Chapter 28

Somatization, Hypochondriasis, and Related Conditions

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DESCRIPTION OF THE DISORDERS

General Considerations

Medical patients often present with physical symptoms, including pain, that have no apparent somatic cause. The complaints are frequently accompanied by anxiety, depression, and denial of psychological problems (Dworkin, 1994; Dworkin, Wilson, & Massoth, 1994). In such cases, subclinical or clinical forms of somatoform disorders should be considered. These are conditions in which psychological conflicts and problems have taken the form of a somatic illness (Ford, 1995).

According to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM–IV*) (American Psychiatric Association [APA], 1994), somatoform disorders are subdivided into five specific conditions: body dysmorphic phobia, hypochondriasis, somatization disorder, conversion disorder, and pain disorder. As well, there are two residual diagnostic categories: undifferentiated somatoform disorder and somatoform disorder not otherwise specified. Many other labels, such as hysteria, functional complaints, vegetative dystonia, and Briquet's syndrome, have previously been used to describe similar conditions. Not infrequently, disorders such as asthma, peptic ulcer, esophageal motility disorder, or nonulcer dyspepsia have been erroneously mixed with somatoform disorders; the former have a clearly more distinct psychophysiological basis (Kellner, 1994; Salkovskis, 1996).

Although somatoform disorders have been of interest for a long time, sound scientific approaches to these conditions have been rare (Rief, 1996). Consequently, these diagnostic terms fail to offer straightforward explanations for the pain problems of the afflicted patients or to lead to conspicuous success in pain management. Hence, the first part of this chapter emphasizes the description of those somatoform disorders that focus on pain problems, particularly somatization disorder, pain disorder, conversion disorder, and hypochondriasis. Later sections deal with speculations on the cause of these conditions and on the implications of concepts such as hypervigilance and somatosensory amplification.

Somatization Disorder and Pain Disorder

Somatization disorder and (somatoform) pain disorder have much in common, because pain is a prime component of both. A somatization disorder has to be considered if, after careful investigation, investigators have failed to find an organic basis for pain and other somatic complaints, and yet the patient's worries have increased instead of decreased. A somatization disorder is also likely if the complaints are grossly beyond the level that can be explained by organic pathology. Patients are very often demanding and complain in a dramatic and emotional fashion (Ford, 1995; Kaplan, Sadock, & Grebb, 1994; Rief, 1996).

The process of somatization is considered "the selective perception and focus on the somatic manifestations of depression" (Katon, A. Kleinman, & Rosen, 1982) or "an idiom of distress in which patients with psychosocial and emotional problems articulate their distress primarily through physical symptomatology" (Katon, A. Kleinman, & Rosen, 1982, p. 127). Somatic complaints without known organic etiology are not necessarily due to somatization, because patients suffering from such conditions may acknowledge psychological problems. Somatizers, however, see themselves as markedly and exclusively vulnerable to bodily problems and downplay their emotional and psychological difficulties (Bass & Benjamin, 1993; Dworkin, 1994; Salkovskis, 1996).

Multiple pains are characteristic and mandatory criteria (at least four pain problems in *DSM–IV*; APA, 1994; see Table 28.1) for a somatization disorder (Dworkin, 1994). Among these are headache, abdominal pain, back pain, articular pain, pain in the extremities, chest pain, anal or genital pain, menstrual pain, or pain during intercourse or micturition (Rief, 1996). In a population-based study, Fink (1992) observed that abdominal pain was the most frequent reason for admission of somatizers to nonpsychiatric health facilities. Rief, Hiller, and Fichter (1997) found back pain and headache to be the most frequent somatoform symptoms among psychosomatic patients. Swartz, Blazer, Woodbury, George, and Landerman

TABLE 28.1
Diagnostic Criteria for Somatization Disorder According to *DSM–IV*

A.	Onset before age of 30 years; chronic course; repeated requests for treatment; severe functional disability.
B.	At least eight symptoms, four of them being pain symptoms, two gastrointestinal symptoms, one a sexual symptom, and one a pseudo-neurological symptom.
C.	No or no sufficient medical explanation for the symptoms in criterion B.
D.	No factitious disorder, no malingering.

(1986) described individuals from the general population who suffered from at least three somatization symptoms. Five of their top 10 symptoms were pain related (headache, menstrual pain, chest pain, abdominal pain, and pain in the extremities).

Dworkin, Von Korff, and LeResche (1990) found that the tendency to suffer from somatization symptoms (not including pain) was strongly associated with the number of painful sites. In other words, somatizers tend to have multiple pains or show considerable pain dispersion. Furthermore, they present with strong pain-related disability and interference with daily living (Dworkin et al., 1994).

