is perhaps the only personality–cognitive one meeting this challenge (Bareket-Bojmel & Shahar, 2011; Clara, Cox, & Enns, 2003; Dunkley, Sanislow, Grilo, & McGlashan, 2006; Johnson, 2003).

Self-criticism is a more specific personality dimension and pertains to individuals' tendency to adopt a punitive stance toward the self once self-standards are not met (Shahar, 2001). Studies suggest that self-criticism, like neuroticism, is a transdiagnostic vulnerability factor, which has been implicated in the onset and course of depression, anxiety, eating, and personality disorders (for a review, see Blatt, 1995, 2004; Luyten & Blatt, 2011), as well as suicide. It has also been shown to negatively affect the course of chronic illness (Luyten et al., 2011)

For instance, recent research attests to the role of self-criticism in CPI, such as in chronic fatigue (Kempke et al., 2011 ; Luyten et al., 2011) and pain-related disorders (Rudich, Lerman, Weksler, Gurevitch, & Shahar, 2008; Rudich, Lerman, Gurevitch, & Shahar, 2010; Lerman, Shahar, & Rudich, 2012). Studies suggest that self-criticism is associated with increased vulnerability for these disorders and also negatively influences their course (Kempke, Van Den Eede, et al., in press; Luyten et al., 2011). Congruent with the active person–context exchange model proposed in this chapter, Luyten and colleagues found that self-criticism and related traits (e.g., persistence and "action-proneness") were not only associated with increased stress sensitivity in patients with chronic fatigue syndrome (CFS), but also with the generation of daily stress, leading to increased levels of depression in the daily course

of life (Luyten et al., 2011). In a subsequent study, these authors demonstrated a prospective relationship between self-criticism and daily fatigue and pain symptoms in CFS patients (Kempke, Luyten, et al., 2013). Moreover, they also demonstrated that self-criticism was clearly distinct from adaptive strivings and adaptive perfectionism (Kempke, Van Houdenhove, et al., 2011) and was related to low self-esteem, leading to a downward spiral of increased levels of depression, fatigue, and pain over time (Kempke, Luyten, Van Houdenhove, et al., 2011). Studies in this context suggest that self-criticism may be a coping style to compensate feelings of low self-esteem related to a history of intrusive parenting and/or trauma (Luyten et al., in press; Soenens, Vansteenkiste, & Luyten, 2010). The clinical importance of these findings cannot be underestimated, particularly since self-criticism, as in other conditions (Blatt, Zuroff, Hawley, & Auerbach, 2010), has been negatively related to treatment outcome in both CFS and chronic pain conditions over and above cognitive factors and severity of depression (Kempke et al., 2010; Kempke, Luyten, Van Wambeke, Coppens, & Morlion, 2011).

Studies in this area are also beginning to shed more light on the mechanisms involved in the relationship between self-criticism, depression, and chronic illness. Some evidence suggests, congruent with studies outlined previously, that self-criticism is associated with a dysregulation of the main human stress system, the HPA axis, leading to a cascade of psychological (e.g., depression) and somatic consequences (e.g., dysregulation of immune and pain processing systems), resulting in increased levels of depression, fatigue, and pain.

In Israel, a similar line of inquiry has focused on chronic pain. Patients with chronic pain conditions (e.g., fibromyalgia, chronic low back and neck pain, CRPS, neuropathic pain, headache) were assessed prior to a first visit to a pain clinic as to their pain level, depression, and self-criticism. Subsequent to the visit, physicians were assessed as to their pessimism/optimism regarding patients' prognosis. Self-criticism was the sole predictor of physicians' pessimism regarding prognosis (Rudich et al., 2008). A follow-up on a subset of these patients revealed that physicians' pessimism predicted an increase in the affective component of pain, as well as in depression (Rudich et al., 2010). Moreover, in two separate studies, self-criticism was shown to interact with the affective, but not sensory, component of pain to prospectively predict an increase in depression (Lerman, Shahar, & Rudich, 2011), anxiety, and pain-related disability (Lerman, Shahar, Brill, & Rudich, in preparation). In the latter study, self-criticism also predicted an increase in both affective and sensory pain.

