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# LAW AND THE PROBLEM OF PAIN

*by Michael Finch\**

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## I. INTRODUCTION

Pain used to be a simple issue. It was caused by physical injury or disease and the sufferer had to rest and take opium. That was about two hundred years ago.<sup>1</sup>

Pain is a universal experience. Its unpleasantness is commonly thought to convey a message of discrete injury or disease. This view, still prevalent in law and clinical medicine, conceives of pain as the central nervous system's immediate response to acute bodily distress. Such acute pain has identifiable origins, occurs through well-known biological mechanisms, and is usually treatable.<sup>2</sup>

Yet for an appreciable segment of the world's population, pain is a chronic and pervasive condition having no clear medical meaning. This

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1. Harold Merskey, *Pain Disorder, Hysteria or Somatization?*, 9 PAIN RES. MGMT. 67 (2004).

2. MAYO CLINIC ON CHRONIC PAIN 9 (David W. Swanson ed., 2d ed. 2002).

pain has no discernible relationship to injury or disease, and many critics wonder if it has an organic basis at all.<sup>3</sup> More than ten percent of the population—predominately women—suffer from chronic pain,<sup>4</sup> and it is often incurable. The resulting personal and social costs are great. Approximately \$85 billion to \$100 billion are spent annually for the diagnosis and management of chronic pain.<sup>5</sup> Almost half of the Social Security disability disputes pending in federal court involve claims of chronic pain.<sup>6</sup> Damages for “pain and suffering” are often the largest component of personal injury awards<sup>7</sup> and the perennial focus of tort reform.<sup>8</sup>

Despite the prevalence of chronic pain, one question will not go away: Is the pain medically “real”?<sup>9</sup> Pain is a quintessentially subjective symptom and no practicable tests exist to verify its presence. Under the current medical regime of disease realism, most chronic pain is fragilely classified as a “syndrome” rather than a disease<sup>10</sup> and grouped with a

3. See *infra* Part III.

4. See Elaine F. Harkness et al., *Mechanical Injury and Psychosocial Factors in the Work Place Predict the Onset of Widespread Body Pain*, 50 ARTHRITIS & RHEUMATISM 1655, 1662 (2004) (reporting chronic widespread pain in the general population to be 10%–13%); Don L. Goldenberg, *Fibromyalgia Syndrome A Decade Later*, 159 ARCHIVES INTERNAL MED. 777 (1999) (estimating that 10%–12% of the general population has chronic widespread pain). Approximately one-half of the population will develop chronic pain at some point in their lifetime. See MAYO CLINIC ON CHRONIC PAIN, *supra* note 2, at 12.

5. See Michael I. Weintraub, *Chronic Pain in Litigation*, 13 NEUROLOGIC CLINICS 341 (1995); DANIEL J. WALLACE & JANICE BROCK WALLACE, ALL ABOUT FIBROMYALGIA x (2002). Wallace and Wallace also note that some “50 million Americans are partially disabled due to chronic pain.” *Id.* at xi.

6. See Aimee E. Bierman, *The Medico-Legal Enigma of Fibromyalgia: Social Security Disability Determinations and Subjective Complaints of Pain*, 44 WAYNE L. REV. 259, 273 (1998).

7. See, e.g., Weintraub, *supra* note 5, at 341 (discussing civil litigation damages and noting that 80% of civil awards result from “subjective and intangible pain and suffering, loss of consortium, and loss of life’s pleasures”); Edward J. McCaffrey et al., *Framing the Jury: Cognitive Perspectives on Pain and Suffering Awards*, 81 VA. L. REV. 1341, 1347 (1995) (estimating that as much as one-half of the total damages paid out in several forms of product liability and malpractice litigation are for pain and suffering).

8. See, e.g., Steve Lohr, *Bush’s Next Target: Malpractice Lawyers*, N.Y. TIMES, Feb. 27, 2005, at 3.1 (discussing the Bush administration’s proposed cap of \$250,000 on noneconomic damages in medical malpractice suits); see generally Paul V. Niemeyer, *Awards for Pain and Suffering: The Irrational Centerpiece of Our Tort System*, 90 VA. L. REV. 1401 (2004); Steven P. Croley & Jon D. Hanson, *The Nonpecuniary Costs of Accidents: Pain-and-Suffering Damages in Tort Law*, 108 HARV. L. REV. 1785 (1995).

9. See, e.g., Jane Brody, *Fibromyalgia: Real Illness, Real Answers*, N.Y. TIMES, Aug. 1, 2000, at 8.

10. Syndromes consist of groupings of symptoms and signs whose causes have yet to be determined. See Mary Sue Henifin, Howard M. Kipen & Susan R. Poulter, *Reference Guide on Medical Testimony*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 463 (2d ed. 2000). Syndromes do not usually achieve the status of disease until their causes are determined. See, e.g., Robert Kendell & Assen Jablensky, *Distinguishing Between the Validity and Utility of Psychiatric Diagnoses*, 160 AM. J. PSYCHIATRY 4, 6 (2003) (explaining that clinical signs and symptoms do not constitute the disease and

host of controversial illnesses whose legitimacy is still questioned.<sup>11</sup>

Chronic pain rouses many of the suspicions traditionally harbored against subjective female illnesses. A variety of social and medical critics view chronic pain as a post-modern illness sharing a lineage with nineteenth-century pseudo-maladies like hysteria and neurasthenia.<sup>12</sup> These illnesses, they contend, originate in vulnerable human psyches and achieve legitimacy through the collusion of physicians, lawyers, and cultural support groups. Central to these suspicions is the seemingly unshakable belief that chronic pain is a psychosomatic disorder, with the implication that the sufferer's pain is not medically "real."<sup>13</sup>

This Article examines the problem of enigmatic chronic pain. The principal contention is that psychosomatic explanations of chronic pain are flawed, despite their inordinate popularity with social critics, expert witnesses, and a growing number of judges. There is little empirical evidence to support the view that chronic pain is principally a symptom of psychic conflict and distress, and much evidence to rebut it. Most compelling is the emerging biological evidence demonstrating that much chronic pain results from pathology in the central nervous system, rather than discrete injury or illness like that which produces acute pain.

The problem of chronic pain is examined by focusing on the most prevalent chronic pain syndrome found in litigation today, fibromyalgia. Fibromyalgia is nothing less than the widespread pain of rheumatism that has distressed humanity throughout recorded history. This pain syndrome has been described as one of the "most controversial conditions in the history of medicine,"<sup>14</sup> and it is at the center of the debate about the nature of chronic pain.<sup>15</sup> Part II begins by examining how chronic pain often straddles competing diagnoses in medicine and psychiatry, thus lending credibility to both organic and psychogenic

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that under the disease-realism model that has prevailed since the late nineteenth century, disease status is not achieved until "causal mechanisms are clearly identified").

11. These include chronic fatigue syndrome, multiple chemical sensitivity syndrome, and mental health problems like post-traumatic stress disorder. See generally KATRINA BERNE, CHRONIC FATIGUE SYNDROME, FIBROMYALGIA AND OTHER INVISIBLE ILLNESSES (2002); WALLACE & WALLACE, *supra* note 5, at 94–100.

12. See *infra* text accompanying notes 54–69.

13. See, e.g., Ruby Afram, *New Diagnoses and the ADA: A Case Study of Fibromyalgia and Multiple Chemical Sensitivity*, 4 YALE J. HEALTH POL'Y, L. & ETHICS 85, 94 (2004) ("For most of its known history, those who studied the condition believed its genesis to be psychosomatic.").

14. See I. Jon Russell, *Muscle and Fibromyalgia*, in THE TEXTBOOK OF PAIN 3 (Ronald Melzack ed., forthcoming).

15. Fibromyalgia alone exacts between \$12 billion and \$15 billion in health care costs each year. See Anil Kumar Jain et al., *Fibromyalgia Syndrome: Canadian Clinical Working Case Definition, Diagnostic and Treatment Protocols—A Consensus Document*, 11 J. MUSCULOSKELETAL PAIN 3, 5 (2003). As discussed later, fibromyalgia is by far the most prevalent chronic pain syndrome presented in contemporary litigation. See *infra* text accompanying notes 19–22.

concepts of pain. Part III then examines how the concept of psychosomatic pain or “somatization” has attained prominence in cultural history and critical medical literature. Although the concept of somatization does not intrinsically disparage chronic pain, it has acquired a distinct secondary meaning—that pain symptoms are exaggerated or feigned and, ultimately, within the control of the sufferer. Segments of the insurance and health care industries have now appropriated the theme of somatization to minimize the chronic pain experience and control health care costs.

Part IV next assesses empirical research concerning the role of psychological distress in the generation of chronic pain. As demonstrated, there is widespread acceptance that psychological factors play a role in the pain process. But research does not support the much broader claim that chronic pain syndromes are largely the consequence of psychological distress. The neo-Freudian concept of somatization has been greatly oversold and is driven more by theory than scientific evidence.

Finally, Part V examines the developing biological model of chronic pain. A compelling body of evidence now demonstrates that the acute-pain model still dominant in medical practice and the law is incomplete. There is growing recognition that the central nervous system is far more plastic than previously imagined. In an appreciable segment of the population the central mechanisms governing pain can be fundamentally altered, resulting in the persistent production of pain with no discernible relationship to bodily injury or illness. These central mechanisms, rather than mental disturbances, dispel the enigma of much chronic pain.<sup>16</sup>

Part VI concludes that, somewhat remarkably, while a “fundamental reassessment of chronic pain”<sup>17</sup> has occurred in the scientific literature, this reassessment has gone unnoticed in the law. Until courts rethink the prevailing model of pain and its mechanisms, chronic pain will remain a stubborn mystery, and its sufferers will remain under suspicion as latter-day hysterics and malingerers.

## II. COMPETING CONSTRUCTS OF CHRONIC PAIN

In the past quarter century, more than two thousand cases have considered the medical phenomenon known by courts as “chronic pain syndrome” or “fibromyalgia.”<sup>18</sup> Although the two terms are often used

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16. See *infra* Part IV.

17. See Jean Marx, *Prolonging the Agony*, 305 SCIENCE 326 (July 16, 2004).

18. A Westlaw search using the terms “fibromyalgia” or “chronic pain syndrome” reveals more than two thousand cases addressing these illnesses since 1978.

interchangeably, this usage is imprecise.<sup>19</sup> Researchers have identified more than three dozen syndromes involving generalized chronic pain.<sup>20</sup> The equation of fibromyalgia with enigmatic chronic pain reflects the fact that fibromyalgia is by far the most common diagnosis presented in disability and tort litigation.<sup>21</sup>

The concept of fibromyalgia is simple and broad. It refers to widespread bodily pain—an “aching all over”—that persists over an extended period of time. For centuries this pain was popularly known as “rheumatism.”<sup>22</sup> Fibromyalgia is predominately a women’s health problem, afflicting women seven to ten times more often than men.<sup>23</sup> This is consistent with the disparate impact of most forms of pain, which women suffer with greater frequency, severity, and duration than men.<sup>24</sup> Fibromyalgia is also associated with other unexplained medical and mental illnesses that disproportionately afflict women, including chronic fatigue, migraine headaches, anxiety, depression, and somatization disorder.<sup>25</sup>

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19. This Article will follow legal convention and use the terms “chronic pain syndrome,” “fibromyalgia,” and sometimes simply “chronic pain” interchangeably. For the medical reader, the author stresses that most of the medical research and findings discussed pertain specifically to the medical construct known as fibromyalgia or, less commonly, “generalized myofascial pain syndrome.” See INT’L ASS’N FOR THE STUDY OF PAIN, CLASSIFICATION OF CHRONIC PAIN xiii (Harold Merskey & Nikolai Bogduk eds., 2d ed. 1994). Although the term “chronic pain syndrome” has acquired a pejorative connotation for some, none is intended in this Article. See *id.* (noting that “the term chronic pain syndrome is often, unfortunately, used pejoratively”).

20. See *id.* at 6–7. Even though this Article’s discussion of chronic pain syndrome or fibromyalgia may be relevant to other pain syndromes, it is not strictly speaking addressed to them.

21. Fibromyalgia is the most common cause of widespread bodily pain. See Robert M. Bennett, *Fibromyalgia: The Commonest Cause of Widespread Pain*, 21 FRONTIERS 269 (1995).

22. See D.J. Clauw, *The Pathogenesis of Chronic Pain and Fatigue Syndromes, with Special Reference to Fibromyalgia*, 44 MED. HYPOTHESES 369 (1995).

23. An estimated 80%-90% of fibromyalgia sufferers are women. See, e.g., WALLACE & WALLACE, *supra* note 5, at 15; Lawrence A. Bradley & Graciela S. Alarcón, *Fibromyalgia*, in 2 ARTHRITIS & ALLIED CONDITIONS 1811 (William J. Koopman ed., 14th ed. 2001). Roughly 4% of women in the population suffer from fibromyalgia, and its prevalence increases substantially in women over 60. Estimates of fibromyalgia prevalence are generally thought to be low, as doctors often mistakenly diagnose fibromyalgia as other medical or mental illness. See Jain et al., *supra* note 15, at 5 (“The prevalence of FMS is likely underestimated as many cases are attributed to other systemic disorders or misdiagnosed as psychiatric in origin.”); Frederick Wolfe et al., *The Prevalence and Characteristics of Fibromyalgia in the General Population*, 38 ARTHRITIS & RHEUMATISM 19, 23 (1995).

24. See, e.g., Anita M. Unruh, *Gender Variations in Clinical Pain Experience*, 65 PAIN 123 (1996) (“In most studies, women report more severe levels of pain, more frequent pain and pain of longer duration than men.”). Unruh’s systematic review of pain studies is by far the most comprehensive treatment of gender variations in pain.