In addition to pain problems, a diagnosis of a somatization disorder in *DSM–IV* (APA, 1994) requires the presence of psychosexual problems (at least one) and gastrointestinal (at least two) and pseudo-neurological symptoms (at least one), all of which cannot be explained by an organic disorder, as well as a chronic course (episodic with a duration of several years in most cases) and an onset before the third decade. The multiplicity of the complaints helps to distinguish somatization disorder from the other somatoform disorders. However, the high number of complaints has been subject to criticism, because it excludes many individuals, with an apparently similar psychopathology but somewhat fewer symptoms, from this diagnostic category (Dworkin, 1994; Eisendrath, 1995; Ford, 1995; Kaplan et al., 1994; Rief, 1996).

Below the level of a full-blown syndrome, somatization occurs very frequently in a diminished form, but still showing widespread and strongly troubling symptoms. It has become increasingly evident that even such "subclinical" conditions are serious enough to require diagnostic acknowledgment and intervention. Therefore, such categories as undifferentiated somatoform disorder and somatoform disorder not otherwise specified have been introduced into the *DSM–IV* (APA, 1994) classification system. Prevalence estimates for the subsyndromal forms range from 4% to 17 % and can reach 40% (e.g., in neurology), but they are below 1% for a full-blown somatization disorder. Women suffer much more frequently from these conditions than men (with a ratio of about 10 to 1). Whether an increased frequency in some families points to a genetic etiology remains to established

(Bass & Benjamin, 1993; Dworkin, 1994; Dworkin et al., 1994, Eisendrath, 1995; Ford, 1995; Kellner, 1994; Rief, 1996; Rief et al., 1997; Salkovskis, 1996).

The comorbidity with panic disorder is extremely high (up to 100%), and also appreciable for major depression (up to 90%), agoraphobia, substance abuse, and personality disorders (Dworkin et al., 1994; Ford, 1995; Lipowski, 1990). There may be a history of physical and sexual abuse. Consequently, unexplained somatic complaints may be part of a posttraumatic stress disorder (Ford, 1995). The strong overlap with other pathological conditions has led to the conclusion that somatization disorder is a strong aggregation of physical and psychiatric syndromes (Lipowski, 1990).

If pain is the predominant symptom, the diagnosis of a pain disorder is to be preferred (Rief et al., 1997). This condition requires that psychological factors play a central but not necessarily an exclusive role in initiating and maintaining the pain (see Table 28.2). Pathognomic type or location of pain is absent. Pain disorder often develops after an injury or a somatic illness associated with pain. The pain in a pain disorder is real; it is not claimed as part of malingering or a factitious disorder. Women are more likely than men to suffer from such a somatoform pain disorder, although the reasons for this difference are unclear (Comer, 1995; Eisendrath, 1995; Ford, 1995; Kaplan et al., 1994).

This definition of a pain disorder seems to describe a large number of chronic pain patients. Many individuals with specific syndromes suffer not only from pain but also from numerous nonspecific somatic symptoms. The diagnostic concept of a somatoform pain disorder is misused, however, if it is employed in addition to the diagnosis of a specific chronic pain syndrome. If the location of pain, the organ affected by pain, the subjective spatial and temporal characteristics of pain, and the course of pain allow the diagnosis of a specific chronic pain syndrome as described, for example, by the IASP classification system (Merskey & Bogduk, 1994), the understanding and the treatment of the pain are based on more solid ground than if the clinician must rely upon vague speculations about the causes and cures of a somatoform pain disorder.

TABLE 28.2
Diagnostic Criteria for Pain Disorder According to *DSM–IV*

A.	Pain in one or more sites as predominant symptom, severe enough to warrant clinical attention.
B.	Significant distress and impairment of psychosocial and occupational functioning caused by the pain.
C.	Onset, severity, exacerbation, or maintenance of the pain are crucially influenced by psychological factors.
D.	No factitious disorder, no malingering.
E.	The pain cannot accounted for by mood, anxiety, or psychotic disorder and is not a dyspareunia.

The interaction between somatization and chronic pain is complex. This is especially true when the interaction occurs, as it frequently does, within a depressive disorder. There, pain is often thought to be a symptom of depression and depression to be a consequence of chronic pain (Lipowski, 1990). Despite this puzzling situation, somatization seems to be both an important risk factor for and a symptom of chronic pain conditions (Dworkin et al., 1994). The level of somatization appears to determine how limiting and disabling a pain disorder turns out to be in the long run. This, in turn, is a major influence on number of treatment attempts and on costs for the health care system (Dworkin et al., 1994).

Descriptions of somatization disorder and pain disorder make it clear that these conditions are similar to psychosomatic syndromes. Thus, many symptoms of somatoform disorders can be caused by conditions, which might be called psychosomatic in another context. Furthermore, in both groups of disorders there is a clustering of multiple complaints, a substantial number of which can be triggered by stress (Kellner, 1994).