According to the *congruency hypothesis* (Hammen, Marks, Mayol, & DeMayo, 1985; Zuroff & Mongrain, 1987), depression and related psychopathology are expected to ensue from interactions between specific psychological vulnerability and vulnerability-congruent life events. Accordingly, self-criticism is expected to interact with failure-related stress, but not with other types of life stress (e.g., interpersonal strife), in predicting depression's onset and/or maintenance. Nevertheless, self-criticism has been found to interact *with all types of life stressors in predicting* psychopathology (Lassri, Soffer-Dudek, Lerman, Rudich, & Shahar, 2012; Lerman

et al., 2011; Shahar, Joiner, Zuroff, & Blatt, 2004), attesting to its broad and general vulnerability status. More importantly, various investigators, primarily in Canada and Israel (e.g., Dunkley, Zuroff, & Blankstein, 2003; Mongrain, 1998; Mongrain, Vettese, Shuster, & Kendal, 1998; Shahar & Priel, 2003; Zuroff, 1992), have demonstrated that self-criticism confers *an active vulnerability* (Shahar & Priel, 2003): it is associated with stress generation, particularly in close relationships, and also "degenerates" (fails to generate) protective interpersonal factors such as positive events and social support (Mongrain, 1998; Priel & Shahar, 2000). As noted in the preceding text, it derails interpersonal relationships both within and outside treatment, thereby impeding therapeutic response (Shahar, Blatt, Zuroff, Krupnick, & Sotsky, 2004; Shahar, Blatt, Zuroff, & Pilkonis, 2003; see Shahar, 2006, see Blatt et al., 2004, for a review). The aforementioned studies attesting to the demoralizing effect of patients' self-criticism on their physician's evaluation of their prognosis exemplify this active nature of self-critical vulnerability.

In fact, we deem active person–context exchanges to be central to the understanding of the role of depression in chronic illness. In Figure 1.1, we present a tentative model describing such exchanges. As shown in the figure, CPI patients' depressive vulnerability (e.g., self-criticism, but also other vulnerabilities such as dependency, pessimism) interferes with relationships in patients' social environment (SE, namely, relationships with family members and friends), as well as with patient–provider relationships (PP), in turn leading to depression. Such an active, albeit pernicious, interpersonal cycle might be activated, or exacerbated, by the onset and fluctuations of chronic illness, and is likely to lead to depression. In turn, depression might complicate the course of chronic illness via the earlier-mentioned mechanisms including non-adherence; symptom amplification and illness intrusiveness; adverse effect on the social context; and the effect on stress, immunity, and pain processing mechanisms, leading to a vicious cycle. Indeed, extant research suggests that dysregulation of the HPA axis typical of depression (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008) is associated with suppression of natural killer cell function and cell-mediated immunity, abnormal activation of innate immunity, the release of proinflammatory cytokines, often giving rise to a so-called "sickness response," a combination of feelings of lethargy, general malaise, sleepiness, concentration difficulties, headache, mild fever, and widespread musculoskeletal pain (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008).

Finally, the stress associated with CPI as well as depression also may lead to impairments in (embodied) mentalizing, i.e., the capacity to understand the (bodily) self in terms of intentional mental states. As a result, patients revert to the so-called "physical stance," being only able to see the body as a dysfunctional organism or machine, instead of the embodied seat of emotions and feelings (Luyten, Fonagy, et al., 2012; Luyten, Van Houdenhove, et al., 2012). In this mode, patients often cling desperately on to some hoped-for biological "miracle" cure, often leading to desperation in professionals and those close to the patient. Others are often seen as unsupportive, uncaring, or indifferent. Although understandable, the dramatic effects that CPI may have on patients' reflective capacities, both with regard to the

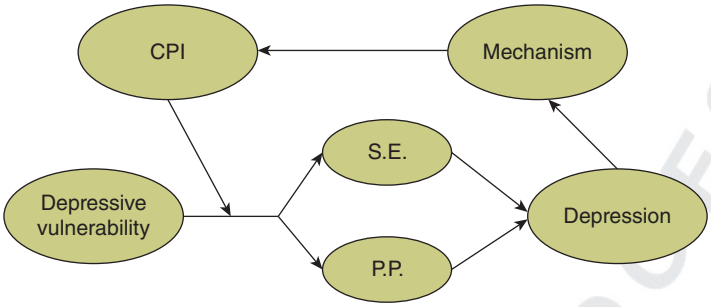

Figure 1.1
Notes: CPI = chronic physical illness, S.E. = social environment, P.P. = patient–provider relationships

embodied self and others, often further puts patient–provider relationships under pressure, is associated with behaviors that perpetuate and exacerbate symptoms (e.g., catastrophizing, unhelpful illness beliefs and behaviors, increased feelings of helplessness and depression), and hinder acceptance and/or attempts to find a new balance in life.