25. See WALLACE & WALLACE, *supra* note 5, at 48–49, 80–81 (migraine headaches and chronic fatigue syndrome); Daniel J. Clauw, *Fibromyalgia*, in KELLEY’S TEXTBOOK OF RHEUMATOLOGY 417, 422 (Shaun Ruddy et al. eds., 6th ed. 2001) (depression and anxiety disorder); Sidney Benjamin et al., *The Association Between Chronic Widespread Pain and Mental Disorder*, 43 ARTHRITIS &

The distinguishing symptom of fibromyalgia—persistent widespread pain—is also the current basis for the disorder’s diagnosis. As with much chronic pain, no laboratory test (e.g., a blood test or muscle biopsy) or similar objective evidence exists to confirm a clinical diagnosis of fibromyalgia. The diagnosis ultimately depends on the patient’s reporting of subjective symptoms. Using a diagnostic “tender point” test, the examining physician applies pressure to select points distributed over the patient’s body and observes the patient’s response.<sup>26</sup> A diagnosis of fibromyalgia is confirmed if the patient reports a sufficient number of painful points and her pain has persisted for at least three months.<sup>27</sup>

This reliance on the patient’s subjective reporting of pain lies at the heart of the controversy concerning the legitimacy of chronic pain syndromes. One of medicine’s irrepressible dogmas is that “illnesses lacking objectivity are not genuine.”<sup>28</sup> Fibromyalgia is one of many “medically unexplained” illnesses whose reality is continually questioned. As Jane Brody of the *New York Times* observes:

“Is it a real disease?” is the most frequently asked question about fibromyalgia, an ailment characterized by chronic widespread musculoskeletal pain. The cynical tone that usually accompanies the question suggests that many think this syndrome is all in a person’s head, the result, perhaps, of an emotional disorder or an attempt to avoid the demands of work and life.<sup>29</sup>

Despite its controversy, subjective-complaint illness is surprisingly routine in medicine.<sup>30</sup> Many common medical symptoms cannot be objectively verified, including fatigue, dizziness, or nausea.<sup>31</sup> Similarly,

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RHEUMATISM 561, 565 (2000) (somatization disorder); BERNE, *supra* note 11, at 95–96, 100–01 (depression and anxiety disorder).

26. See Frederick Wolfe et al., *The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia: Report of the Multicenter Criteria Committee*, 33 ARTHRITIS & RHEUMATISM 160, 162 (1990).

27. See WALLACE & WALLACE, *supra* note 5, at 9–11.

28. See Goldenberg, *supra* note 4, at 782; see also ANDREW MALLESON, WHIPLASH AND OTHER USEFUL ILLNESSES 362 (2003) (“What science cannot encompass, it discredits. Neither medicine nor science has ever been comfortable with subjectivity, though for most of us, such interiority provides meaning and significance to our lives.”).

29. Brody, *supra* note 9.

30. See David L. Eaton, *Scientific Judgment and Toxic Torts—A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & POL’Y 5, 21 (2003) (“Symptoms, and especially those without clear underlying medical explanations, account for a large percentage of clinical encounters.”); Lois B. Morris, *Mind and Body: She Feels Sick. The Doctor Can’t Find Anything Wrong*, N.Y. TIMES, June 24, 2001, at WH4 (“Chest pain, difficulty in breathing, muscle and skeletal pain, dizziness, constipation, abdominal upset, insomnia, fatigue: these symptoms are the bread and butter of the daily practice of medicine. Yet in as many as three-quarters of all cases, doctors can find no disease process to explain their presence.”).

31. See Kevin P. White, *Fibromyalgia: The Answer is Blowin’ in the Wind*, 31 J.

many well-recognized disorders lack objective diagnostic tests, including migraine headaches and epilepsy.<sup>32</sup>

For the present, fibromyalgia pain syndrome retains its hold on medical legitimacy. Indeed, it has been recognized by the American Medical Association, the National Institute of Health, and the World Health Organization.<sup>33</sup> But the diagnosis has withstood withering attack in the past decade. To many medical critics, fibromyalgia is one of several “somatic syndromes” driven by sensationalized media coverage, self-interest, and litigation.<sup>34</sup> For these critics, chronic pain syndromes ultimately reside in the minds of the sufferers.

Psychiatry provides alternative diagnoses for enigmatic chronic pain. These diagnoses give scientific footing to those who believe chronic pain syndromes are fundamentally psychosomatic. The American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR)<sup>35</sup> recognizes a category of mental disorders called “somatoform disorders.” Persons suffering from these disorders unintentionally produce somatic symptoms that mimic true “medical” illness. According to the DSM-IV-TR, the common feature of somatoform disorders is the “presence of physical symptoms that suggest a general medical condition (hence the term *somatoform*) and are not fully explained by a general medical condition . . . or by another mental disorder.”<sup>36</sup> This form of mental disorder, like most chronic pain, occurs overwhelmingly in women.<sup>37</sup>

Within the somatoform disorders is a diagnostic classification devoted especially to those whose principal somatic symptom is pain. This

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RHEUMATOLOGY 636, 637 (2004).

32. See, e.g., Simon Wessely, *Chronic Fatigue Syndrome: A True Illness*, in 10 HORIZONS IN MEDICINE 501–16 (G. Williams ed., 1999), available at [www.kcl.ac.uk/cfs/articles.html](http://www.kcl.ac.uk/cfs/articles.html) (last visited Mar. 8, 2004); Goldenberg, *supra* note 4, at 782. The lack of specific diagnostic tests is common among the rheumatic diseases with which fibromyalgia is classified. See PRIMER ON THE RHEUMATIC DISEASES 133 (John H. Klippel et al. eds., 12th ed. 2001) (“Tests with high specificity are rare in rheumatic diseases.”).

33. See WALLACE & WALLACE, *supra* note 5, at ix. As Wallace and Wallace observe, “There has never been a published peer-review study or report from a medical society challenging the validity of fibromyalgia as a syndrome or construct.” *Id.* at 13. Moreover, neither professional nor judicial acceptance of fibromyalgia has assuaged misgivings about the illness. For example, a recent study of ADA (Americans with Disabilities Act) litigation discovered that “fibromyalgia suits . . . appear to be an almost total failure.” See Afram, *supra* note 13, at 107.

34. See Arthur J. Barsky & Jonathan F. Borus, *Functional Somatic Syndromes*, 130 ANNALS INTERNAL MED. 910, 910 (1999).

35. AM. PSYCHIATRIC ASS’N, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS, DSM-IV-TR (4th ed. rev. 2000) [hereinafter DSM-IV-TR].

36. *Id.* at 485 (emphasis in original).

37. See, e.g., *id.* at 487 (observing that somatization disorder occurs in approximately 0.2%–2.0% of women, and less than 0.2% of men). In the United States, somatization disorder occurs only “rarely” in men. See *id.*

classification is called “pain disorder” and was crafted to designate a group of chronic pain sufferers whose pain is substantially attributable to “psychological factors.”<sup>38</sup> Like somatoform disorders generally, pain disorder cannot be diagnosed if the patient’s pain is attributable to a “general medical condition.”<sup>39</sup>

The DSM-IV-TR’s criteria for somatoform disorders beg a critical question: How does a diagnosing physician decide whether the patient’s chronic pain is explained by a “general medical condition,” like fibromyalgia, rather than a mental disorder? Much depends on whether the physician believes fibromyalgia is a legitimate medical diagnosis.<sup>40</sup> By positing psychogenesis as a default explanation for somatic symptoms, the DSM-IV-TR may indulge physicians’ historical propensity for interpreting enigmatic, intractable symptoms as mental illness.<sup>41</sup> By “diagnostic fiat,” a physician can seemingly transform enigmatic pain into mental illness.<sup>42</sup> As discussed below, this appears to be occurring with increasing frequency.

### III. CHRONIC PAIN AS PSYCHOSOMATIC ILLNESS

The medical community’s acceptance of diagnoses for *both* fibromyalgia and somatoform pain disorder would suggest that, whether styled as an organic or mental illness, chronic pain syndrome has achieved medical legitimacy. This inference would be mistaken, however. Doubt and disparagement inhere in the very notion of psychosomatic illness. Even more sympathetic critics write with an apologetic tone, emphasizing that psychosomatic pain is nonetheless “real.”<sup>43</sup> As they implicitly recognize, psychogenic explanations of illness “inevitably lead to moralistic judgments, such as that psychiatric

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38. *See id.* at 503. The diagnosing physician should also look for signs of “clinically significant distress or impairment in social, occupational, or other important areas of functioning.” *See id.* In the case of fibromyalgia patients who report long-term pain throughout the body, distress or social impairment is common. *See infra* text accompanying notes 183–91.

39. *See id.*

40. The diagnostician’s opinion about cause is central in assessing a “pain disorder.” Diagnostic criteria requires that a clinician assess (a) whether psychological factors play a “major” role in chronic pain, (b) whether these factors play a contributing role along with “a general medical condition,” or (c) whether “a general medical condition” plays a “major” role in chronic pain—in which case a psychiatric disorder is not assessed. *See id.* at 503.

41. *See* Ian R. McWhinney et al., *Rethinking Somatization*, 126 ANNALS INTERNAL MED. 747, 747–48 (1997) (“Given the looseness of these definitions, it is a short step to diagnosing any patients with unexplained symptoms as having a somatoform disorder.”).

42. *See* Laurence J. Kirmayer et al., *Explaining Medically Unexplained Symptoms*, 49 CAN. J. PSYCHIATRY 663, 664 (2004).

43. *See, e.g.*, EDWARD SHORTER, FROM PARALYSIS TO FATIGUE (1992); ELAINE SHOWALTER, HYSTORIES 117 (1997).



diagnoses imply blame or personal weakness.”<sup>44</sup> This is especially true of the psychiatric concept of “somatization,”<sup>45</sup> which implies that “patients are the authors of their own bodily suffering.”<sup>46</sup>

Most patients and sympathetic physicians have come to avoid diagnoses that hint at somatization. First, psychosomatic explanations cast chronic pain into the disfavored realm of “psychic damages,” which are notoriously difficult to prove and thought of as less deserving of compensation.<sup>47</sup> This is aptly illustrated by the recent decision of several leading disability insurers in California to re-classify fibromyalgia as a mental disorder. In *Kennedy v. Unumprovident Corp.*, a class of insured individuals sued to enjoin these insurers from “improperly classifying fibromyalgia as a mental disorder rather than a physical illness in order to pay reduced disability benefits.”<sup>48</sup> The insurers’ attitudes toward fibromyalgia were injudiciously disclosed when their medical consultant described fibromyalgia as “neurosis with a new banner” and its sufferers as “a sorry sick lot.”<sup>49</sup>

A second consequence of psychosomatic pain explanations is that they invite broad-ranging inquiries into the psychological credibility of chronic pain sufferers, which can be especially risky given the higher incidence of emotional illness and stress in chronic illness sufferers.<sup>50</sup> Finally, psychosomatic explanations of chronic pain require fine

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44. See SIMON WESSELY ET AL., CHRONIC FATIGUE AND ITS SYNDROMES 220 (1998); McWhinney et al., *supra* note 41, at 749 (“A diagnosis of somatization, especially when associated with the idea of primary and secondary gain, carries with it the implication of moral failure.”); Kirmayer, *supra* note 42, at 666 (“Locating symptom origins in psychological processes within the patient tends to ascribe responsibility, and even moral blame.”).

45. A “somatizer” is a person who presents ostensibly physical symptoms like pain or fatigue but who is “actively producing physical symptoms through an unconscious intention or tension.” See Harold Merskey, *Pain, Psychogenesis, and Psychiatric Diagnosis*, 12 INT’L REV. PSYCHIATRY 99, 101 (2000) [hereinafter Merskey, *Psychiatric Diagnosis*]. The concept of somatization is inapplicable when there are medical explanations for the symptoms. See Harold Merskey, Editorial, *Beware Somatization*, 4 EUR. J. PAIN 3 (2000) [hereinafter Merskey, Editorial].

46. McWhinney et al., *supra* note 41, at 748.

47. See Lars Noah, *Pigeonholing Illness: Medical Diagnosis as a Legal Construct*, 50 HASTINGS L.J. 241, 306 (1999) (commenting on “[t]he disdain for psychological explanations of illness, and suggesting that such a diagnosis makes the patient’s complaint less real genuine . . .”); Kelly L. Centofanti, *Introducing Evidence of Psychological Treatment for Chronic Pain*, ASS’N OF TRIAL LAWYERS OF AM., July 2000, available at Westlaw 2 Ann. 2000 ATLA CEL 2797 (conference reference materials) (2000) (noting a “universal skepticism” among jurors).

48. 50 Fed. Appx. 354 (9th Cir. 2002) (refusing class certification for failure to satisfy “typicality” requirements of relevant procedural rules).

49. *Id.*, Appellant’s Reply Brief, 2002 WL 32115565 (2002).

50. See *infra* text accompanying notes 185, 191. One authority observes that plaintiffs’ lawyers avoid claims of traumatically induced mental illness because of the fear that “the claim of psychological injury can be easily challenged and rebuffed by defense counsel through her mental health experts.” See Jerry von Talge, *Major Depressive Disorder*, 26 AM. JUR. PROOF OF FACTS 3D 1 (2003).

distinctions between symptoms that are the legitimate result of “unconscious” motives and the illegitimate result of patient fabrication.<sup>51</sup> As discussed below, many advocates of the psychosomatic theory of chronic pain find little distinction between “somatizing” and malingering,<sup>52</sup> and courts likewise appear to have difficulty drawing this distinction.

The literature on psychosomatic pain is extensive. The following discussion offers a representative sample of both popular and medical advocacy of the psychosomatic theory of chronic pain arguing from two premises. First, chronic pain is squarely placed within the social history of “fashionable illnesses” like neurasthenia and hysteria, which are thought to have plagued vulnerable populations of Anglo-American women for more than a century. Second, this cultural narrative is sustained by the seeming absence of medical evidence confirming a biological basis for chronic pain.

#### A. Psychosomatic Pain in the Critical Literature

Edward Shorter’s *From Paralysis to Fatigue* is the most well-researched cultural history of psychosomatic illness. Shorter traces the evolution of somatization from nineteenth-century hysteria<sup>53</sup> and neurasthenia<sup>54</sup> to modern syndromes like fibromyalgia and chronic fatigue. He contends that mentally distressed persons unconsciously seek to express their distress through socially acceptable symptoms having the appearance of “legitimate organic disease.”<sup>55</sup> Pain and fatigue are the most prevalent symptoms of psychosomatic illness, with chronic pain being the “most common form of somatization in American society.”<sup>56</sup> According to Shorter, fibromyalgia and chronic fatigue syndrome are examples of pseudo-organic illnesses that have been

51. See generally Jack H. Olender, *Showing Pain and Suffering*, 5 AM. JUR. TRIALS 921, 934 (noting that psychogenic pain poses problems for plaintiffs as it “encourages the charge by defense counsel that the plaintiff is malingering”).