Conversion Syndrome

If there are one or more neurological symptoms that cannot be explained by a known neurological disorder and that can be related to psychological problems (but not to intentional malingering or feigning), the diagnosis of a conversion disorder should be considered (Kaplan et al., 1994; Rief et al., 1997; see Table 28.3). In contrast to the other somatoform disorders, objective functional deficits are required instead of only subjective ones. These are paralysis, blindness, seizures, aphonia, anesthesia, and others. Anesthesia is important in the present context because of its relation to the perception of pain. Nevertheless, almost nothing is known about the clinical consequences of this problem. However, patients with conversion syndrome do also present with pain (Merskey, 1994).

TABLE 28.3
Diagnostic Criteria for Conversion Disorder According to *DSM–IV*

A.	Deficits of voluntary motor and sensory function suggesting a neurological or other general medical condition.
B.	Psychological factors associated with the symptoms (initiation or exacerbation after conflict or other stressor).
C.	No factitious disorder, no malingering.
D.	No sufficient explanation of the symptom by a general medical condition, by substance effects, or as culturally sanctioned behavior or experience.
E.	Significant distress and impairment of psychosocial and occupational functioning caused by the symptoms; symptoms severe enough to warrant medical evaluation.
F.	Not merely pain or sexual dysfunction as the symptom; the symptom is not exclusively part of a somatization disorder or better explained by another mental disorder.

The difficulty in differentiating objective and subjective dysfunctions for diagnosis, plus the still-influential psychodynamic history of this notion, have led some authors to question the validity of the concept of conversion disorder. This situation is made even more troublesome by the fact that neurological signs often become evident at later stages of the disorder (Comer, 1995; Ford, 1995; Salkovskis, 1996). In addition, as Merskey (1994) has noted, chronic pain patients often keep their limbs immobile or use them in an awkward fashion to avoid discomfort, but this misuse or disuse should not be taken as a sign of conversion.

The prevalence of a comprehensive conversion disorder has been estimated to be low and becoming increasingly rare. However, reliable estimates are difficult to obtain because of sizable differences between various health care settings. Furthermore, it is possible that instead of disappearing, conversion disorder has changed its appearance in more medically knowledgeable Western societies. It has been argued that syndromes involving pain, similar to the somatoform pain disorder described earlier, have become more frequent, replacing the classical forms of conversion syndrome. However, at least among female adolescents and young adults with little education, low intelligence, and low socioeconomic status, conversion symptoms are still felt to be common (Comer, 1995; Ford, 1995; Kaplan et al., 1994; Pilowsky, 1994; Salkovskis, 1996).

Interestingly, even pain patients with physical diagnoses can be classified as suffering from a conversion disorder. Fishbain, Goldberg, Meagher, Steele, and Rosomoff (1986) found that 38% of their patients with organic pain fit the diagnosis of a conversion disorder. This diagnosis was based mainly on the finding of "non-anatomical sensory losses." However, the authors questioned the validity of this label because of the lack of knowledge about the characteristics of that term.

Hypochondriasis

Despite being a syndrome with somewhat different characteristics, hypochondriasis is nevertheless thought to be related to the other somatoform disorders. Contrary to the syndromes described earlier, the outstanding feature is a strong fear or conviction of having a serious illness, maintained despite intensive attempts at correction (see Table 28.4). Somatic complaints may be present and disproportionate, but they are not the hallmarks. The multiplicity of somatic problems seen in somatization disorder is not present in hypochondriasis. Rather, hypochondriacal patients focus on one or two symptoms. The precise relationship between somatization and hypochondriasis is still unclear and there may be some overlap between the two conditions. The situation becomes even more complicated by the fact, discussed later, that the term hypochondriasis is used not only to

TABLE 28.4
Diagnostic Criteria for Hypochondriasis According to *DSM–IV*

A.	Preoccupation with fear or conviction of having a serious disease because of the misinterpretation of bodily symptoms.
B.	Preoccupation despite appropriate medical evaluation and reassurance.
C.	Criterion A is not delusional in nature and restricted to a circumscribed concern about appearance (as in body dysmorphic disorder).
D.	Significant distress and impairment of psychosocial and occupational functioning caused by the preoccupation.
E.	Disturbance more than 6 months.
F.	The preoccupation is not better accounted for by generalized anxiety disorder, obsessive-compulsive disorder, panic disorder, a major depressive episode, separation anxiety, or another somatoform disorder.

describe a syndrome but also to name behaviors and attitudes as well as cognitive and affective styles (Barsky & Klerman, 1983; Dworkin et al., 1994; Rief et al., 1997; Salkovskis, 1996).