Practice Implications and Recommendations

The implications of the active person–context exchange view on CPI and depression proposed in this chapter is that one has to adopt a person-centered and not purely disorder-centered approach to patients with CPI, attending to the complexity of interactions among psychosocial and biological factors, and between the person and his or her context (family, professionals). This necessarily requires narrative competence of the clinician (Charon, 2001): the ability to appreciate and understand the complex interactions between the patient's disease, his or her life history, and past and current (interpersonal) context.

A good screening for depression (including depression history to assess potential vulnerability for depression) is crucial in this regard, and appropriate treatment should be considered when feelings of depression exceed "normal" feelings of dejection and despondence that are associated with CPI. In addition, the focus should not only be on vulnerabilities of the individual, but also on his or her strengths and resilience. Reinforcing areas of strength and resilience in the person and/or his context may often be more important than focusing on vulnerabilities.

This is particularly true for those with high levels of self-criticism. For these individuals, normalizing possible feelings of depression and despair when confronted with CPI may already bring considerable relief, as these individuals often struggle to admit that they feel despondent. Similarly, pointing out the high

emotional and interpersonal costs of attitudes and coping strategies that are associated with self-criticism (e.g., the belief that one should be able to cope with problems without the help of others) is often extremely helpful to these patients. Yet, when self-critical depressive tendencies are more persistent, patients may benefit from a range of more specialized interventions, ranging from interventions that specifically address self-criticism and related features (Arpin-Cribbie, Irvine, & Ritvo, in press; Brooks, Rimes, & Chalder, 2011; Gilbert, 2009; Mongrain & Trambakoulos, 2007; B. Shahar et al., 2012) to specialized treatments that target vulnerability for depression, such as cognitive-behavioral therapy (Beck, Rush, Shaw, & Emery, 1979) or brief dynamic therapy (Lemma, Target, & Fonagy, 2011; Luyten, Van Houdenhove, et al., 2012).

Conclusions

This chapter clearly attests to the importance of considerations concerning the role of depression in CPI. Depression can be a pernicious consequence of CPI. Also, depression can have a massive impact on the course and prognosis of CPI, both directly and indirectly thought its effect on adherence and relationships with professionals, and the patients' support system.

In addition, we have emphasized the importance of attending to possible depressive vulnerability. CPI seriously threatens the person's resources, and, particularly in those with low levels of resilience and high levels of self-criticism, this can lead to a depressive response. Psychoeducation about the impact of CPI, the effect of self-criticism on coping, and the social environment promises to lead to a more positive course. However, in severely depressed patients, appropriate treatment is indicated.

References

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Anda, R., Felitti, V., Bremner, J., Walker, J., Whitfield, C., Perry, B., . . . Giles W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience, 256,* 174–186.

Anderson, G., & Horvath, J. (2004). The growing burden of chronic disease in America. *Public Health Reports, 119*, 263–270.

Arpin-Cribbie, C., Irvine, J., & Ritvo, P. (in press). Web-based cognitive-behavioral therapy for perfectionism: A randomized controlled trial. [doi: 10.1080/10503307.2011.637242]. *Psychotherapy Research.*

Bair, M. J., Robinson, R. L., Katon, W. J., & Kroenke, K. (2003). Depression and pain comorbidity. *Archives of Internal Medicine, 163,* 2433–2445.

Bareket-Bojmel, L., & Shahar, G. (2011). Personality discloses: Emotional and interpersonal effects of self-criticism, openness to experiences, and experiential avoidance on self-disclosure online. *Journal of Social & Clinical Psychology, 30,* 732–760.

Beach, S. R. H., & Amir, N. (2003). Is depression taxonic, dimensional, or both? *Journal of Abnormal Psychology, 112,* 228–236.