52. The term “malingering” is usually defined as “conscious and willful feigning or exaggeration or a disease or effect of an injury in order to obtain specific external gain.” See, e.g., AM. MED. ASS’N, GUIDES TO EVALUATION OF PERMANENT IMPAIRMENT 601 (2001).

53. Hysteria, perceived as a disease of the central nervous system, was never well described in nineteenth century medicine, but was generally associated with depression, stress reactions, and physical symptoms like loss of sensation, headache, and convulsive fits. See generally SHORTER, *supra* note 43, at 175–76; Merskey, *supra* note 1, at 67. Hysteria was primarily diagnosed in women. See SHOWALTER, *supra* note 43, at 62–65.

54. Neurasthenia, also thought to be a disease of the central nervous system, was principally characterized by chronic fatigue and has been called “a masculine form of hysteria.” See WESSELY ET AL., *supra* note 44, at 105–06.

55. SHORTER, *supra* note 43, at 3.

56. *Id.* at 297.

appropriated by modern-day hysterics. Shorter describes how illness constructs like fibromyalgia have achieved epidemic proportions:

An epidemic of illness attribution, or epidemic hysteria, seems to involve two phases: (1) appropriating a genuine organic disease—whose cause is difficult to detect and substantiate—as a template; (2) broadcasting this template to individuals with often quite different symptoms, who then embrace this template as the explanation of their problems. This broadcasting is effected by sympathetic physicians, patient support groups, and the media.<sup>57</sup>

Those afflicted with psychosomatic illness, Shorter argues, “cling tenaciously” to their pseudo-organic symptoms rather than confront their mental distress.<sup>58</sup> At the same time, illness sufferers display an extraordinary “pathoplasticity”<sup>59</sup> enabling them to exchange symptoms with the advent of new, fashionable illnesses.

English professor Elaine Showalter restates Shorter’s thesis from a feminist perspective in her popular book, *Hystories*.<sup>60</sup> Similar to Shorter, she views illnesses like fibromyalgia and chronic fatigue syndrome as latter-day “hysterical syndromes.”<sup>61</sup> The United States, Showalter tells us, “has become the hot zone of psychogenic diseases, new and mutating forms of hysteria amplified by modern communications and *fin de siècle* anxiety.”<sup>62</sup> Hysteria, she observes, “‘has come to imply behavior that produces the appearance of disease,’ although the patient is unconscious of the motives for feeling sick.”<sup>63</sup> Women, she notes, outnumber men “in virtually every category of unexplained illness” and demonstrate a special “aptitude for somatization.”<sup>64</sup> Somatizers, she suggests, occupy a shadow world between legitimate illness and malingering. Showalter observes:

We need not assume that patients are either describing an organic

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57. *Id.* at 305.

58. *Id.* at 301.

59. *Id.* at 318.

60. Showalter’s book does not discuss fibromyalgia except to note that it is used synonymously with “chronic fatigue syndrome,” which she discusses extensively. SHOWALTER, *supra* note 43, at 124. Showalter has indicated that she views fibromyalgia as one of the “hystories” that is the subject of her text. *See, e.g.*, Canadian Broadcasting Corporation, <http://www.cbc.ca/ideas/features/shows/syndromes> (CBC radio production on fibromyalgia where Showalter includes fibromyalgia among the syndromes she criticizes in her writings). Critics of fibromyalgia employ Showalter’s work to support their attacks on chronic pain syndrome. *See, e.g.*, George E. Ehrlich, *Pain is Real; Fibromyalgia Isn’t*, 30 J. RHEUMATOLOGY 1666 (2003).

61. SHOWALTER, *supra* note 43, at 12.

62. *Id.* at 4.

63. *Id.* at 14 (quoting PHILLIP R. SLAVNEY, PERSPECTIVES ON HYSTERIA 1–2 (1990)).

64. *Id.* at 9, 17.

disorder or else lying when they present similar narratives of symptoms. Instead, patients learn about diseases from the media, unconsciously develop the symptoms, and then attract media attention in an endless cycle. The human imagination is not infinite, and we are all bombarded by these plot lines every day. Inevitably, we all live out the social stories of our time.<sup>65</sup>

According to Showalter, hysterical syndromes are a form of “protolanguage”<sup>66</sup> and protest, which exist on “one extreme of a continuum with feminism.”<sup>67</sup> This psychosomatic protest, Showalter emphasizes, is “ultimately self-destructive.”<sup>68</sup>

The most recent contribution to the popular literature of somatization is Andrew Malleon’s *Whiplash and Other Useful Illnesses*.<sup>69</sup> Malleon is a psychiatrist and specialist in internal medicine. He shares the view that many popular illnesses affecting “disproportionate numbers of women” are a form of somatization.<sup>70</sup> Unlike Shorter and Showalter, Malleon supports his view of somatization with medical research rather than social history. The basis for his contention that fibromyalgia is a form of somatizing is that “no hard evidence has ever been forthcoming” to show that fibromyalgia has a “physical basis.”<sup>71</sup> Regarding research efforts to find the biological origins of fibromyalgia, Malleon makes the breathtaking claim that “[a]ll laboratory investigations have proved negative.”<sup>72</sup> As shown later, this claim is demonstrably false.<sup>73</sup>

Malleon ultimately shows little sympathy for the sufferers of chronic pain, whose life of somatizing he views as a fraud on the health care and legal systems.<sup>74</sup> He observes, of somatizers in the legal process: “Whether the plaintiff’s intentions are unconscious or not, the results are the same. The plaintiff, by his behavioural strategies, manipulates his world to get what he wants.”<sup>75</sup> Malleon is also harsh in his appraisal of medical and legal professionals who exploit “useful illnesses.” He derides the “immense amount of medical literature . . . devoted to

65. *Id.* at 6.

66. *Id.* at 7 (quoting Robert M. Woolsey, *Hysteria: 1875 to 1975*, 37 DISEASES CENTRAL NERVOUS SYS. 379 (1976)).

67. *Id.* at 10 (citing ELAINE SHOWALTER, *THE FEMALE MALADY* (1985)).

68. *Id.*

69. MALLEON, *supra* note 28.

70. *Id.* at 319 (“I have repeatedly emphasized the disproportionate numbers of women who develop fashionable illnesses.”).

71. *Id.* at 170. Malleon also asserts that “many of fibromyalgia’s symptoms are clearly somatoform,” an assertion he never chooses to support. *See id.*

72. *Id.*

73. *See infra* text accompanying notes 242–67.

74. *See id.* at 301.

75. *Id.*

ingenious attempts” to find a physical basis for chronic pain.<sup>76</sup> For Malleson, fibromyalgia is one of many compensation-driven illnesses, including post-traumatic stress disorder, post-traumatic brain injury, “TMJ” disorder, and chronic fatigue syndrome.<sup>77</sup>

An appreciable amount of medical literature on fibromyalgia now echoes the themes of popular writers like Shorter, Showalter, and Malleson. Malleson’s rhetorical style often prevails, however, as one finds an abundance of hyperbole in what have been labelled the “fibromyalgia wars.”<sup>78</sup> According to several medical critics, fibromyalgia is the quintessential “junk science,”<sup>79</sup> a “bogus construct”<sup>80</sup> that should not be dignified with a diagnostic label. Fibromyalgia has led to a “life of somatizing,”<sup>81</sup> and “malinger,”<sup>82</sup> and the “manufacturing of victims”<sup>83</sup> who seek “secondary gains”<sup>84</sup> in the disability and tort systems. Fibromyalgia has created a compensation “monster”<sup>85</sup> and a “cash cow”<sup>86</sup> exploited by lawyers and physicians.<sup>87</sup> The result is a legal crisis.<sup>88</sup>

In sum, several different strands to the psychosomatic critique of chronic pain exist, many of which are unflattering to the pain sufferer. More benign critics interpret chronic pain as latter-day hysteria. Pain is

76. *Id.* at 29. Malleson makes this observation in the context of research concerning whiplash pain, which he views as the precursor to fibromyalgia. *See id.* at 164 (commenting that, through developing medical theories of fibromyalgia, “whiplash and other musculoskeletal pain received a new lease on life”).

77. *See id.* at 63, 103, 139, 211.

78. *See* Frederick Wolfe, *Stop Using the American College of Rheumatology Criteria in the Clinic*, 30 J. RHEUMATOLOGY 1671 (2003) (noting the “FM wars that, even now, continue to rage”).

79. *See, e.g.*, Thomas Bohr, *Problems with Myofascial Pain Syndrome and Fibromyalgia Syndrome*, 46 NEUROLOGY 593, 594 (1995) [hereinafter Bohr, *Fibromyalgia Syndrome*]; Thomas Bohr, *Letter to the Editor*, 74 AM. J. PHYSICAL MED. & REHABILITATION 476 (1996).

80. *See* Milton L. Cohen & John L. Quintner, *Fibromyalgia Syndrome and Disability: A Failed Construct Fails Those in Pain*, 168 MED. J. AUSTRALIA 402, 404 (1998).

81. *See, e.g.*, Nortin M. Hadler, *Fibromyalgia: La Maladie est Morte. Vive le Malade!*, 24 J. RHEUMATOLOGY 1250-1251 (1997); Nortin M. Hadler, *Viewpoint, Fibromyalgia, Chronic Fatigue, and Other Iatrogenic Diagnostic Algorithms*, 102 POSTGRADUATE MED. 161, 176 (1997).

82. *See, e.g.*, Frederick Wolfe, *For Example Is Not Evidence*, 27 J. RHEUMATOLOGY 1115, 1116 (2000); Angelis Mailis & Ann Taylor, *Letter to the Editor*, <http://www.jrheum.com/subscribers/01/03/correspondence.html>.

83. *See* Mailis & Taylor, *supra* note 82.

84. *See, e.g.*, Barsky & Borus, *supra* note 34, at 915.

85. *See* Simon Carette, *Fibromyalgia 20 Years Later: What Have We Really Accomplished?*, 22 J. RHEUMATOLOGY 590, 592 (1995).

86. *See* Bohr, *Fibromyalgia Syndrome*, *supra* note 79, at 593.

87. *See, e.g.*, Frederick Wolfe, *The Fibromyalgia Problem*, 24 J. RHEUMATOLOGY 1247 (1997); Robert Ferrari & Anthony Russell, *Letter to the Editor*, <http://www.jrheum.com/subscribers/01/03/correspondence.html>.

88. *See, e.g.*, Wolfe, *supra* note 87, at 1247. With the exception of Wolfe, none of the cited critics has published original research on chronic pain.

the manifestation of unresolved psychic conflict or, in Showalter's rhetoric, "a code used . . . to communicate a message which, for various reasons, cannot be verbalized."<sup>89</sup> Less benign critics show faint sympathy for the chronic pain patient or her supporting professionals, and view chronic pain as a form of fraud. Under this view, psychosomatic pain "gives lawyers a contentious issue about which they can litigate; healthcare practitioners an illness that they can endlessly treat and never cure; and patients an illness for which they can claim both sympathy and compensation."<sup>90</sup>

### *B. Psychosomatic Pain Theory in the Courts*

"Chronic pain syndrome" or "fibromyalgia" was first introduced to the courts in disability litigation.<sup>91</sup> Half of the Social Security disability disputes now pending in federal court involve claims of chronic pain, and no signs indicate that the controversy attending these claims has waned.<sup>92</sup>

Courts generally accept that subjective pain can result in disability, and that fibromyalgia or chronic pain syndrome is a legitimate illness within the meaning of disability laws. This has been facilitated by congressionally mandated investigation of the problem of chronic pain,<sup>93</sup> as well as the Social Security Administration's recognition that subjective symptoms can be validated through use of medical "signs" like the tender point test.<sup>94</sup> Widespread use of this test remains controversial, however, and one of the test's co-developers, Frederick Wolfe, has now urged that the test be abandoned in disability

89. See Woolsey, *supra* note 66, at 379.

90. See MALLESON, *supra* note 28, at 178.

91. See, e.g., *Szwandrok v. Bowen*, 658 F. Supp. 847 (N.D. Ill. 1987). The decision in *Bowen* is the first reported decision that actually employed the term "fibromyalgia." The illness was usually referred to as "chronic pain syndrome" in decisions before *Bowen*.

92. See Bierman, *supra* note 6, at 273.

93. See Michael G. Ruppert, *Developments in Social Security Law*, 22 IND. L. REV. 401, 404-06 (1989) (discussing the congressionally authorized Commission on the Evaluation of Pain); Jon C. Dubin, *Poverty, Pain, and Precedent: The Fifth Circuit's Social Security Jurisprudence*, 25 ST. MARY'S L.J. 81, 95-96 (1993) (same).

94. See Social Security Ruling 99-2p: Policy Interpretation Ruling Titles II and XVI: Evaluating Cases Involving Chronic Fatigue Syndrome (CFS) (1999) (citing as example of "medical signs that establish the existence of a medically determinable impairment" the "presence of tender points" upon medical examination). Another ruling, SSR 96-7p (1996) emphasizes that claimant symptoms cannot be the basis for a finding of disability "unless there are medical signs and laboratory findings demonstrating the existence of a medically determinable physical or mental impairment . . ." See Notice of Social Security Ruling, 61 Fed. Reg. 34488-01 (July 2, 1996). However, once signs like the tender point examination confirm the existence of a medically determinable impairment, administrative judges are adjured to "carefully consider" the claimant's subjective reporting of symptoms and not disregard them "solely because they are not substantiated by objective medical evidence." *Id.*

proceedings.<sup>95</sup>

Disability courts have yet to agree upon a single illness paradigm for chronic pain syndrome. Virtually no discussion exists regarding the emerging biological model of chronic pain, which, as discussed later, views this pain as primarily a disease of the central nervous system.<sup>96</sup> As a consequence, claims of chronic pain are frequently challenged because they make no sense under the prevailing biological model of acute pain that “equates pathogenesis with tissue damage or dysfunction.”<sup>97</sup>

The psychiatric model of chronic pain, on the other hand, is often urged. Defendants commonly argue that the claimant suffers from a somatoform disorder and related mental illness and, therefore, is not truly disabled.<sup>98</sup> Allegations of somatization often dovetail with challenges to the claimant’s credibility.<sup>99</sup> Somewhat surprisingly, the occasional claimant actually attributes her pain to a somatoform disorder.<sup>100</sup> This tack reflects the fact that disability law covers “mental” as well as “physical” impairments. However, it is questionable whether such a strategy is wise.