Pain can be present if a hypochondriacal individual amplifies normal somatic sensations, which is often the case. However, pain is not a necessary criterion for hypochondriasis. The pain in hypochondriasis—like all of the other complaints—is often vague, variable, and generalized. Most frequent locations are the head, neck, chest, and abdomen (Barsky & Klerman, 1983; Dworkin et al., 1994; Eisendrath, 1995).

There has been considerable discussion as to whether hypochondriasis is a discrete and cohesive psychopathological condition in its own right, whether it is mainly secondary to other psychological problems, particularly to depression, or whether it is a cluster of attitudes and behaviors, which are not closely related to any disorders. Primary hypochondriasis is said to exist if hypochondriacal behavior exceeds a critical duration (6 months in *DSM–IV*; APA, 1994) and is not clearly tied to other psychiatric problems. Transient and symptomatic forms exist in patients with major depression, panic disorder, and schizophrenia (Barsky & Klerman, 1983; Ford, 1995; Salkovskis, 1996).

There are several components of hypochondriasis. On an affective level, it appears as body-related phobia and an obsessive preoccupation with one's own body; on a cognitive level, it presents as a rigid conviction that one is ill or is threatened by illness, with sometimes almost delusional character. The cognitive distortion often includes the unrealistic perspective that true health does not permit any bodily discomfort (Barsky & Klerman, 1983; Rief, 1996; Salkovskis, 1996).

Because of their somatic bias, hypochondriacal individuals rarely visit psychiatrists and psychologists. Rather, they look for help from "somatic experts," that is, family physicians or pain specialists, although they often fail to trust medical authorities. In primary-care settings, 3% to 14% of

patients may be classified as hypochondriacs and may have extensive histories of previous medical care. In contrast to somatization disorder, men are affected equally often as women. Hypochondriasis starts later in life than somatization disorder (Barsky & Klerman, 1983; Comer, 1995; Eisendrath, 1995; Ford, 1995; Salkovskis, 1996).

CAUSAL FACTORS

Exaggerated Stress Responses

Although, in general, there seems to be no straightforward causal relationship between stress and chronic pain, some findings suggest that exaggerated stress-related responses in the striated muscles form the basis of regional pain in some pain patients (Flor & Birbaumer, 1994; Merskey, 1994). Correspondingly, some authors assume that a more generalized pattern of hyperreactivity to stress affecting various organ systems might be seen in psychosomatic disorders or somatoform disorders. The hypothesized stress responses in both types of disorders may include endocrine changes, striated muscle tension, smooth muscle activity, and hyperventilation (Kellner, 1994).

Augmentation, Amplification, and Hypervigilance

When awareness is reached during processing of bodily signals, perceptual mechanisms become relevant. It is at this stage when amplification, augmentation, and hypervigilance, which are perceptual distortions of potential relevance for somatoform disorders, can occur (Dworkin, 1994). Barsky and Klerman (1983) assumed that hypochondriacal patients experience normal bodily sensation as painful because they augment and amplify bodily sensations, are less stoical, and have low thresholds for discomfort. Such a nonspecific perceptual enhancement of distressing events was labeled by Barsky (1992) as *somatosensory amplification.* However, Spinhoven and van der Does (1997) provided some evidence that somatosensory amplification may not explain the process of somatization particularly well. An even more important causal factor for somatization in their study appeared to be anxiety.

Because there is no direct evidence, from longitudinal studies, that somatosensory amplification, augmentation, or hypervigilance are indeed risk factors for somatoform disorders (Rief, 1996), there are many unanswered questions about the influence of perceptual mechanisms in their genesis. Patients with hypochondriasis and disease phobia have exhibited lowered pain thresholds (Kellner, 1994; Merskey & Evans, 1975), although

contrary findings also exist (Lautenbacher, Pauli, Zaudig, & Birbaumer, 1998). Moreover, a lowered pain threshold might be the result or the cause of a somatoform disorder.

Barsky and Klerman (1983) speculated that besides perceptual distortions, hypochondriacal patients misinterpret normal bodily sensations as signals for serious diseases. Such a cognitive distortion may enhance each small discomfort to life-threatening proportions. This phenomenon is also related to catastrophizing, an exaggerated style of cognitive and emotional appraisal (Dworkin, 1994).

Abnormal Illness Behavior

Abnormal illness behavior represents an inappropriate and maladaptive mode of perceiving, evaluating, and acting despite appropriate management of a disease by health care providers and relatives. Illness behavior depends strongly on contingencies determined by the "sick role" in a society. If, for example, it seems more favorable for an individual to present with physical problems instead of depression because the former, but not the latter, leads to sympathy, encouragement, attention, support, and compensation, a somatic masking of psychological problems or, more simply, somatization may occur (Barsky & Klerman, 1983). More attractive contingencies for physical than for psychological complaints appear to be part of our highly "medicalized" Western society (Dworkin et al., 1994). Furthermore, abnormal illness behavior may include the conviction that true health excludes any somatic discomfort. Consequently, the patient is likely to take each sign of discomfort as symptomatic of a disease (Rief, 1996).