Beck, A. T., Rush, J., Shaw, B., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for Beck depression inventory II (BDI-II).* San Antonio, TX: Psychology Corporation.

Blatt, S. J. (1995). The destructiveness of perfectionism: Implications for the treatment of depression. *American Psychologist, 50,* 1003–1020.

Blatt, S. J. (2004). *Experiences of depression.* Washington, DC: American Psychological Association Press.

Blatt, S. J. (2008). *Polarities of experiences.* Washington, DC: American Psychological Association Press.

Blatt, S. J., Zuroff, D. C., Hawley, L. L., & Auerbach, J. S. (2010). Predictors of sustained therapeutic change. *Psychotherapy Research, 20,* 37–54.

Borson, S., McDonald, G. J., Gayle, T., Deffenbach, M., Lakshmi-Narayan, S., & Van Tuinen, C. (1992). Improvement in mood, physical symptoms, and function with nortriptyline for depression in patients with chronic obstructive pulmonary disease. *Psychosomatics, 33,* 190–201.

Brooks, S. K., Rimes, K. A., & Chalder, T. (2011). The role of acceptance in chronic fatigue syndrome. *Journal of Psychosomatic Research, 71*, 411–415.

Chapman, D. P., Perry, G. S., & Strine, T. W. (2005). The vital link between chronic disease and depressive disorders. *Preventing Chronic Disease, 2,* 1–10. Available at: www.cdc.gov/pcd/issues/2005/jan/04_0066.htm

Charon, R. (2001). Narrative medicine: A model for empathy, reflection, profession, and trust. *JAMA: The Journal of the American Medical Association, 286,* 1897–1902.

Clara, I. P., Cox, B. J., & Enns, M. W. (2003). Hierarchical models of personality and psychopathology: The case of self-criticism, neuroticism, and depression. *Personality & Individual Differences, 35,* 91–99.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*, 676–684.

Costa, P. T., Jr., & McCrae, R. R. (1992). *Revised NEO personality inventory (NEO-PI-R) and NEO five-factor inventory (NEO-FFI) professional manual.* Odessa, FL: Psychological Assessment Resources.

Curtin, M., & Lubkin, I. (1995). What is chronicity? In I. Lubkin (Ed.), *Chronic illness: Impact and intervention* (3rd ed.). Sudbury, MA: Jones & Bartlett.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience, 9,* 46–56.

Devins, G. M., Dion, R., Pelletier, L. G., Shapiro, C. M., Abbey, S., Raiz, L. R., . . . Edworthy S. M. (2001). Structure of lifestyle disruptions in chronic disease: A confirmatory factor analysis of the illness intrusiveness ratings scale. *Medical Care, 39,* 1097–1104.

Devins, G. M., Edworthy, S. M., & ARAMIS Lupus State Models Research Group. (2000). Illness intrusiveness explains race-related quality-of-life differences among women with systemic lupus erythematosus. *Lupus, 9,* 534–541.

Dickens, C., McGowan, L., Clark-Carter, D., & Creed, F. (2002). Depression in rheumatoid arthritis: A systematic review of the literature with meta-analysis. *Psychosomatic Medicine, 64,* 52–60.

DiMatteo, M. R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology, 23,* 207–218.

DiMatteo, M. R., Lepper, H. S., & Croghan, T. W. (2000). Depression is a risk factor for noncompliance with medical treatment: A meta-analysis of the effects of anxiety and depression on patient adherence. *Archives of Internal Medicine, 160,* 2101–2107.

Dunkley, D. M., Sanislow, C. A., Grilo, C. M., & McGlashan, T. H. (2006). Perfectionism and depressive symptoms three years later: Negative social interactions, avoidant coping, and perceived social support as mediators. *Comprehensive Psychiatry, 47,* 106–115.

Dunkley, D. M., Zuroff, D. C., & Blankstein, K. R. (2003). Self-critical perfectionism and daily affect: Dispositional and situational influences on stress and coping. *Journal of Personality and Social Psychology*, *84*, 234–252.

Egede, L. E. (2007). Major depression in individuals with chronic medical disorders: Prevalence, correlates and association with health resource utilization, lost productivity and functional disability. *General Hospital Psychiatry, 29,* 409–416.