A recent decision by Judge Richard Posner for the U.S. Court of Appeals for the Seventh Circuit demonstrates how the popular debate about somatization has worked its way into disability litigation. In

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95. See Frederick Wolfe & Joshua Potter, *Fibromyalgia and Work Disability: Is Fibromyalgia a Disabling Disorder?*, 22 RHEUMATIC DISEASE CLINICS OF N. AM. 369 (1996). See also Norton M. Hadler & George Ehrlich, Editorial, *Fibromyalgia and the Conundrum of Disability Determination*, 45 J. OCCUPATIONAL & ENVTL. MED. 1030 (2003).

96. See *infra* text accompanying notes 242–67. The sole reported decision even mentioning the view that chronic pain results from dysfunction in the central nervous system is *Hocraffer v. Secretary Health & Human Services*, No. 99-533V, 2004 WL 627777 (Fed. Cl. Mar. 12, 2004). In *Hocraffer*, the testifying expert successfully argued that a disability claimant’s upper chest pain and other symptoms resulted from a “[c]entral [s]ensitization [s]yndrome” she suffered after receiving vaccinations. *Id.* at \*11.

97. See Robert M. Bennett, *Fibromyalgia and the Disability Dilemma*, 39 ARTHRITIS & RHEUMATISM 1627 (1996).

98. See, e.g., *Carradine v. Barnhart*, 360 F.3d 751 (7th Cir. 2004) (arguing that claimant’s chronic pain resulted from somatoform disorder and malingering); *Kennedy v. Unumprovident Corp.*, 50 Fed. Appx. 354 (9th Cir. 2002) (Appellant’s Brief at 2002 WL 32115565) (arguing that chronic pain resulted from pain disorder and depression); *Bates v. Apfel*, 69 F. Supp. 2d 1143 (N.D. Iowa 1999) (arguing that claimant suffered from hypochondriasis and symptom exaggeration); *Lange v. Long-Term Disability Plan of Sponsor Applied Remote Tech., Inc.*, 125 F.3d 794 (9th Cir. 1997) (arguing that claimant’s chronic pain was a “mental disorder” not compensable under ERISA disability plan). See generally Gene Stephens Connolly, *Hidden Illness, Chronic Pain: The Problems of Treatment and Recognition of Fibromyalgia in the Medical Community*, 5 DEPAUL J. HEALTH CARE L. 111, 119–28 (discussing various decisions in which psychosomatic theories of chronic pain were argued defensively).

99. See *infra* notes 101–07.

100. See, e.g., *Mitchell v. Sec’y of Health & Human Servs.*, No. 93–1612, 1994 WL 96966, at \*5 (1st Cir. Mar. 24, 1994).

*Carradine v. Barnhart*,<sup>101</sup> the middle-aged victim of a slip-and-fall accident sought disability benefits for her chronic pain and fibromyalgia. The administrative law judge (ALJ) denied benefits based on his conclusion that the claimant was “exaggerating” her pain as the result of a diagnosed somatoform disorder.<sup>102</sup> The Seventh Circuit reversed and criticized the ALJ for inferring that the discrepancy between the claimant’s medical reports and her avowals of pain indicated an intentional magnification of her symptoms. According to Judge Posner, the ALJ had confused somatization with malingering:

Pain is always subjective in the sense of being experienced in the brain. The question whether the experience is more acute because of a psychiatric condition is different from the question whether the applicant is pretending to experience pain, or more pain than she actually feels. The pain is genuine in the first, the psychiatric case, though fabricated in the second. The cases involving somatization recognize this distinction.<sup>103</sup>

Judge Posner’s understanding of the psychiatric origins of pain “exaggeration” is shared by other courts. For example, the U.S. Court of Appeals for the Eighth Circuit has described somatization as a legitimate “psychiatric disorder that causes the sufferer to have a distorted perception of physical ailments.”<sup>104</sup> Still other courts have referred to the legitimate “exaggeration of symptoms” occurring in psychogenic pain syndrome and distinguished this exaggeration from intentional falsification of symptoms.<sup>105</sup> As these decisions illustrate, even benign characterizations of somatized pain tend to employ a language that invites doubt. Distinctions between legitimate and illegitimate pain are not easily drawn.<sup>106</sup> Judge Posner, for example, has commented that the symptoms of fibromyalgia are “easy to fake.”<sup>107</sup> Not surprisingly,

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101. 360 F.3d 751 (7th Cir. 2004).

102. *Id.* at 754–55.

103. *Id.* at 754.

104. *Metz v. Shalala*, 49 F.3d 374, 377 (8th Cir. 1995).

105. *See Bates v. Apfel*, 69 F. Supp. 2d 1143, 1149 (N.D. Iowa. 1999).

106. *See, e.g., Benecke v. Barnhart*, 379 F.3d 587, 592 (9th Cir. 2004) (psychiatrist examining claimant concluded that her claimed physical symptoms were “scarcely credible” and diagnosed her with “somatization disorder”); *Wirth v. Barnhart*, 318 F. Supp. 2d 726, 743 (E.D. Wis. 2004) (reversing administrative law judge who rejected credibility of claimant who was diagnosed with “chronic pain syndrome and somatization”). *See also Goldthwait v. Barnhart*, No. 04–110–P–H, 2004 WL 2862167, at \*4 (D. Me. Dec. 13, 2004) (counsel asserted that psychiatrist’s diagnosis referred to “suffering from somatoform disorder or . . . malingering” as opposed to “legitimate[] . . . pain”).

107. *See Sarchet v. Chater*, 78 F.3d 305, 307 (7th Cir. 1996). Whether Judge Posner is correct is unclear. Many critics of chronic pain argue that reliable diagnostic tests can screen out malingerers. *See, e.g., Kevin W. Greve & Kevin J. Bianchi, More on the Clinical and Scientific Relevance of “Symptom Amplification” and Psychological Factors in Pain*, 110 PAIN 499 (2004) (claiming there are



chronic pain litigation often focuses on both the claimant's mental state and her credibility.

Moving from the disability to the tort setting, somatization becomes almost exclusively a defendant's diagnosis. Chronic pain claims are especially contentious in tort and workers' compensation disputes, where plaintiffs attempt to link their pain symptoms to physical traumas such as motor vehicle accidents,<sup>108</sup> workplace accidents,<sup>109</sup> slip-and-fall mishaps,<sup>110</sup> chemical exposure,<sup>111</sup> food poisoning,<sup>112</sup> vaccination,<sup>113</sup> and environmental toxins.<sup>114</sup> Most of these claims are viable only if chronic pain originates in "physical" processes. For this reason, a common defense strategy is to attribute the plaintiff's chronic pain to pre-existing psychological distress, especially a recognized somatoform disorder. This strategy appears to have been successful in some cases.<sup>115</sup>

It seems, then, that the concept of psychosomatic pain has gained solid footing in the courts. Most often this works to the disadvantage of the chronic pain sufferer. Psychosomatic explanations of pain inevitably raise questions about the legitimacy of pain symptoms and the credibility of the sufferer. Therefore, when plaintiffs attempt to link their chronic pain to discrete physical trauma, psychosomatic explanations undermine the plaintiff's attempt to establish both causation and damages.

A review of judicial attitudes about chronic pain syndromes would be incomplete without mentioning a Canadian court decision that has achieved international notoriety. Courts in Canada and the United

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"powerful psychological assessment techniques capable of detecting . . . [s]omatization and the intentional exaggeration of physical symptoms"); Robert Ferrari, *Letter to the Editor*, 110 PAIN 500, 501 (2004) (arguing that fibromyalgia patients should be screened with "effort tests" to detect feigned symptoms).

108. See *Vargas v. Lee*, 317 F.3d 498 (5th Cir. 2003).

109. See *Jones v. Conrad*, No. CA2000-12-257, 2001 WL 1001083 (Ohio Ct. App. Sept. 4, 2001).

110. See *Black v. Food Lion*, 171 F.3d 308 (5th Cir. 1999).

111. See *Alder v. Bayer Corp.*, 61 P.3d 1068 (Utah 2002).

112. See *Gross v. King David Bistro, Inc.*, 83 F. Supp. 2d 597 (D. Md. 2000).

113. See *Awad v. Sec'y of Health & Human Servs.*, No. 92-79V, 1995 WL 366013 (Fed. Cl. June 5, 1995).

114. See *Minner v. Am. Mortgage & Guaranty Co.*, 791 A.2d 826 (Del. Super. Ct. 2000).

115. See, e.g., *Brown v. Patriot Maintenance, Co.*, 99 P.3d 544, 546 (Alaska 2004) (defense expert argued that plaintiff suffered from "major depression and perhaps a miscellaneous somatoform disorder" and that symptoms were more "psychiatric" than "physical"); *State v. Sch. Employees Ret. Bd.*, No. 99AP-1474, 2004 WL 2803446, at \*6 (Ohio Ct. App. 2004) (defense expert argued that plaintiff probably suffered from "somatoform disorder" and could return to work); *Pociask v. Sec'y of Health & Human Servs.*, No. 96-569V, 1999 WL 199053 (Fed. Cl. 1999) (physician attributed claimant's chronic pain to "life stresses"); *Awad*, 1995 WL 366013, at \*16 (physician associated claimant's chronic pain with "depression or other psychological problems").

Kingdom have, like their counterparts in the United States, struggled to understand the phenomenon of chronic pain. In 1994, the Queen's Bench in Alberta, Canada, issued a decision that dismayed chronic pain advocates and disclosed a judicial skepticism of chronic pain that some suspect is widespread.

In *Mackie v. Wolfe*,<sup>116</sup> a plaintiff with obvious credibility issues sued for chronic pain damages allegedly caused by an auto accident. The litigants summoned an impressive array of expert witnesses to essentially debate the medical validity of fibromyalgia, or "chronic benign pain syndrome" as it is sometimes referred to by Canadian courts. The trial court began its opinion with the unconvincing avowal that the plaintiff's pain was "real."<sup>117</sup> After reviewing extensive expert testimony, the court quoted the "most helpful" expert. He testified that "the one thing we know for sure, patients with fibromyalgia do follow certain personality traits."<sup>118</sup> This pain-prone person, the expert indicated, had an inability to cope with stress<sup>119</sup> and was likely a particularly out-of-shape, overweight woman.<sup>120</sup> These psychologically predisposed persons "become very focused on themselves, on their own symptoms, and go on to develop that secondary gain either psycho-social . . . or can be monetary as well [sic]."<sup>121</sup>

The court was ultimately persuaded that chronic pain lacks any organic basis. As one defense expert convincingly argued, "the condition has been around since the Spanish Inquisition or before" but is not really "a condition that exists physically."<sup>122</sup> Several defense experts persuasively testified that fibromyalgia is not "an organic diagnosis," and that the plaintiff's "problems were psychological."<sup>123</sup> A variety of psychiatric diagnoses were liberally applied to the plaintiff,

116. 153 A.R. 81 (Alta. Queens Bench 1994).

117. *Id.* at 96.

118. *Id.* at 114. There is overwhelming consensus that research reveals no "typical" pain patient or a pain-prone personality. See Ann Gamsa, *The Role of Psychological Factors in Chronic Pain. II. A Critical Appraisal*, 57 PAIN 17, 22-23 (1994). The court's belief that there is pain-prone personality has been described by Gamsa as the "uniformity myth." See *id.* at 22 (citation omitted).

119. The theme that chronic pain sufferers lack the "ability to cope" is pervasive in medical literature critical of this pain. Expressions of the theme are seldom clear about whether the inability to cope reflects a psychological disorder, learned behavior, or malingering. See, e.g., Anthony S. Russell, *Fibromyalgia—A Historical Perspective*, 3 J. MUSCULOSKELETAL PAIN 43, 44 (1995) ("I believe that the constellation of symptoms we recognize is a reflection of an inability to cope, often because of patterns of behavior or expectation set up in childhood. As long as the rewards of this behavior pattern, provided by society, by doctors, etc., outweigh the debits, the pattern will be encouraged both at an individual and societal level.").

120. 153 A.R. at 114.

121. *Id.*

122. *Id.* at 116.

123. *Id.* at 120.

including “somatoform disorder,”<sup>124</sup> “hysterical conversion,”<sup>125</sup> “hypochondriasis,” and “malingering.”<sup>126</sup> The court also integrated social history into its opinion, featuring the psychosomatic theories of Shorter’s *From Paralysis to Fatigue*,<sup>127</sup> and quoted an expert wryly observing that the tender point exam for diagnosing fibromyalgia bore similarities to medieval tests for identifying witches.<sup>128</sup>

In conclusion, the court declared that fibromyalgia “does not exist as a physical condition” but is instead “a court-driven ailment that has mushroomed into big business for plaintiffs”:<sup>129</sup>

The evidence in this case satisfies me that the symptoms diagnosed as fibromyalgia are a relabeling of a condition . . . that has been with mankind for hundreds of years and represents a personality disorder. This particular disorder is often found in individuals who will not or cannot cope with everyday stresses of life and convert this inability into acceptable physical symptoms to avoid dealing with reality.<sup>130</sup>

The trial court’s judgment for the defense was affirmed on appeal, although the appellate court oddly observed that “the trial judge nowhere expressly says that fibromyalgia does not exist.”<sup>131</sup>

#### IV. RE-ASSESSING PSYCHOGENIC PAIN

Terrible ideas, formed only in the imagination, will affect the brain and the body with painful sensations.<sup>132</sup>

##### A. A Role for Psychological Factors in Pain

The belief that psychological factors are involved in the experience of pain is now widely accepted. Since Ronald Melzak and Patrick Wall announced their famous “gate” theory of pain in 1965,<sup>133</sup> researchers have recognized that thoughts and emotions can modify the common

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124. *Id.* at 121.