Commonly, patients are given reinforcement (e.g., avoidance of responsibilities and increased caring responses from those close to them) for their somatization. Even physicians tend to direct their attention to their patients' somatic symptoms instead of their psychological ones (Bass & Benjamin, 1993; Ford, 1995; Salkovskis, 1996). It is not surprising, then, that increased somatic focus occurs and that patients become preoccupied with monitoring and reporting their physical complaints.

Some authors believe that the concept of abnormal illness behavior provides a theoretical framework for all somatoform disorders (Pilowsky, 1994). Eisendrath (1995) argued that the type of somatoform disorder attributable to abnormal illness behavior depends mainly on whether the patient's motivation is conscious or not.

A result obtained by Dworkin et al. (1994) is of special interest for the management of chronic pain. The authors reported that whether chronic pain patients attributed the cause of their pain to physical or behavioral factors was dependent on their individual level of somatization as measured by the SCL–90. Hence, somatization is associated with a belief system that

blames organic factors for chronic pain. Consequently, it is very likely that somatizers will also look for a "somatic expert" to cure their pain problem.

Phobic and Obsessive-Compulsive Response Pattern

Hypochondriasis shares several cognitive features with phobic and obsessive-compulsive disorders. The similarity with obsessive-compulsive disorders involves mainly the narrowing and automatization of thinking that focuses on the threat of a serious illness, that tends to catastrophize, and that withstands all attempts of correction. Not surprisingly, some hypochondriacal patients meet the criteria for obsessive-compulsive disorders. The emotional similarity with anxiety disorder is also obvious because of the constant worry and concern about health status. Therefore, some authors prefer to group hypochondriasis with anxiety disorders and treat it accordingly, often with considerable success (Dworkin et al., 1994; Ford, 1995; Merskey, 1994).

TREATMENT CONSIDERATIONS

Most patients with somatoform disorders are seen and treated first by family physicians (Rief et al., 1997). When the somatoform disorder is finally diagnosed, a therapeutic nihilism often results, because even psychotherapists consider somatizers as extremely difficult patients. Somatoform disorders have been thought to be extremely resistant to change, a view that needs correction, at least in the case of hypochondriasis, which can be treated with some success (Rief, 1996; Salkovskis, 1996).

Physicians' usual emphasis on seeking evidence of a physical disorder is understandable, but it is often detrimental to patients suffering from somatoform disorders. Contact with many medical personnel merely increases the patient's opportunity to demonstrate various somatic complaints. The major aim of therapy should be a gradual replacement of a patient's physical perspective by a psychosocial one, using regularly timed appointments rather than ones scheduled in response to somatic complaints. This approach may include training in various psychological coping strategies and in expressing emotions in a nonsomatic fashion. Such psychological treatments are most likely to be effective if they can decrease the patient's catastrophizing and increase her or his self-efficacy. A major reason for dropping out of therapy and for paradoxical exacerbations of symptoms is a management style that pushes a patient too hard and too early toward a psychological perspective and that consequently challenges the "sick role" of the patient without sufficient preparation (Barsky &

Klerman, 1983; Bass & Benjamin, 1993; Dworkin et al., 1994; Kaplan et al., 1994; Pilowsky, 1994, 1995; Rief, 1996).

Many patients with somatoform disorders have unrealistic ideas: that a healthy person does not experience any kind of bodily discomfort and that bodily discomfort can be caused only by a somatic disease and not by psychological problems or stress. The offer of alternative explanations can be very beneficial and can help to prepare patients for "coping not curing." However, somatic beliefs are deeply rooted and often withstand vigorous attempts at modification. Nevertheless, treatment of somatizers can hardly be successful without a change in cognitions, because the patient's conceptions of his or her illness determines what type of help is asked for, from whom, when, and where. A purely physical perspective and an overly aggressive psychological one can each prove ineffective. The former reinforces the patient's inappropriate somatic convictions, whereas the latter shatters the patient's system of illness beliefs. Strong dysfunctional illness beliefs and too many contacts with health care providers are both predictive of chronic courses of an illness (Bass & Benjamin, 1993; Dworkin et al.; 1994; Pilowsky, 1995; Rief et al., 1997).

Despite the pharmacological emphasis of contemporary psychiatry, it is still doubtful whether there are indications for using drugs to treat patients with somatoform disorders. Systematic treatment attempts have been rare. The use of antidepressants has the best empirical basis (Pilowsky, 1994; Rief et al., 1997). It is noteworthy that the so-called "psychogenic" pain of somatoform disorders does not respond any better than do "somatic" pains to antidepressive medication (Volz, Stieglietz, Menges, & Möller, 1994).