Evans, D. L., Charney, D. S., Lewis, L., Golden, R. N., Gorman, J. M., Krishnan, K. R., . . . Valvo, W. J. (2005). Mood disorders in the medically ill: Scientific review and recommendations. *Biological Psychiatry, 58,* 175–189.

Farmer, A., Redman, K., Harris, T., Mahmood, A., Sadler, S., Pickering, A., & McGuffin, P. (2002). Neuroticism, extraversion, life events and depression. *British Journal of Psychiatry, 181,* 118–122.

Forsgren, L., & Nystrom, L. (1990). An incident case-referent study of epileptic seizures in adults. *Epilepsy Research, 6,* 66–81.

Franklin, C. L., Strong, D. R., & Greene, R. L. (2002). A taxometric analysis of the MMPI-2 Depression Scales. *Journal of Personality Assessment, 79,* 110–121.

Frasure-Smith, N., Lespérance, F., & Talajic, M. (1993). Depression following myocardial infarction: Impact on 6-month survival. *JAMA: The Journal of the American Medical Association, 270*, 1819–1825.

Gallant, M. P. (2003). The influence of social support on chronic illness self-management: A review and directions for research. *Health Education & Behavior, 30,* 170–195.

Gilbert, P. (2009). Introducing compassion-focused therapy. *Advances in Psychiatric Treatment, 15,* 199–208.

Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist, 48,* 26–34.

Green, R. C., Cupples, L. A., Kurz, A., Auerbach. S., Go, R., Sandovnick, D., . . . Farrer, L. (2003). Depression as a risk factor for Alzheimer disease: The MIRAGE study. *Archives of Neurology, 60,* 752–759.

Grove, W. M., Andreasen, N. C., Young, M. A., Keller, M. B., Endicott, J., Lavori, P. W., . . . Reich, T. (1987). Isolation and characterization of a nuclear depressive syndrome. *Psychological Medicine, 11,* 471–484.

Hammen, C., Marks, T., Mayol, A., & DeMayo, R. (1985). Depressive self-schemes, life stress, and vulnerability to depression. *Journal of Abnormal Psychology, 98,* 154–160.

Hankin, B. L., Fraley, R. C., Lahey, B. B., & Waldman, I. D. (2005). Is depression best viewed as a continuum or discrete category? A taxometric analysis of childhood and adolescent

depression in a population-based sample. *Journal of Abnormal Psychology, 114,* 96–110.

Harpole, L. H., Williams, J. W. Jr., Olsen, M. K., Stechuchak, K. M., Oddone, E., Callahan, C. M., ... Unützer, J. (2005). Improving depression outcomes in older adults with comorbid medical illness. *General Hospital Psychiatry, 27*, 4–12.

Heim, C., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2008). The link between childhood trauma and depression: Insights from HPA axis studies in humans. *Psychoneuroendocrinology, 33*, 693–710.

Hesdorffer, D. C., Hauser, W. A., Annegers, J. F., & Cascino, G. (2000): Major depression is a risk factor for seizures in older adults. *Annals of Neurology, 47,* 246–249.

Holman, H. (2004). Chronic disease—the need for a new clinical education. *Journal of the American Medical Association, 292,* 1057–1059.

Hudson, J. I., Mangweth, B., Pope, H. G., Jr., De Col, C., Hausmann, A., Gutweniger, S., . . . Tsuang, M. T. (2003). Family study of affective spectrum disorder. *Archives of General Psychiatry, 60*, 170–177.

Insel, T. R., & Charney, D. S. (2003): Research on major depression. *JAMA: The Journal of the American Medical Association, 289,* 3167–3168.

Johnson, M. (2003). The vulnerability status of neuroticism: Over-reporting or genuine complaints? *Personality & Individual Differences, 35,* 877–887.

Judd, L. L., Akiskal, H. S., & Paulus, M. P. (1997). The role and clinical significance of subsyndromal depressive symptoms (SSD) in unipolar major depressive disorder. *Journal of Affective Disorders, 45,* 5–18.

Karakus, M. C., & Patton, L. C. (2011). Depression and the onset of chronic illness in older adults: A 12-year prospective study. *Journal of Behavioral Health Services and Research, 38,* 373–382.