125. *Id.* at 122.

126. *Id.* at 123, 131.

127. *Id.* at 123–24.

128. *Id.* at 123.

129. *Id.* at 138.

130. *Id.* at 139.

131. *See Mackie v. Wolfe*, 184 A.R. 339 (Alta. Ct. App. 1996).

132. RICHARD BLACKMORE, A TREATISE OF THE SPLEEN AND VAPOURS: OR HYPOCHONDRIACAL AND HYSTERIAL AFFLICTIONS (1725).

133. *See* Ronald Melzack & Patrick D. Wall, *Pain Mechanisms: A New Theory*, 150 SCIENCE 971 (1965).

sensation of pain.<sup>134</sup> In fact, there is growing acceptance of the role of psychological factors in disease generally, which is captured in the “biopsychosocial” model of disease proposed by George Engel in 1977.<sup>135</sup> As Engel contended, “psychophysiologic responses to life change may interact with existing somatic factors to alter susceptibility and thereby influence the time of onset, the severity, and the course of a disease.”<sup>136</sup> Engle challenged exclusive reliance on the “biomedical” model then prevalent in medicine, and explicitly called upon researchers to “include the psycho-social without sacrificing the enormous advantages of the biomedical approach.”<sup>137</sup>

The biopsychosocial model of illness has been widely accepted in medicine<sup>138</sup> and had noticeable impact on the concept of pain. This is reflected in the leading definition of pain developed by psychiatrist Harold Merskey and adopted in the authoritative *Classification of Chronic Pain*.<sup>139</sup> Pain is broadly defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”<sup>140</sup> As Merskey recently commented, this definition recognizes that pain is a “monistic” experience and that clinicians must identify “the individual contributions of all relevant organic, psychological and social factors to pain . . . .”<sup>141</sup>

The biopsychosocial model of pain now draws support from biological insights into the pain process. Brain-mapping studies, for example, confirm that “pain centers and emotion-processing regions overlap.”<sup>142</sup> Similarly, chemical neurotransmitters like serotonin and endorphin are known to play a role in both pain and mood disorders.<sup>143</sup>

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134. See Robert J. Gatchel & Jeffrey Dersh, *Psychological Disorders and Chronic Pain: Are There Cause-and-Effect Relationships*, in PSYCHOLOGICAL APPROACHES TO PAIN MANAGEMENT 30 (2d ed. 2002).

135. See George J. Engel, *The Need for a New Medical Model: A Challenge for Biomedicine*, 196 SCIENCE 129 (1977).

136. *Id.* at 132.

137. *Id.* at 131.

138. There is still controversy about the manner in which psycho-social factors influence disease onset and course. An illustration of this controversy is found in a recent debate featured in Psychosomatic Medicine. See, e.g., Redford B. Williams & Neil Schneiderman, *Resolved: Psychosocial Interventions Can Improve Clinical Outcomes in Organic Disease (Pro)*, 64 PSYCHOSOMATIC MED. 552 (2002); Arnold S. Relman & Marcia Angell, *Resolved: Psychosocial Interventions Can Improve Clinical Outcomes in Organic Disease (Con)*, 64 PSYCHOSOMATIC MED. 558 (2002).

139. See INT’L ASS’N FOR THE STUDY OF PAIN, *supra* note 19.

140. *Id.* at 210 (emphasis added).

141. See Harold Merskey, *Distortion of the Biopsychosocial Approach*, 113 PAIN 241 (2005).

142. See Caroline Ash, *Pain Viewed Dispassionately*, 304 SCIENCE 826 (2004).

143. See, e.g., Thorstein Giesecke et al., *The Relationship Between Depression, Clinical Pain, and Experimental Pain in a Chronic Pain Cohort*, 52 ARTHRITIS & RHEUMATISM 1577 (2005) (discussing neurotransmitters involved in both chronic pain and major depressive disorder); Melanie Thernstrom,

Further, ample evidence suggests that the pain process is influenced by dysfunction in the hypothalamic-pituitary-adrenal (HPA) axis, which is integrally involved with the human stress response.<sup>144</sup> These findings emphasize that “biological” and “psychosocial” factors can play an interactive role in the pain process. They also highlight how the distinction between “physical” and “mental” aspects of illness may be a heuristic device rather than an ontological divide.<sup>145</sup>

The biopsychosocial model of pain cannot, however, be equated with psychosomatic explanations of pain. The biopsychosocial model of illness recognizes the contribution of mental factors to the physiological processes involved in pain. Psychosomatic explanations ultimately *reduce* chronic pain to mental factors, the consequences of which are significant.<sup>146</sup> One consequence, mentioned previously, is that psychosomatic pain is inevitably devalued and the credibility of its sufferers is questioned.<sup>147</sup> Another consequence is that accepted treatments for “physical” pain, like analgesics,<sup>148</sup> may be discouraged in

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*Pain, The Disease*, N.Y. TIMES, Dec. 16, 2001, at 66 (“Anxiety and depression are not merely cognitive responses to pain; they are physiologic consequences of it. Pain and depression share neural circuitry. The hormones that modulate a healthy brain, like serotonin and endorphins, are the same ones that modulate depression.”).

144. See, e.g., Anthony J. Cleare, *Stress and Fibromyalgia—What is the Link?*, 57 J. PSYCHOSOMATIC RES. 423, 424 (2004) (“There does appear to be accumulating evidence that the HPA axis is dysregulated in fibromyalgia, in the direction of . . . an altered response to stressful challenge.”); Jain et al., *supra* note 15, at 56 (observing relationship between pain-related neurotransmitters and blunted stress response); Stanley R. Pillemer et al., *The Neuroscience and Endocrinology of Fibromyalgia*, 40 ARTHRITIS & RHEUMATISM 1928, 1931–32 (1997) (discussing physiological influence of stress on pain perception and mood).

145. As the DSM-IV-TR recognizes, modifiers like “mental” and “physical” are “terms of convenience.” See DSM-IV-TR, *supra* note 35, at xxx, xxxv.

146. A useful comparison can be made with concededly organic disease. For example, researchers have found that even mild symptoms of depression can increase the risk of death from myocardial infarction (heart attack). See D.E. Bush et al., *Even Minimal Symptoms of Depression Increase Mortality Risk After Acute Myocardial Infarction*, 88 AM. J. CARDIOLOGY 337 (2001). Similarly, persons suffering from multiple sclerosis may have an increased risk of symptom exacerbation after experiencing a stressful life event. See D. Buljevac et al., *Self-reported Stressful Life Events and Exacerbations in Multiple Sclerosis: Prospective Study*, 327 BRITISH MED. J. 646 (2003). Yet while heart attacks and the symptoms of multiple sclerosis can be more fully understood using a biopsychosocial model, no one would suggest that these phenomena are “psychosomatic.” Perhaps it is suggestive that multiple sclerosis was earlier thought to be a psychosomatic illness until its organic basis was identified. See BERNE, *supra* note 11, at 92; White, *supra* note 31, at 626.

147. Part of the reason for disparagement of psychosomatic pain may be that no one has developed a plausible psycho-physiological explanation for how mental distress produce the same or equivalent physiological events like those known to cause “real” pain. Psychosomatic pain theories largely assume the existence of some pathogenesis by which mental distress results in medical pain. See *infra* text accompanying notes 275–77.

148. A consensus panel of clinicians who have treated more than 20,000 chronic pain patients recently reported that analgesics are the most favored prescription for chronic pain syndrome. See Jain et al., *supra* note 15, at 30. The wisdom of this treatment is called into question if chronic pain is a

favor of mental health therapies and patient self-help.<sup>149</sup> A final consequence is that those who believe pain is psychogenic minimize evidence of the biological causes of pain,<sup>150</sup> and discourage the continuation of biological research.<sup>151</sup> Much is at stake, then, if chronic pain is conceived as psychogenic.

### *B. Mental Disorders in Chronic Pain Sufferers*

The concepts of psychogenic or somatized pain appear to have no uniform meaning in the literature. The term “somatization,” for example, has been used in at least seven different ways.<sup>152</sup> The only common element in these varying uses appears to be that chronic pain has no physiological cause—the key diagnostic element, causation, is determined by default. This is an unusual approach to the classification of a mental disorder, because most mental disorders are defined by clinical symptoms rather than their cause or lack thereof.<sup>153</sup> For this reason, the phenomenon of psychosomatic pain as used in the critical literature is difficult to specify, validate, or falsify.

If psychogenesis and somatization are not to be treated as mere literary devices, they must be understood by using accepted notions of mental disorder. For better or worse, we are left with the diagnoses of mental disorder found in the DSM-IV-TR. Although these diagnoses have been criticized as the product of prevailing professional opinion rather than research,<sup>154</sup> their professional acceptance provides a modicum of scientific validity under the prevailing *Daubert* standard.<sup>155</sup>

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psychosomatic illness. See also Giesecke et al., *supra* note 143, at 1582 (discussing distinct effects of antidepressants and analgesics on pain, independent of mood effects).

149. For example, the DSM-IV-TR prescribes as treatment for “pain disorder” such things as: patient “acknowledgement of pain,” “psychological adaptation to chronic illness,” and “not allowing the pain to become the determining factor in his or her lifestyle.” See DSM-IV-TR, *supra* note 35, at 502. The theme of patient responsibility seems prominent in this prescription.

150. See, e.g., Hadler & Ehrlich, *supra* note 95, at 1030 (dismissing evidence of the biological bases for fibromyalgia as “quantitatively trivial, irreproducible, or nonspecific”).

151. See, e.g., MALLESON, *supra* note 28, at 29 (criticizing the “immense amount of medical literature . . . devoted to ingenious attempts” to identify physiological causes of chronic pain).

152. See Merskey, *Psychiatric Diagnosis*, *supra* note 45, at 101.

153. See Kendell & Jablensky, *supra* note 10, at 8 (noting that most psychiatric disorders are “still defined by their clinical syndromes because their etiology is still largely unknown”).

154. See generally Mary L. Malik & Larry E. Beutler, *The Emergence of Dissatisfaction with the DSM*, in *RETHINKING THE DSM 3*, 7 (Larry E. Beutler & Mary L. Malik eds., 2002) (discussing concerns that DSM diagnostic categories reflect “the influence of consensus seeking and political agenda” as much as research).

155. See *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594 (1993) (stressing that general acceptance remains an important factor in determining the reliability of expert testimony). Notably, DSM-IV-TR classifications correspond with those found in both the INTERNATIONAL CLASSIFICATION

Equally important, DSM-IV-TR diagnoses provide standard definitions that permit more reliable testing of the suspected phenomenon.

The most pertinent evidence on somatization is found in the somatoform disorders, which trace back to early theories of somatization. The term “somatization” was coined by an early twentieth century psychoanalyst, Wilhelm Stekel, a member of Sigmund Freud’s early circle.<sup>156</sup> Stekel’s concept of somatization derived from Freud’s concept of conversion, which proposed that repressed psychological conflict might be “converted” into physical symptoms.<sup>157</sup> Eventually somatization came to be understood by many professionals as a “tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and seek medical help for them.”<sup>158</sup>

When “somatoform disorders” were first introduced into the DSM-III in 1980, a distinct classification was recognized for “psychogenic pain disorder.”<sup>159</sup> This classification reflected its psychoanalytic roots by referring to the diagnostic importance of identifying a “psychological conflict or need” related to the development of pain.<sup>160</sup> The new classification also called attention to how pain might be “enabling the individual to avoid some activity that is noxious.”<sup>161</sup> This classification served as the “equivalent for pain of conversion hysteria, *i.e.*, a symptom that serves a purpose, solves a problem, is not based upon physical illness and is not based upon any other standard psychiatric disorder.”<sup>162</sup>

This earlier DSM classification of pain disorder proved unsatisfactory over time for reasons pertinent to current use of somatization theory. First, pain disorder was “being used too frequently” by diagnosing clinicians.<sup>163</sup> Second, critics began questioning whether behaviors like

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OF DISEASE AND RELATED HEALTH PROBLEMS (ICD) and the INT’L ASS’N FOR THE STUDY OF PAIN, *supra* note 19.

156. See SHORTER, *supra* note 43, at 259.

157. See Harold Merskey, *The History of Pain and Hysteria*, 8 NEUROREHABILITATION 157, 159 (1997); Merskey, *Psychiatric Diagnosis*, *supra* note 45, at 101; SHORTER, *supra* note 43, at 260. The DSM-IV-TR still contains vestiges of its psychoanalytic origins. It retains within the somatoform disorders a diagnosis called “conversion disorder.” This disorder involves classic “conversion” symptoms including paralysis and blindness. See DSM-IV-TR, *supra* note 35, at 492–93. Conversion disorder has been described as the “crown jewel” of the somatoform disorders. See John F. Kihlstrom, *To Honor Kraepelin . . . : From Symptoms to Pathology in the Diagnosis of Mental Illness*, in RETHINKING THE DSM, *supra* note 154, at 279, 296.

158. See Merskey, *Psychiatric Diagnosis*, *supra* note 45, at 101 (2000).

159. See Merskey, *supra* note 1, at 69.

160. See *id.*

161. See *id.*

162. See Merskey, *Psychiatric Diagnosis*, *supra* note 45, at 101.

163. See Merskey, *supra* note 1, at 69.

seeking medical help (a definitional element of modern “somatization”) might not be a normal consequence of chronic illness rather than a sign of psychological illness. Third, some physicians began to take seriously biological evidence suggesting that chronic pain resulted from biological abnormalities in the central nervous system rather than mental distress.<sup>164</sup>

Eventually, Merskey, acting on behalf of the International Association for the Study of Pain, persuaded the American Psychiatric Association to revise its classification for somatoform pain.<sup>165</sup> The new pain classification was called “pain disorder” and eliminated any direct reference to “psychogenic” or “somatoform.” The new classification also eliminated earlier references to psychological “conflict” or “avoidance.” This was consistent with abundant research rejecting the central tenet of psychoanalytic explanations for chronic pain—“that emotional conflict gives rise to bodily pain.”<sup>166</sup> The new criteria for “pain disorder” clarified that it should only be applied when the patient’s pain is not attributable to some other disorder (like depression or anxiety), no medical explanation for the patient’s symptoms exists, and psychological factors are specifically identified as having an “important role” in the patient’s pain.<sup>167</sup> As Merskey observed, the condition now known as pain disorder was to be “diagnosed very rarely.”<sup>168</sup>

Contemporary proponents of somatization have made extravagant claims that equate fibromyalgia with somatoform disorders in the DSM-IV-TR, or allege a strong association between the diagnoses.<sup>169</sup> Yet, scarce evidence indicates that the somatoform disorders are present in a significant percentage of chronic pain sufferers.<sup>170</sup> For example, an

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164. *See id.*

165. *See id.* at 70. Merskey also served as formal adviser to the DSM-IV-TR for somatoform disorders. *See* DSM-IV-TR, *supra* note 35, at 931.