SOMATIZATION, HYPOCHONDRIASIS, AND HYPERVIGILANCE IN OTHER PAIN DISORDERS

Questions about somatization, hypochondriasis, and related matters arise in attempts to understand pain syndromes whose symptoms are clear but whose underlying processes remain murky. Consider, for example, fibromyalgia. This disorder, characterized by widespread bodily pain, multiple tender points, fatigue, sleep disturbances, stiffness, and attentional difficulties, has no evident pathophysiological basis.

Numerous investigators have reported that fibromyalgia patients score high on scales of depression, anxiety, and hypochondriasis (e.g., Ahles, Yunus, & Riley, 1984; Goldenberg, 1987; Payne et al., 1982; Wolfe, Cathey, & Kleinheksel, 1984; Yunus, Ahles, Aldag, & Masi, 1991). Interpretations of such findings, however, must be made with caution. The Minnesota Multiphasic Personality Inventory (MMPI) has often been used as the personality questionnaire in these studies. The MMPI has been deemed

inappropriate for use with pain patients by some specialists (e.g., Merskey et al., 1985; Smythe, 1984) because individuals suffering from painful disorders will almost inevitably affirm health-related items, such as "I feel weak all over much of the time," or answer in the negative to "I am about as able to work as I ever was," both of which raise their scores on psychopathological scales. Patients suffering from disorders that have an identifiable organic pathology will also, not surprisingly, answer in the same way and, consequently, show scale elevations.

Scudds, Rollman, Harth, and McCain (1987) examined personality measures for fibromyalgia and arthritis patients, as well as normal controls, on the Basic Personality Inventory, an instrument designed with strong psychometric criteria. They eliminated items that might be included among the symptomatic features of the two disorders. Fibromyalgia patients scored markedly higher than controls on the hypochondriasis scale, but those scores were only slightly higher than those obtained for rheumatoid arthritis sufferers. It is inappropriate to conclude from personality tests such as these, which fail to appropriately assess all of the necessary diagnostic criteria, that fibromyalgia is a hypochondriacal disorder.

Fibromyalgia patients were also elevated on measures of depression and anxiety when compared to normals, but this is not surprising given that they are suffering from a disorder that is painful, troubling, and difficult for the patients, their families, and their physicians to understand. Still, it does not rule out a common pathopsychophysiology of affective disorders, anxiety disorder, and fibromyalgia. Related findings, also using the Basic Personality Inventory, were obtained by Schnurr, Brooke, and Rollman (1990). They compared individuals with temporomandibular pain and dysfunction syndrome (TMPDS), who have intense pain in the masticatory muscles, with patients who had painful conditions arising from injury and with a group of pain-free controls. TMPDS patients also suffer from a disorder whose pathophysiology is unknown and that has been linked to stress. As well, they, too, have been reported to show abnormal elevations in a variety of personality characteristics (e.g., Gale, 1978; Merskey et al., 1987). Schnurr et al. found that the hypochondriasis scores of TMPDS patients were strikingly elevated compared to pain-free subjects but were essentially identical to those of the patients with pain due to injury. Again, one cannot conclude that hypochondriacal traits are the root of their disorder. However, they may well serve as a risk factor for the transition from acute to chronic pain.

At least three contrasting possibilities can be suggested by findings such as these. First, traditional psychological scales that assess psychological distress and bodily complaints, often developed and normed for psychiatric patients, are not applicable to the study of medical patients, especially for those suffering from serious pain. Second, hypochondriacal traits may be

seen in syndromes with pain that has an organic basis as well as with pain of unknown origin. Third, elevated levels of hypochondriasis, as well as anxiety and depression, might be a consequence of pain and not necessarily its cause. Similar considerations apply when looking at the relation between somatization and various pain syndromes (McKinney, Londeen, Turner, & Levitt, 1990). More investigations, using instruments for assessing psychological distress and bodily complaints that are normed on pain patients and that look also at individuals suffering from chronic illnesses that are not marked by pain, are needed to help distinguish among these alternatives.

Clues about factors that contribute to disorders such as fibromyalgia and TMPDS have come from a series of perceptual studies. It is a given that fibromyalgia patients will have exquisite sensitivity at 11 or more of the 18 tender points specified in the American College of Rheumatology criteria for the disorder (Wolfe et al., 1990). Scudds et al. (1987), among others, found that they also show much lower pain threshold and pain tolerance to pressure applied at control sites. Whatever differentiates the pain reaction of fibromyalgia patients and controls is not limited to specific loci. Nor is the difference to be found only for pain involving the muscles. Lautenbacher, Rollman, and McCain (1994) showed that fibromyalgia patients had significantly lower pain thresholds when the noxious stimulus was heat (delivered to the arm and the trapezius by means of a Peltier thermode system) or a train of electrical pulses (presented to a pressure-sensitive point on the trapezius through a pair of silver electrodes). At the least, fibromyalgia patients generally show a considerable degree of responsiveness to noxious stimuli, whatever the origin.