Katon, W. J. (2003). Clinical and health services relationships between major depression, depressive symptoms, and general medical illness. *Biological Psychiatry, 54,* 216–226.

Katon, W., & Ciechanowski, P. (2002). Impact of major depression on chronic medical illness. *Journal of Psychosomatic Research, 53,* 859–863.

Katon, W., & Schulberg, H. C. (1992). Epidemiology of depression in primary care. *General Hospital Psychiatry, 14,* 237–247.

Kempke, S., Goossens, L., Luyten, P., Bekaert, P., Van Houdenhove, B., & Van Wambeke, P. (2010). Predictors of outcome in a multi-component treatment program for chronic fatigue syndrome. *Journal of Affective Disorders, 126*, 174–179.

Kempke, S., Luyten, P., Claes, S., Goossens, L., Bekaert, P., Van Wambeke, P., . . . Van Houdenhove, B. (2013). Self-critical perfectionism and its relationship to fatigue and pain in the daily flow of life in patients with chronic fatigue syndrome. *Psychological Medicine, 43*, 995–1002.

Kempke, S., Luyten, P., Van Houdenhove, B., Goossens, L., Bekaert, P., & Van Wambeke, P. (2011). Self-esteem mediates the relationship between maladaptive perfectionism and depression in chronic fatigue syndrome. *Clinical Rheumatology, 30,* 1543–1548.

Kempke, S., Luyten, P., Van Wambeke, P., Coppens, E., & Morlion, B. (2011). Self-critical perfectionism, but not interpersonal dependency, predicts treatment response in patients with chronic pain. *Manuscript submitted for publication.*

Kempke, S., Van Den Eede, F., Schotte, C., Claes, S., Van Wambeke, P., Van Houdenhove, B., . . . Luyten, P. (2013). Prevalence of DSM-IV Personality Disorders in Patients with

Chronic Fatigue Syndrome: A Controlled Study. *International Journal of Behavioral Medicine, 20*, 219–228.

Kempke, S., Van Houdenhove, B., Luyten, P., Goossens, L., Bekaert, P., & Van Wambeke, P. (2011). Unraveling the role of perfectionism in chronic fatigue syndrome: Is there a distinction between adaptive and maladaptive perfectionism? *Psychiatry Research, 186,* 373–377.

Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking "big" personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin, 136,* 768–821.

Lahey, B. B. (2009). Public health significance of neuroticism. *American Psychologist, 64,* 241–256.

Larsen, P. D., & Lubkin, I. M. (2009). *Chronic illness: Impact and intervention* (7th ed.). Sudbury, MA: Jones & Bartlett.

Lassri, D., Soffer-Dudek, N., Lerman, S. F., Rudich, Z., & Shahar, G. (2013). Self-criticism confers vulnerability to psychopathology in the face of perceived stress related to missile attacks: Three longitudinal studies. *International Journal of Cognitive Therapy*, *6*, 221–234.

Lemma, A., Target, M., & Fonagy, P. (2011). *Brief dynamic interpersonal therapy: A clinician's guide.* Oxford: Oxford University Press.

Lerman, S. F., Shahar, G., & Rudich, Z. (2012). Self-criticism interacts with the affective component of pain to predict depressive symptoms in female patients. *European Journal of Pain, 16,* 115–122.

Lerman, S. F., Shahar, G., Brill, S., & Rudich, Z. Role of self-critical vulnerability in chronic physical pain: An extensive evaluation. *A paper in preparation.*

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., & Murray, C. J. (2006). Global and regional burden of disease and risk factors, 2001: Systematic analysis of population health data. *Lancet, 367,* 1747–1757.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10,* 434–445.

Luyten, P., & Blatt, S. J. (2011). Integrating theory-driven and empirically-derived models of personality development and psychopathology: A proposal for DSM-V. *Clinical Psychology Review, 31,* 52–68.

Luyten, P., Kempke, S., Van Wambeke, P., Claes, S. J., Blatt, S. J., & Van Houdenhove, B. (2011). Self-critical perfectionism, stress generation and stress sensitivity in patients with chronic fatigue syndrome: Relationship with severity of depression. *Psychiatry: Interpersonal and Biological Processes, 74,* 21–30.