166. *See* Ann Gamsa, *The Role of Psychological Factors in Chronic Pain. I. A Half Century of Study*, 57 PAIN 5, 8 (1994).

167. *See* DSM-IV-TR, *supra* note 35, at 503.

168. *See* Merskey, *supra* note 1, at 71; *see also* Merskey, Editorial, *supra* note 45, at 4 (“[P]ain disorder should be rare.”).

169. *See, e.g.*, John B. Winfield, *Psychological Determinants of Fibromyalgia and Related Syndromes*, 4 CURRENT REV. PAIN 276, 281 (2000) (arguing that fibromyalgia is “a somatoform disorder” but “not entirely”); Barsky & Borus, *supra* note 34, at 913 (arguing that patients with fibromyalgia have “elevated rates of psychiatric disorders, particularly . . . somatoform disorders”).

170. The core somatoform disorder, somewhat confusingly styled “somatization disorder,” requires the presence of “pseudoneurological” symptoms like paralysis or vision problems, as well as the onset of illness by the age of 30. *See* DSM-IV-TR, *supra* note 35, at 486. It is generally agreed that most fibromyalgia sufferers do not satisfy these requirements. *See, e.g.*, BERNE, *supra* note 11, at 110 (noting that fibromyalgia patients cannot be classified as suffering from somatization disorder because of “symptom prevalence, age, suddenness of onset, lack of prevalent personality disorders, and exercise and alcohol intolerance”); WALLACE & WALLACE, *supra* note 5, at 15, 120–21 (reporting that



earlier study investigating somatoform disorders in a small group of clinical patients found that 23% had some form of somatoform disorder.<sup>171</sup> A larger population study that followed, however, found only 3% of persons with widespread chronic pain suffered from a somatoform disorder.<sup>172</sup> As the later researchers observed, the higher rate of somatoform disorders reported in the earlier study likely reflected the increased incidence of mental illness found in clinical patients generally at that time, as well as the earlier study's use of "less stringent criteria for diagnosis . . . of somatoform disorders."<sup>173</sup>

The most recent study examining the relationship between chronic pain and somatization used an indirect measure of somatization more indulgent than formal DSM-IV-TR diagnostic criteria, and found that approximately 21% of the study group with higher "somatic symptom scores" later developed chronic pain. In contrast some 10% of those having lower scores later developed chronic pain.<sup>174</sup> But more noteworthy was the finding that most chronic pain developed in persons *without* strong signs of somatization. In particular, 89% of persons who developed chronic pain had *lower* somatic symptom scores. On the other hand, almost 80% of those with higher scores failed to develop chronic pain.<sup>175</sup> These findings are consistent with those of an earlier population study using the same proxy for somatization. The early study found that the great majority of persons experiencing chronic widespread pain, whether currently seeking medical treatment or not, failed to meet the threshold for somatization.<sup>176</sup>

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pseudoneurologic symptoms are "clearly not present part of fibromyalgia" and that most patients develop fibromyalgia in their 30s and 40s); Jain et al., *supra* note 15, at 46 (same).

171. See Leslie A. Aaron et al., *Psychiatric Diagnoses in Patients With Fibromyalgia are Related to Health Care-Seeking Behavior Rather Than to Illness*, 39 ARTHRITIS & RHEUMATISM 436, 441 (1996) (study based on sample of sixty-four patients).

172. See Benjamin et al., *supra* note 25, at 565.

173. See *id.* at 566. A widely recognized problem with measures of somatization, like scores for hysteria and hypochondriasis found in the Minnesota Multiphasic Personality Inventory (MMPI), is that they measure this phenomenon based on the presence of unexplained medical symptoms like bodily pain and fatigue. This begs the question of whether the unexplained symptoms result from mental or physical illness. See, e.g., Harold Merskey, *Pain and Psychological Medicine*, in TEXTBOOK OF PAIN 929 (4th ed. 1999).

174. See John McBeth et al., *Features of Somatization Predict the Onset of Chronic Widespread Pain*, 44 ARTHRITIS & RHEUMATISM 940, 944 (2001).

175. See *id.* This finding is not emphasized in the article, but can be derived from data reported at 944 tbl.5. "Low" somatic symptom scores included those where the number of somatic features were *not* a reliable predictor of somatoform disorder in general populations. See *id.* at 941 (discussing "sensitivity" of "high" somatic scores). The use of somatic symptom scores as a proxy for somatization is unsatisfactory for a reason we have seen when looking at DSM-IV-TR criteria. Measures of somatization assume that physical symptoms reported by patients have no "medical" explanation and instead are the result of mental distress.

176. See Gary J. Macfarlane et al., *Chronic Widespread Pain in the Community: The Influence of*

Current research, then, fails to show that somatoform disorders fully account for all chronic pain, even when less formal diagnostic criteria are employed.<sup>177</sup> Nothing indicates that somatoform disorders are present in a majority of chronic pain sufferers, and there is certainly no evidence to suggest that such a disorder caused their chronic pain.<sup>178</sup> As for the specific somatoform disorder known as “pain disorder,” there is no reliable data indicating its prevalence.<sup>179</sup>

Merskey’s skepticism about the diagnosis of somatoform disorders in chronic pain sufferers is especially instructive. In addition to playing a significant role in the evolution of the DSM-IV-TR’s classification for pain disorder, Merskey was a proponent of “hysterical” chronic pain earlier in his career.<sup>180</sup> Merskey now rejects the validity of somatization as an explanation for the chronic pain of fibromyalgia. As he recently commented, “the concept of Somatization is unsatisfactory because of a multiplicity of meanings . . . as was its partial predecessor, Hysteria.”<sup>181</sup> Regarding pain disorder in the DSM-IV-TR, Merskey now believes there are “precious few cases in which the diagnosis can be made correctly.”<sup>182</sup>

Advocates of psychogenic pain have also attempted to explain

*Psychological Symptoms and Mental Disorder on Healthcare Seeking Behavior*, 26 J. RHEUMATOLOGY 413, 416 (1999).

177. Recent research has also called into question one tenet of somatization—that patients have hidden psychological conflicts inaccessible to therapists, which must be expressed somatically. See Kirmayer et al., *supra* note 42, at 669. The authors found that patients with unexplained somatic symptoms “provided clear cues . . . about the presence of important psychosocial issues.” *Id.*

178. Recent research indicates that clinicians are far more likely to diagnose a somatoform disorder by using the most unspecific diagnosis, “undifferentiated somatoform disorder.” See Margot W.M. De Waal et al., *Somatoform Disorders in General Practice*, 184 BRITISH J. PSYCHIATRY 470, 472 (2004) (reporting that 80% of somatoform disorders diagnosed in one clinical group were “undifferentiated”). The essential features of “undifferentiated” somatoform disorder are a persistent physical symptom, lacking complete medical explanation, resulting in long-term stress or impairment. See DSM-IV-TR, *supra* note 35, at 492. The fact that “undifferentiated” somatoform disorder has such diagnostic prominence has prompted some researchers to question whether the concept of somatoform disorder retains clinical utility. See Michael Sharpe & Richard Mayou, *Somatoform Disorders: A Help or Hindrance to Good Patient Care?*, 184 BRITISH J. PSYCHIATRY 465 (2004) (stating that “neither the overall category of somatoform disorder nor its sub-categories satisfy the accepted criteria for validity or reliability”). Sharpe and Mayou argue that a diagnosis of “undifferentiated” somatoform disorder may be little more than a “relabeling” of the patient’s symptoms. *Id.*

179. The DSM-IV-TR concedes this point. See DSM-IV-TR, *supra* note 35, at 501 (“[T]he prevalence of Pain Disorder is unclear.”).

180. See Merskey, *supra* note 157, at 161.

181. Harold Merskey, *Intra-nasal Kestamine for Somatization?*, 110 PAIN 765 (2004).

182. *Id.* During the past decade, the diagnosis for “pain disorder” has been sharply criticized as being over-inclusive, subjective, and ultimately nothing more than a “diagnosis of exclusion” remaining after putative medical causes of pain have been ruled out by a clinician. See, e.g., D.A. Fishbain, *Where Have Two DSM Revisions Taken Us for the Diagnosis of Pain Disorder in Chronic Pain Patients?*, 153 AM. J. PSYCHIATRY 137 (1996).

chronic pain by focusing on DSM-IV-TR disorders not included among the somatoform disorders.<sup>183</sup> The disorder most frequently invoked is depression.<sup>184</sup> Depression can produce pain<sup>185</sup> and several studies have shown a significantly higher rate of depression among persons suffering from chronic pain syndromes like fibromyalgia.<sup>186</sup> Depression also shares a characteristic of chronic pain syndromes—it occurs more frequently in women than men.<sup>187</sup>

Medical research, however, fails to support the claim that chronic pain, like that associated with fibromyalgia, is primarily the product of depression. First, most fibromyalgia sufferers do not have diagnosable depression.<sup>188</sup> Second, among those who suffer from both depression and fibromyalgia syndrome, many likely have become depressed as a *consequence* of their chronic pain.<sup>189</sup> A significant amount of psychological disturbance often accompanies chronic medical illness, but to assume that the illness is a *product* of psychological distress is a mistake.<sup>190</sup> Third, several studies confirm that the elevated levels of

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183. As mentioned earlier, *see supra* note 166 and accompanying text, somatoform disorders specifically exclude those whose somatic symptoms are best explained using some other DSM diagnosis. Strictly speaking, then, chronic pain linked to disorders like depression and anxiety are not examples of somatization.

184. The principal depression disorder is called “major depressive disorder.” *See* DSM-IV-TR, *supra* note 35, at 369.

185. *See* INT’L ASS’N FOR THE STUDY OF PAIN, *supra* note 19, at 56.

186. *See* WALLACE & WALLACE, *supra* note 5, at 118–19.

187. *See* DSM-IV-TR, *supra* note 35, at 372.

188. It is generally agreed that the large majority of persons with fibromyalgia do not suffer from clinical depression. *See* WALLACE & WALLACE, *supra* note 5, at 118; Jain et al., *supra* note 15, at 45 (“The all too common assertion that FMS symptoms are the manifestation of underlying depression or other psychological difficulties is not supported by available data.”); Goldenberg, *supra* note 4, at 779 (observing that “most patients with FM are not depressed” and that studies find no greater prevalence of major depression than is found in those “with rheumatoid arthritis and in healthy controls”). A recent national population sample identified depression in 20% of persons reporting chronic pain. *See* Lachlan A. McWilliams et al., *Mood and Anxiety Disorders Associated With Chronic Pain: An Examination in a Nationally Representative Sample*, 106 PAIN 127 (2003).

189. *See* D.A. Fishbain et al., *Chronic Pain-Associated Depression: Antecedent or Consequence of Chronic Pain? A Review*, 13 CLINICAL J. PAIN 116 (1997) (reporting that most research supports the view that depression is caused by chronic pain). As emphasized in INT’L ASS’N FOR THE STUDY OF PAIN, *supra* note 19, at 56, “it is important not to confuse the situation of depression causing pain as a secondary phenomenon with depression which commonly occurs when chronic pain arising for physical reasons is troublesome.”

190. *See, e.g.,* Akiko Okifuji et al., *Evaluation of the Relationship Between Depression and Fibromyalgia Syndrome: Why Aren’t All Patients Depressed?*, 27 J. RHEUMATOLOGY 212 (2000) (noting higher prevalence of depression patients with arthritis and other rheumatological diseases); Jain et al., *supra* note 15, at 45 (“It is reasonable to conclude that the depression seen in a subset of FMS patients is more likely secondary, reactive depression and not the cause of the painful syndrome.”); WALLACE & WALLACE, *supra* note 5, at 118 (noting that depression is associated with high rates of musculoskeletal pain and may precede illness); PRIMER ON THE RHEUMATIC DISEASES, *supra* note 32, at 563 (observing that painful conditions like rheumatoid arthritis can lead to depression).

psychological distress found in clinical patients with chronic pain are often peculiar to those who specifically persist in *seeking* specialized health care.<sup>191</sup> Finally, recent brain imaging studies indicate that the presence of depression in pain patients does not affect the brain's processing of the sensory aspects of pain, and that in these patients "amplification of the sensory dimension of pain . . . is totally independent of mood or emotion."<sup>192</sup>

Anxiety disorders have also been associated with chronic pain.<sup>193</sup> As with depression, however, anxiety disorders do not provide an adequate explanation of chronic pain. Notably, studies of chronic pain populations do not reveal a greatly increased incidence of anxiety disorders.<sup>194</sup> Most fibromyalgia sufferers do not suffer from a diagnosable anxiety disorder.<sup>195</sup> Further, no evidence indicates that abnormal stress is the primary cause of chronic pain.<sup>196</sup> As with depression, anxiety and stress can often be the *consequence* of a pain-ridden life.