McDermid, Rollman, and McCain (1996) demonstrated that this hypersensitivity extends beyond the traditional pain domain. Fibromyalgia patients, when presented with 3-second bursts of white noise through a headset, had a tolerance level of about 66 dB, an intensity that would sound only moderately loud to control subjects (whose noise tolerance was 100 dB). Rheumatoid arthritis patients also showed a diminished noise tolerance, but to a lesser extent (76 dB). Fibromyalgia patients stood out from both of the other two groups in their scores on the Noise Sensitivity Scale (Weinstein, 1978), a questionnaire designed to elicit information about the disturbance and interference elicited by environmental sounds.

More recent studies have employed psychophysical methods to look at possible deficiencies of pain modulation in fibromyalgia patients. Using the diffuse noxious inhibitory control paradigm, in which a tonic noxious stimulus (such as a tourniquet or a long-lasting intense heat stimulus) at one site on the body, such as the arm, suppresses the pain threshold or rated painfulness of a noxious phasic stimulus (such as electrical shock) applied to a distant body part, both Kosek and Hansson (1997) and Lautenbacher and Rollman (1997) found less suppression of pain in patients

than in controls. It remains to be determined whether this reflects a dysfunction of physiological inhibition or an attentional disorder in which the fibromyalgia sufferers concentrate on all noxious inputs whereas pain-free individuals channel their attentional capacity to the more lasting and noxious input.

The hyper-responsiveness and the lack of inhibitory capacities in fibromyalgia patients may explain why it is associated with such a wide range of bodily symptoms and complaints including headache, irritable bowel, dysmenorrhea, light sensitivity, temporomandibular dysfunction, and paresthesias (Waylonis & Heck, 1992; Yunus et al., 1991). Smythe (1986), in observing their response to both internal and external stimuli, has described fibromyalgia patients as suffering from the "irritable everything syndrome" (p. 2). McDermid et al. (1996) found confirmatory evidence through the Pennebaker Inventory for Limbic Languidness (Pennebaker, 1982), a checklist that assesses the frequency of occurrence of 54 common physical symptoms and sensations. Scores for the fibromyalgia patients were considerably greater than for the controls. The arthritis patient scores were also elevated, but to a much smaller degree.

A host of factors may contribute to the hyper-responsiveness seen in chronic, multisymptom pain disorders such as fibromyalgia and TMPDS. Recent studies on fibromyalgia have tended to emphasize biological rather than psychosocial mechanisms, concentrating on muscles, substance P, serotonin, neuroendocrine agents, central processing of pain as revealed by positron emission tomography scans, and others. Alterations have been seen in many of these, but McDermid et al. (1996) cautioned, "while some of these factors may be of primary importance in the pathophysiology of the disease, most must be secondary influences and thus should be viewed as correlates rather than causes of the disorder" (p. 140). Hence, there is no reason to stop the quest for causal factors or to exclude explanations on a psychological level.

Such perceptual studies on pain thresholds or pain inhibition in pain patients provide insights into some of the mechanisms that underlie their disorders. Rollman and Lautenbacher (1993), examining evidence from a number of investigations, suggested that fibromyalgia involves a generalized pattern of hyper-responsiveness to internal and external discomfort that could be characterized under the heading of *hypervigilance*. They noted that hypervigilance is a more focused concept than hypochondriasis. As Barsky, Wyshak, and Klerman (1990) observed, "hypochondriacal symptoms can be thought of as the product of psychodynamic forces, interpersonal miscommunication, formative learning experiences, or an amplifying cognitive and perceptual style" (p. 323). Most notions of hypochondriasis imply a psychopathological process. Hypervigilance emphasizes a perceptual and cognitive one.

Barsky, Wyshak, and Klerman (1986) found that disease fear, disease conviction, bodily preoccupation, and somatic symptoms, all of which are compatible with the *DSM–IV* (APA, 1994) diagnosis of hypochondriasis, also cluster together in many medical patients. Consequently, it is important to distinguish individuals who have hypochondriacal attitudes and beliefs that are distinct and severe enough for a diagnosis of hypochondriasis from those who do not suffer from a psychiatric disorder but who do demonstrate heightened awareness and sensitivity to bodily events. The latter might be characterized by some as somatosensory amplification (Barsky & Wyshak, 1990) and by others as hypervigilance. Somatosensory amplification is measured by means of questionnaires whose items are similar to those that are used to assess hypochondriasis. Hypervigilance is typically determined through examination of perceptual performance. It remains to be established whether the two tap into common behavioral and cognitive elements.