Luyten, P., Fonagy, P., Lemma, A., & Target, M. (2012). Depression. In A. Bateman & P. Fonagy (Eds.), *Handbook of mentalizing in mental health practice* (pp. 385–417). Washington, DC: American Psychiatric Association.

Luyten, P., Van Houdenhove, B., Lemma, A., Target, M., & Fonagy, P. (2012). An attachment and mentalization-based approach to functional somatic disorders. *Psychoanalytic Psychotherapy, 26,* 121–140.

Luyten, P., Van Houdenhove, B., Lemma, A., Target, M., & Fonagy, P. (in press). Vulnerability for functional somatic disorders: A contemporary psychodynamic approach. *Journal of Psychotherapy Integration.*

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews, 87,* 873–904.

Mongrain, M. (1998). Parental representations and support-seeking behaviors related to dependency and self-criticism. *Journal of Personality, 66,* 151–173.

Mongrain, M., & Trambakoulos, J. (2007). A musical mood induction alleviates dysfunctional attitudes in needy and self-critical individuals. *Journal of Cognitive Psychotherapy, 21,* 295–309.

Mongrain, M., Vettese, L. C., Shuster, B., & Kendal, N. (1998). Perceptual biases, affect, and behavior in the relationships of dependents and self-critics. *Journal of Personality & Social Psychology, 75,* 230–241.

Monroe, S. M., & Reid, M. W. (2009). Life stress and major depression. *Current Directions in Psychological Science, 18,* 68–72.

Moussavi, S., Chatterji, S., Verdes, E., Tandon, A., Patel, V., & Ustun, B. (2007). Depression, chronic diseases, and decrements in health: Results from the World Health Surveys. *Lancet, 370,* 851–858.

Murray, C. J., & Lopez, A. D. (1997). Alternative projections of mortality and disability by cause, 1990–2020: Global burden of disease study (comment). *Lancet, 349,* 1498–1504.

Pae, C. U., Luyten, P., Marks, D. M., Han, C., Park, S. H., Patkar, A. A., . . . Van Houdenhove, B. (2008). The relationship between fibromyalgia and major depressive disorder: A comprehensive review. *Current Medical Research and Opinion, 24*, 2359–2371.

Pettit, J. W., & Joiner, T. E. (2006). *Chronic depression: Interpersonal sources, therapeutic solutions.* Washington, DC: American Psychological Association.

Pilling, S., Anderson, I., Goldberg, D., Meader, N., & Taylor, C. (2009). Depression in adults, including those with a chronic physical health problem: Summary of NICE guideline. *British Medical Journal, 339*, 1025–1027.

Priel, B., & Shahar, G., (2000). Dependency, self-criticism, social context and distress: Comparing moderating and mediating models. *Personality and Individual Differences, 28,* 515–525.

Radloff, L. S. (1977). A self-reported depression scale for research in the general population. *Applied Psychological Measures, 1,* 385–401.

Rudich, Z., Lerman, S. F., Gurevich, B., & Shahar, G. (2010). Pain specialists' evaluation of patient's prognosis during the first visit predicts subsequent depression and the affective dimension of pain. *Pain Medicine, 11,* 446–452.

Rudich, Z., Lerman, S. F., Wexler, N., Gurevitch, B., & Shahar, G. (2008). Self-criticism is a stronger predictor of physician's evaluation of prognosis than pain severity and diagnosis. *Journal of Pain, 9,* 210–216.

Ranjan, R. (2001). *Social relations and chronic pain.* New York, NY: Plenum.

Ruscio, J., & Ruscio, A. M. (2000). Informing the continuity controversy: A taxometric analysis of depression. *Journal of Abnormal Psychology*, *109*(3), 473–487.

Schattner, E., Shahar, G., Lerman, S., & Abu Shakra, M. (2010). Depression in systemic lupus Erythematosus: The key role of illness intrusiveness and concealment of symptoms. *Psychiatry, 73,* 329–340.

Shahar, B., Carlin, E. R., Engle, D. E., Hedge, J., Szepsenwol, O., & Arkowitz, H. (2012). A pilot investigation of emotion-focused two-chair dialogue intervention for self-criticism. *Clinical Psychology & Psychotherapy*, *19*, 496–507.