Two recent studies conducted by Karen Raphael have undermined the claim that chronic pain originates in psychological stress. In the first study, Raphael examined whether unexplained pain could be traced to childhood abuse.<sup>197</sup> This hypothesis has special relevance to somatization theory, because the concept of somatization derives historically from the psychoanalytic theory that somatic symptoms result

191. See, e.g., Aaron et al., *supra* note 171 (finding that psychological disorders like depression are correlated with those who seek health care, rather than with sufferers of fibromyalgia per se); Bradley & Alarcón, *supra* note 23, at 152 ("Psychological factors . . . may contribute to the decision of individuals to seek specialized health care for their fibromyalgia symptoms, but not to the etiopathogenesis of fibromyalgia."); WALLACE & WALLACE, *supra* note 5, at 118 (observing that the population of patients seen at medical centers, upon whom many studies are based, "tend to be more symptomatic and have not responded to interventions by community physicians"); McWhinney et al., *supra* note 41, at 749 ("Those who go to physicians regularly are likely to be more anxious than those who cope with their symptoms in other ways.").

192. See Giesecke et al., *supra* note 143, at 1582.

193. See DSM-IV-TR, *supra* note 35, at 500.

194. See, e.g., Benjamin et al., *supra* note 25, at 565 (reporting 7% more anxiety disorders in chronic pain sufferers); Hagit Cohen et al., *Prevalence of Post-Traumatic Stress Disorder in Fibromyalgia Patients: Overlapping Syndromes or Post-Traumatic Fibromyalgia Syndrome?*, 32 SEMINARS IN ARTHRITIS & RHEUMATISM 38, 39 (2002).

195. See Cohen et al., *supra* note 194, at 39.

196. See, e.g., Mika Kivimäki et al., *Work Stress and Incidence of Newly Diagnosed Fibromyalgia*, 57 J. PSYCHOSOMATIC RES. 417 (2004) ("No prospective evidence is available to confirm that stress actually precedes the onset of fibromyalgia."). For a discussion of the considerable methodological problems in measuring the effects of stress, see Anthony J. Cleare, *Stress and Fibromyalgia—What is the Link?*, 57 J. PSYCHOSOMATIC RES. 423 (2004).

197. Karen G. Raphael et al., *Childhood Victimization and Pain in Adulthood: A Prospective Investigation*, 92 PAIN 283 (2001).

from repressed psychic conflict.<sup>198</sup> A link between childhood abuse and chronic pain would tend to affirm that pain is somatized distress carried over from childhood—and have obvious resonance with psychoanalytic theories of repression. However, Raphael's prospective study found no link between childhood victimization and unexplained pain, thus undermining one of the more favored premises of somatization theory.<sup>199</sup>

In a second prospective study, Raphael examined the alleged increase in chronic pain thought to have occurred among those suffering stress and depression related to the September 11, 2001 terrorist attacks on the World Trade Center (9/11).<sup>200</sup> Raphael hypothesized that persons in proximity to these occurrences provided an opportune experimental population in which to study the effects of stress on chronic pain. Yet, Raphael found no significant increase in fibromyalgia-like symptoms resulting from post-9/11 stress and depression. This finding, Raphael observed, “raises doubts about the extent to which fibromyalgia-like symptoms are triggered by stress and depression.”<sup>201</sup>

In sum, existing research does not support the view that chronic pain syndromes are primarily the product of any recognized mental disorder. Attempts to explain chronic pain through somatization, depression, anxiety, and stress have been disappointing.<sup>202</sup> Mental disorders may explain chronic pain in some patients, and psychosocial factors clearly can play a role in the pain experience.<sup>203</sup> But, there is little “serious evidence” indicating that chronic pain syndromes are *primarily* the

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198. *See id.* at 283–84.

199. *See id.* at 292 (“Early childhood victimization and medically unexplained pain are not intrinsically linked. The widespread belief in this link has contributed to the view of unexplained pain as ‘psychogenic.’ However, our findings reinforce the notion that a medically unexplained pain complaint is not necessarily a psychogenic one. This revised view may help to reduce the stigmatization of patients . . . who are led to believe that their pain problems are caused by psychological rather than physical factors.”).

200. Karen G. Raphael et al., *A Community-Based Survey of Fibromyalgia-like Pain Complaints Following the World Trade Center Terrorist Attacks*, 100 PAIN 131 (2002).

201. *Id.* at 137. In addition, a recent survey of the efficacy of “psychosocial treatments” in fibromyalgia patients, including stress-reduction treatments, failed to find any significant, lasting effects. *See* Lesley A. Allen et al., *Psychosocial Treatments for Multiple Unexplained Physical Symptoms: A Review of the Literature*, 64 PSYCHOSOMATIC MED. 939, 942–45 (2002).

202. Researchers recently studied the role of “catastrophizing” among chronic pain patients, an alternative to somatization that traces pain to fear, avoidance, and hypervigilance. *See* Rudy Severeijns et al., *Pain Catastrophizing and Consequences of Musculoskeletal Pain: A Prospective Study in the Dutch Community*, 6 J. PAIN 125 (2004). They found a “feeble” relationship between measures of catastrophizing and pain symptoms. *Id.* at 129.

203. *See, e.g.,* Okifuji et al., *supra* note 190, at 217 (“Depression in FM appears to develop independently of the cardinal features of FM . . . but is related to FM patients’ cognitive appraisal of the effect of pain on their lives.”).

product of mental distress.<sup>204</sup>

### *C. The Persistence of Psychogenic Explanations for Chronic Pain*

Despite the absence of empirical evidence demonstrating that enigmatic chronic pain results from mental illness, psychogenic explanations have not waned in recent years. Several explanations for the persistence of psychogenic theory are likely.

First, psychogenesis had long been a default explanation for enigmatic medical symptoms. Medical professionals, in particular, have a historical proclivity for reverting to psychogenic explanations when biological ones are not available.<sup>205</sup> The list of recognized diseases once dismissed by the profession as psychogenic (or imagined) phenomena is extensive and includes the following: rheumatoid arthritis,<sup>206</sup> multiple sclerosis, polio, diabetes, ulcers, lupus, Lyme disease, AIDS, and schizophrenia.<sup>207</sup> As one researcher comments, “a diagnosis based on psychological theory may offer the researcher or clinician the security of scientific moorings even when conceptualizations are vague, methods of observation questionable, and the data have been shaped to fit.”<sup>208</sup>

This tendency to fall back on psychogenic explanations is particularly

204. See Merskey, *supra* note 141, at 241; see also Jain et al., *supra* note 15, at 45 (“The all too common assertion that FMS symptoms are the manifestation of underlying depression or other psychological difficulties is not supported by available data.”).

205. See, e.g., Jack W. Snyder et al., *Love, Injury and Causation on Trial: The Phenomenon of “Multiple Chemical Sensitivities,”* 2 WIDENER L. SYMP. J. 97, 103 (1997) (“In the absence of objective, verifiable, and measurable signs of deviation from biologic normality, the subjective reporting of chronic symptoms is typically classified as a psychogenic problem in orthodox western medical practice.”).

206. See Edward A. Walker et al., *Psychosocial Factors in Fibromyalgia Compared with Rheumatoid Arthritis: I. Psychiatric Diagnoses and Functional Disability*, 59 J. PSYCHOSOMATIC MED. 565 (1997) (“For decades rheumatoid arthritis was considered one of the ‘classic’ psychosomatic diseases.”).

207. See, e.g., BERNE, *supra* note 11, at 92 (multiple sclerosis, rheumatoid arthritis, polio, AIDS, ulcers, and diabetes); White, *supra* note 31, at 626 (multiple sclerosis and hypothyroidism); Thomas L. English, *Letter to the Editor*, 132 ANNALS OF INTERNAL MED. 329 (2000) (lupus, multiple sclerosis, AIDS, and Lyme disease); see also Harold Merskey, *Physical and Psychological Considerations in the Classification of Fibromyalgia*, 19 J. RHEUMATOLOGY SUPP. 72, 75 (1989) (noting medical conditions like cerebral tumors and reflex sympathetic dystrophy were once attributed to hysteria); Roger K. Pitman & Scott P. Orr, *Psychophysiological Testing for Post-Traumatic Stress Disorder: Forensic Psychiatric Application*, 21 BULL. AM. ACAD. PSYCHIATRY & L. 37, 40 (1993) (noting how advances of biological understanding of schizophrenia and affective disorders refuted belief that these mental illnesses were a myth); John J. Ratey, *Shadow Syndromes: People With Mild Forms of Serious Disorders*, [http://www.addresources.org/article\\_shadow\\_syndromes\\_ratey.php](http://www.addresources.org/article_shadow_syndromes_ratey.php) (observing that schizophrenia and manic-depression were thought to be the product of bad parenting until their biological origins were discovered).

208. See Gamsa, *supra* note 118, at 23–24. The same view is expressed in Kirmayer et al., *supra* note 42, at 664.

conspicuous in the case of women's pain, which had long been caricatured in medicine.<sup>209</sup> When pain cannot be readily explained as the direct consequence of tissue damage, physicians treating women are apt to make a "leap to the head."<sup>210</sup> Even when pain symptoms have an identifiable organic basis, "the therapeutic literature is characterized by an unscientific recourse to psychogenesis and a correspondingly inadequate, even derisory, approach to their management."<sup>211</sup> As has been observed, "[a] large part of the problem in treating chronic pain is the common belief that patients are exaggerating and embellishing a trivial pain problem and if they were made of the 'right stuff' they would improve."<sup>212</sup> Psychosomatic explanations of pain not only affirm stereotypical views of the hysteria-ridden female popularized by cultural historians like Shorter and Showalter,<sup>213</sup> they give comfort to physicians unable to understand or treat intractable pain.

A second explanation for the persistence of psychosomatic pain explanations is that clinicians frequently observe a great deal of psychological distress in chronic pain patients. This distress can be misinterpreted as the *source* of medically unexplained pain. However, this diagnosis overlooks the fact that chronic medical illness often *causes* a significant amount of psychological disturbance.<sup>214</sup> Further, several studies confirm that the higher levels of psychological distress found in clinical patients with chronic pain are often *peculiar* to those who persist in seeking health care for an essentially incurable condition.<sup>215</sup> For these reasons, clinical impressions of pain patients are

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209. See Unruh, *supra* note 24, at 158. See also Diane E. Hoffman & Anita J. Tarzian, *The Girl Who Cried Pain: A Bias Against Women in the Treatment of Pain*, 29 J.L. MED. & ETHICS 13, 20 (2001) (emphasizing the importance of patient credibility in assessments of pain symptoms, and the prevalence of cultural images of women as being emotional, hypersensitive, and overly reactive).

210. See Unruh, *supra* note 24, at 158.

211. K. Jean Lennane & R. John Lennane, *Alleged Psychogenic Disorders in Women—A Possible Manifestation of Sexual Prejudice*, 288 NEW ENG. J. MED. 288, 288 (1973).

212. See Robert M. Bennett, *Emerging Concepts in the Neurobiology of Chronic Pain: Evidence of Abnormal Sensory Processing in Fibromyalgia*, 74 MAYO CLINIC PROC. 385, 394 (1999).

213. Researchers have noted the predominance of female patients in medical literature depicting psychogenic pain. See Hoffman & Tarzian, *supra* note 209, at 20.

214. See, e.g., Jain et al., *supra* note 15, at 45 ("It is reasonable to conclude that the depression seen in a subset of FMS patients is more likely secondary, reactive depression and not the cause of the painful syndrome."); WALLACE & WALLACE, *supra* note 5, at 118 (noting that depression is associated with high rates of musculoskeletal pain and may precede illness); PRIMER ON THE RHEUMATIC DISEASES, *supra* note 32, at 563 (observing that painful conditions like rheumatoid arthritis can lead to depression). This is illustrated by a recent population study that found an increased incidence of mood and anxiety disorders among persons suffering from chronic pain related to recognized medical conditions like arthritis, bone or joint disease. See McWilliams et al., *supra* note 188.

215. See, e.g., McWilliams et al., *supra* note 188, at 128 ("The vast majority of studies in this area have utilized highly select treatment-seeking samples . . . and as a result, the prevalence rates and associations found in these studies were likely biased."); Bradley & Alarcón, *supra* note 23, at 152

often highly skewed.

A final reason for the persistence of the somatization theory illustrates how undisciplined the theory has become in popular literature. Among many critics, somatization has become code for malingering and fraud. Malleson's *Whiplash and Other Useful Illnesses* is just the latest example of how critics of enigmatic symptoms tend to impugn the authenticity of illness when it becomes entangled with compensation issues.<sup>216</sup> This line of criticism has produced colorful tort neologisms like "accident neurosis," "compensation hysteria," "greenback neurosis," "traumatic hysteria," "traumatic neurosis," and "secondary gain neurosis."<sup>217</sup>

The DSM-IV-TR poses an obvious problem for those who equate somatization with the intentional misrepresentation of symptoms. Somatoform disorders specifically *exclude* symptoms "intentionally feigned or produced."<sup>218</sup> The DSM-IV-TR does recognize a "condition" called malingering, which applies to persons who intentionally produce medical symptoms to secure some external gain like disability benefits.<sup>219</sup> Yet, virtually all researchers agree that malingering among chronic pain sufferers is not common, even though it is a popular theme among pain critics.<sup>220</sup> Perhaps the strongest refutation of malingering is the persistence of chronic pain in the absence of economic incentive.

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("Psychological factors . . . may contribute to the decision of individuals to seek specialized health care for their fibromyalgia symptoms, but not to the etiopathogenesis of fibromyalgia."); WALLACE & WALLACE, *supra* note 5, at 118 (observing that the population of patients seen at medical centers, upon whom many studies are based, "tend to be more symptomatic and have not responded to interventions by community physicians"); McWhinney et al., *supra* note 41, at 749 ("Those who go to physicians regularly are likely to be more anxious than those who cope with their symptoms in other ways.").

216. See, e.g., Harold Merskey & Robert W. Teasell, *The Disparagement of Pain: Social Influences on Medical Thinking*, 4 PAIN RES. & MGMT 259, 262-64.

217. See George Mendelson, "Compensation Neurosis" Revisited: Outcome Studies of the Effects of Litigation, 39 J. PSYCHOSOMATIC RES. 695, 695-96 (1995). Mendelson's article contains the classic refutation of assertions that personal injury claimants miraculously improve once legal compensation is secured.

218. See DSM-IV-TR, *supra* note 35, at 486.

219. Clinical malingering is defined as the "intentional production of false or grossly exaggerated physical or psychological symptoms motivated by external incentives such as . . . financial compensation." DSM-IV-TR, *supra* note 35, at 739.