We prefer to use the term hypervigilance when dealing with disorders such as fibromyalgia and TMPDS. This is partly for historical reasons, because Naliboff, Cohen, Schandler, and Heinrich (1981) contrasted Chapman's (1978) model of hypervigilance with Rollman's (1979) model of adaptation level in examining the threshold for noxious input in pain patients. The first suggests that pain patients have diminished pain threshold and tolerance levels, hyper-responding to both internal and external stimuli. In contrast. the adaptation level model suggests that pain patients compare external stimuli to their endogenous pain, rating the stimuli as less intense than they would if they did not have a high level of pain as an internal anchor or comparison point. Both models have received support (Naliboff & Cohen, 1989; Rollman, 1992), but for different populations of pain patients.

Kellner (1994) suggested that many of the phenomena of disorders such as fibromyalgia, chronic fatigue, irritable bowel, nonulcer dyspepsia, urethral syndrome, and others are caused by clustering of psychosomatic syndromes and a low sensation threshold. Naliboff et al. (1997) found that patients with irritable bowel syndrome showed Ahypervigilance for visceral stimuli, manifested as lowered response criteria for using the descriptor 'discomfort' " (p. 505).

Hypervigilance, as a descriptive term, has tended to focus attention on perceptual performance. The differences in pain thresholds, tolerance values, or ratings between certain groups of pain patients and other pain patients or pain-free controls, have often been substantial. However, it would be premature to suggest that this simply reflects a difference in transduction or transmission of noxious signals. Hypervigilance may be associated with a series of processes, beyond the perceptual ones, that have potentially pathogenic relevance (Rollman, 1998).

Those who are hypervigilant may be more likely to (a) show greater sensitivity to stimuli (although this may turn out not to be an integral characteristic), (b) monitor internal and external events, (c) attribute bodily signs to physiological causes rather than to environmental or psychological ones, (d) demonstrate a cognitive pattern of catastrophizing in attempting to cope with their situation, and (e) react to negative events and cognitions with one or more of a number of bodily reactions such as localized or widespread muscle tension, altered gastric motility, or marked autonomic or cardiovascular function.

In a recent study, McDermid and Rollman (1996) were able to discriminate among fibromyalgia patients, TMPDS patients, arthritis patients, and pain-free controls on measures of monitoring, symptom attribution, anxiety, and coping response. In a discriminant function analysis, fibromyalgia patients stood out from the other pain groups on a dimension characterized as monitoring and both the fibromyalgia and TMPDS groups had significantly higher scores than the arthritis patients on a second dimension related to symptom anxiety.

Issues concerning the interplay of biological and psychosocial factors in pain remain both fascinating and controversial. To stress the importance of one is not to negate the other; nor should it be seen, by patient or physician, as either favorable or pejorative to emphasize a particular predisposing or contributing factor. Monitoring of somatic signals, interpretation of bodily symptoms, evaluation of symptoms, and the marshaling of coping responses (Dworkin, 1990; Mechanic, 1985) occur to varying degrees in all individuals. Moreover, much as one might wish to see concepts such as hypochondriasis, somatization, illness behavior, and hypervigilance as independent constructs, the evidence indicates that they are closely related to one another (Spekens, Spinhoven, Sloekers, Bolk, & van Hemert, 1996).

A host of questions remain unanswered concerning the underpinnings of various painful disorders: the leap from correlation to causation, the role of various psychological and pharmacological approaches in dealing with psychological manifestations of pain, the reasons why many of the pain disorders marked by hypervigilant behavior are found strikingly more often in women than in men (Berkley, 1997; Rollman, 1995; Unruh, 1996), and the distinction between behavioral and physiological manifestations of stress (Jones, Rollman, & Brooke, 1997). Longitudinal studies are badly needed to determine the predictors of various psychologically linked pain disorders, the determinants of good and bad outcomes relating to their pain problem, and the incidence of comorbid psychiatric disorders (Walker et al., 1997). We need a better understanding of why some patients with disorders such as fibromyalgia (Aaron et al., 1996; Wolfe, Ross, Anderson, Russell, & Hebert, 1995) and irritable bowel syndrome (Drossman et al., 1988) meet all criteria for the disorder but feel no need to seek medical

treatment. Finally, we need to identify the mechanisms that determine why a somatic presentation occurs at a particular system or site (Robbins, Kirmayer, & Hemami, 1997) and why, when there is an overlap of symptoms (e.g., Hudson, Goldenberg, Pope, Keck, & Schlesinger, 1992), a patient often chooses to report one to a physician (say, pain rather than fatigue), thereby possibly affecting both the diagnosis and the treatment that follow.

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Biopsychosocial Perspectives

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