Shahar, G. (2001). Personality, shame, and the breakdown of social ties: The voice of quantitative depression research. *Psychiatry: Interpersonal and Biological Processes, 64*, 229–238.

Shahar, G. (2006). Clinical action: Introduction to the special section on the action perspective in clinical psychology. *Journal of Clinical Psychology, 29,* 1053–1064.

Shahar, G., Blatt, S. J., Zuroff, D. C., & Pilkonis, P. A. (2003). Role of perfectionism and personality disorder features in patients' responses to brief treatment for depression. *Journal of Consulting & Clinical Psychology, 71,* 229–233.

Shahar, G., Blatt, S. J., Zuroff, D. C., Kuperminck, G. P., & Leadbeater, B. J. (2004). Reciprocal relations between depressive symptoms and self-criticism (but not dependency) among early adolescent girls (but not boys). *Cognitive Therapy & Research, 28,* 85–103.

Shahar, G., Joiner, T. E., Jr., Zuroff, D. C., & Blatt, S. J. (2004). Personality, interpersonal behavior, and depression: Co-existence of stress-specific moderating and mediating effects. *Personality and Individual Differences, 36,* 1583–1596.

Shahar, G., & Priel, B. (2003). Active vulnerability, adolescent distress, and the mediating/suppressing role of life events. *Personality and Individual Differences, 35,* 199–218.

Soenens, B., Vansteenkiste, M., & Luyten, P. (2010). Towards a domain-specific approach to the study of parental psychological control: Distinguishing between dependency-oriented and achievement-oriented psychological control. *Journal of Personality, 78,* 217–256.

Spitzer, R. L. M. D., First, M. B. M. D., Shedler, J. P., Westen, D. P., & Skodol, A. E. M. D. (2008). Clinical utility of five dimensional systems for personality diagnosis: A "consumer preference" study. *Journal of Nervous & Mental Disease, 196,* 356–374.

Steel, P., Schmidt, J., & Shultz, J. (2008). Refining the relationship between personality and subjective well-being. *Psychological Bulletin, 134,* 138–161.

Sullivan, M., Katon, W., Russo, J., Dobie, R., & Sakai, C. (1993). A randomized trial of nortriptyline for severe chronic tinnitus: Effects on depression disability and tinnitus symptoms. *Archives of Internal Medicine, 153,* 2251–2259.

Van Houdenhove, B., Egle, U., & Luyten, P. (2005). The role of life stress in fibromyalgia. *Current Rheumatology Reports, 7,* 365–370.

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Thousand Oaks, CA: Sage.

Welch, C. A., Czerwinski, D., Ghimire, B., & Bertsimas, D. (2009). Depression and costs of health care. *Psychosomatics, 50,* 392–401.

Wells, K. B., Golding, J. M., & Burnham, M. A. (1988). Psychiatric disorder in a sample of the general population with and without medical disorder. *American Journal of Psychiatry, 145,* 976–981.

Wells, K. B., Stewart, A., Hays, R. D., Burnham, M. A., Rogers, W., Daniels, M., . . . Ware, J. (1989). The functioning and well-being of depressed patients: Results from the Medical Outcomes Study. *JAMA: The Journal of the American Medical Association*, *262,* 914–919.

Whisman, M. A., & Pinto, A. (1997). Hopelessness depression in depressed inpatient adolescents. *Cognitive Therapy and Research, 21,* 345–358.

World Health Organization (2008). *ICD-10: International statistical classification of diseases and related health problems* (10th Rev. ed.). New York, NY: Author.

Zubenko, G. S., Zubenko, W. N., McPherson, S., Spoor, E., Marin, D. B., Farlow, M. R., . . . Sunderland, T. (2003). A collaborative study of the emergence and clinical features of major depressive syndrome of Alzheimer's disease. *American Journal of Psychiatry, 160,* 857–866.

Zuroff, D. C., & Mongrain, M. (1987). Dependency and self-criticism: Vulnerability factors for depressive affective states. *Journal of Abnormal Psychology, 96,* 14–22.

Zuroff, D. C. (1992). New directions for cognitive models of depression. *Psychological Inquiry, 3,* 274–277.