220. The congressionally authorized Commission on the Evaluation of Pain, reporting in 1986, specifically found that malingering is not a significant problem. See Michael G. Ruppert, *Developments in Social Security Law*, 22 IND. L. REV. 406, 410 (1989). More recent study confirms this finding. See, e.g., Chris J. Main & Chris C. Spanswick, "Functional Overlay" and Illness Behaviour in Chronic Pain: Distress or Malingering? Conceptual Difficulties in Medico-Legal Assessment of Personal Injury Claims, 39 J. PSYCHOSOMATIC RES. 737, 740 (1995); Simon Wessely, *Liability for Psychiatric Illness*, 39 J. PSYCHOSOMATIC RES. 659, 664 (1995). As pointed out in INT'L ASS'N FOR THE STUDY OF PAIN, *supra* note 19, there is no internationally recognized illness classification for malingering, which "suggests that the final application of the label of malingering is a judicial (legal) process and not a medical one." *Id.* at 56.



The majority of fibromyalgia sufferers never seek disability benefits,<sup>221</sup> and among those who obtain some form of compensation, symptoms persist after legal action is resolved and the potential for secondary gain is dissipated.<sup>222</sup> Finding the prospect of secondary gain in some groups of chronic pain sufferers is especially difficult. For example, an estimated 15% of children and adolescents experience chronic pain.<sup>223</sup> Additionally, a recent study of the Amish, who do not accept disability benefits, revealed that more than 7% met the diagnostic criteria for fibromyalgia and approximately 3% had been diagnosed with the illness.<sup>224</sup>

Malleson, who recognizes the diagnostic incompatibility between somatoform disorders and malingering, characteristically dismisses it by questioning the validity of the distinction. Discussing whiplash syndrome—which he views as the precursor of modern fibromyalgia—Malleson observes that “perhaps the essential difference between malingering, factitious disorder,<sup>225</sup> and somatoform disorders depends upon how well Nature has done her work—the effectiveness with which the victim of whiplash can deceive himself and therefore others.”<sup>226</sup> Malleson’s effort to salvage his theory of somatization illustrates an irony in much criticism of chronic pain: Extravagantly unscientific views of somatization are invoked to challenge what critics see as soft medical diagnoses like chronic pain syndrome. Consider the recent reworking of the concept of somatization by one group of medical researchers:

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221. See, e.g., Wolfe & Potter, *supra* note 95, at 369 (reporting that 25% of fibromyalgia patients have received some type of disability); WALLACE & WALLACE, *supra* note 5, at 204 (same); BERNE, *supra* note 11, at 46 (reporting disability rates of 15%–30%).

222. See, e.g., WALLACE & WALLACE, *supra* note 5, at 205 (“Several studies have shown that over 90 percent of the time, fibromyalgia does not stop after litigation is settled.”). One meta-analysis of the chronic pain literature found that a small 6% variance in the pain experience could be attributed to “compensation” status. See M.L. Rohling et al., *Money Matters: A Meta-Analytic Review of the Association Between Financial Compensation and the Experience and Treatment of Chronic Pain*, 14 HEALTH PSYCHOLOGY 537 (1995).

223. See, e.g., Peter N. Malleson et al., *Chronic Musculoskeletal and Other Idiopathic Pain Syndromes*, 84 ARCHIVES DISEASE CHILDHOOD 189, 189 (2001) (“Chronic or recurrent pain in children and adolescents, for which no specific cause can be found, is very common with a point prevalence of at least 15%.”); Jain et al., *supra* note 15, at 5 (reporting that 6% of school children in randomly selected population met the criteria for fibromyalgia).

224. See Kevin P. White & John Thompson, *Fibromyalgia Syndrome in an Amish Community: A Controlled Study to Determine Disease and Symptom Prevalence*, 30 J. RHEUMATOLOGY 1835, 1836–37 (2003).

225. Factitious disorder, like malingering, involves the intentional production of symptoms to satisfy some need. In factitious disorders the person seeks to satisfy a psychological need that is not usually obvious; in the case of malingering, the person acts with some external incentive like obtaining an economic benefit. See DSM-IV-TR, *supra* note 35, at 513, 739.

226. See MALLESON, *supra* note 28, at 301.

The process of somatization has been described as the expression of personal and social distress through physical symptoms, often accompanied by patterns of illness behavior such as increased medical help-seeking for those symptoms. As such, it is viewed as a process of illness behavior rather than a distinct psychiatric diagnostic criterion such as somatization disorder. Somatization disorder may only represent the “tip of the iceberg” of persons who somatize.<sup>227</sup>

So defined, somatization is a protean concept with power to delegitimize many medical symptoms, while retaining the virtues of being untestable and seemingly irrefutable.<sup>228</sup>

It is increasingly apparent that the legitimacy of chronic pain will only be vouchsafed with evidence of its organic nature and causes. As discussed in the last section, an emerging consensus now believes that enigmatic chronic pain derives from organic processes, although not the processes traditionally associated with acute pain. The paradox of this research is that it has helped us to “understand better than ever the physiological basis of chronic pain at a time when its denial is being institutionalized.”<sup>229</sup>

## V. THE EMERGENT BIOLOGY OF CHRONIC PAIN

It’s widely accepted at least in the pain community that chronic pain has no function at all and is in fact a pathology. It’s its own disease.<sup>230</sup>

Well into the twentieth century, pain was believed to be the body’s inevitable sensory response to tissue damage.<sup>231</sup> Pain developed through a process called “nociception,” in which nerve receptors in parts of the body like the skin or bones—the peripheral nervous system—sense damage to tissue and relay a message of pain to the spinal cord

227. See McBeth et al., *supra* note 174, at 941. As discussed earlier, McBeth and his co-researchers not only employ a highly elastic concept of somatizing, they offer a highly strained interpretation of research findings to conclude that somatization “predict[s] the onset of chronic widespread pain” in the face of their finding that 89% of persons who developed this form of pain had low somatization scores. See *supra* notes 174–175 and accompanying text.

228. It is even arguable that many assertions of somatization are not scientific propositions at all, insofar as the Supreme Court has emphasized that the scientific status of theory depends on its “falsifiability.” See *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 593 (1993). In their work, *Judging Science: Scientific Knowledge and the Federal Courts*, Kenneth R. Foster and Peter W. Huber give the example of hysterical conversion—a prototype for modern somatization concepts—to illustrate “nonfalsifiable” diagnoses. See KENNETH R. FOSTER & PETER W. HUBER, *JUDGING SCIENCE: SCIENTIFIC KNOWLEDGE AND THE FEDERAL COURTS* 59 (1999).

229. Merskey & Teasell, *supra* note 216, at 266.

230. See Marx, *supra* note 17, at 326 (quoting Jeffry Mogil of McGill University).

231. See Gamsa, *supra* note 166, at 5, 6.

and brain—the central nervous system.<sup>232</sup> The paradigm for the process of nociception is acute pain, which “usually results from a recent . . . injury that has a known cause, a clearly perceived severity, and predictable prognosis.”<sup>233</sup>

Acute pain remains the working model for most doctors, who receive little instruction in chronic pain during their medical training.<sup>234</sup> As a consequence, many share a belief that “chronic pain is simply a type of acute pain that has persisted too long. When such pain does not respond to the usual therapeutic regimens for acute pain, the patient may be treated pejoratively, viewed as a malingerer, labeled a somatizer, or totally ignored.”<sup>235</sup>

The prevailing model of nociceptive pain also assimilates well into traditional tort law concepts of causation. Tort causation has long emphasized mechanistic causal chains<sup>236</sup> and common-sense, linear relationships between the severity of trauma and the severity of pain.<sup>237</sup> This is illustrated by the classic slip-and-fall case, where courts understandably show a lenient attitude toward scientific evidence of pain causation and may even completely dispense with the need for expert testimony.<sup>238</sup>

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232. See MAYO CLINIC ON CHRONIC PAIN, *supra* note 2, at 5–6.

233. See I. Jon Russell, *Neurotransmitters, Cytokines, Hormones, and the Immune System in Chronic Non-Neuropathic Pain*, in TEXTBOOK OF FIBROMYALGIA SYNDROME AND OTHER NON-NEUROPATHIC PAIN SYNDROMES 11 (Daniel J. Wallace & Daniel J. Clauw eds., forthcoming).

234. See, e.g., Leslie J. Crofford & Daniel J. Clauw, *Fibromyalgia: Where Are We a Decade After the American College of Rheumatology Classification Criteria Were Developed?*, 46 ARTHRITIS & RHEUMATISM 1136, 1137 (2002) (“Distress on the part of physicians is likely, because mechanisms underlying FM symptoms are poorly understood and are outside the realm of mechanisms traditionally studied by rheumatologists . . .”).

235. See Bennett, *supra* note 212, at 385.

236. See, e.g., Troyen A. Brennan, *Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous-Substance Litigation*, 73 CORNELL L. REV. 469, 483–501 (1988). As Brennan comments, “But for causation assumes the existence of causal chain analysis, depends on a mechanistic understanding of causation, and coincides with everyday, common sense notions of causation.” *Id.* at 486. See also Snyder et al., *supra* note 205, at 139–40 (“Proof of ‘factual causation[] usually involves proof of logical relationships between events linked in a deductive ‘causal chain.’”).

237. See Daniel V. Voiss, *Occupational Injury: Fact, Fantasy, or Fraud*, in NEUROLOGIC CLINIC 431, 435 (Michael I. Weintraub ed. 1995) (“The common wisdom and most legal jurisdictions define an injury as a sudden and tangible incident of a traumatic nature that produces a relatively immediate result and occurs from without . . .”) (citing Wash. Rev. Code § 51.08.100(1993)).

238. See, e.g., *Hamburger v. State Farm Mut. Auto. Ins. Co.*, 361 F.3d 875 (5th Cir. 2004) (expert testimony not required to prove neck pain immediately resulting from accident); *Dodge-Farrar v. Am. Cleaning Serv. Co.*, 54 P.3d 954, 957–58 (Idaho Ct. App. 2002) (holding that expert testimony not required where “alleged injuries are of a common nature, and arise from a readily identified cause). See generally Note, *Navigating Uncertainty: Gatekeeping in the Absence of Hard Science*, 113 HARV. L. REV. 1467, 1473 (2000) (observing that in slip-and-fall cases courts have “customarily . . . felt comfortable admitting causation testimony that lacks true scientific rigor but that is supported by a combination of ‘reasonable medical certainty,’ physician expertise, and adherence to standard diagnostic

Widespread chronic pain poses a problem under the prevailing medical and legal models of acute pain. Chronic pain usually develops over a period of months, has no apparent immediate cause, and involves no ongoing tissue damage by which to gauge its severity.<sup>239</sup> Chronic pain “outlives its original causes, worsens over time and appears to take on a puzzling life of its own.”<sup>240</sup> Because chronic pain is often associated with mood disorders,<sup>241</sup> the tendency of treating physicians to attribute unexplained symptoms to psychiatric disturbance is reinforced.

The traditional model of nociceptive pain was essentially static and assumed that the peripheral and central nervous systems are hardwired to communicate painful stimuli in a predictable manner.<sup>242</sup> Current scientific thinking, however, recognizes “neuroplasticity” in the body’s central mechanisms for processing pain.<sup>243</sup> Aberrations in these central mechanisms, most pain researchers now believe, produce much chronic pain.<sup>244</sup> In contrast to common acute pain, “chronic pain resembles a disease, a pathology of the nervous system that produces abnormal changes in the brain and spinal cord.”<sup>245</sup>

As explained by Jon Russell, one of the world’s leading researchers in the pathogenesis of chronic pain, the normal process of nociception “involves detecting pain when tissue injury is present and not feeling pain when no tissue injury exists.”<sup>246</sup> This process is facilitated by a complex array of neurochemicals—some of which transmit pain signals and some of which modulate their strength. Among the modulators,

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techniques”).

239. See Russell, *supra* note 233, at 11; Bennett, *supra* note 212, at 385 (“The correlation between tissue pathology and the perceived severity of the chronic pain experience is poor or even absent. Furthermore, the sharp spatial localization of acute pain is not a feature of chronic pain; chronic pain is more diffuse and often spreads to areas beyond the original site.”).

240. See Thernstrom, *supra* note 143, at 66.

241. See *supra* notes 185, 191 and accompanying text.

242. See Bennett, *supra* note 212, at 387; Gamsa, *supra* note 166, at 163.

243. See WALLACE & WALLACE, *supra* note 5, at 27. Wallace gives as a classic example of the nervous system’s plasticity the phenomenon of “phantom limb,” where pain is felt in the absence of nociceptive stimulus.

244. See, e.g., Jain et al., *supra* note 15, at 51 (“A comprehensive biological model of the important role of the central nervous system . . . and autonomic nervous system . . . is emerging, in which the normal coordination between the brain and the other body systems is disrupted in many ways.”); PRIMER ON THE RHEUMATIC DISEASES, *supra* note 32, at 189 (“Most investigators . . . believe that the primary abnormality leading to the expression of symptoms in fibromyalgia . . . is aberrant central nervous system function.”); Pillemer et al., *supra* note 144, at 1930 (“There is increasing behavioral and biologic evidence that, similar to nerve injury pain, the pathogenesis of FMS pain is related to altered CNS processing of nociceptive stimuli.”); Clauw, *supra* note 22, at 372 (“Most investigators now agree that aberrant central mechanisms are likely to be responsible for the majority of the clinical symptoms findings in fibromyalgia.”).

245. See Thernstrom, *supra* note 143.

246. See Russell, *supra* note 233, at 9.

some neurochemicals are “pro-nociceptive” and amplify pain signals, while others are “anti-nociceptive” and suppress signals.<sup>247</sup> When the normal balance of pain modulators is disrupted, non-noxious stimuli can be sensed as pain, or noxious stimuli may be painless.

As Russell observes,

[T]he pain component of [fibromyalgia] is now viewed as resulting from a pathological amplification . . . of normal incoming signals from the periphery. The result is an enhanced central . . . interpretation of pain even when the signal from the periphery would have been perceived as non-noxious . . . from a normal person without [fibromyalgia].<sup>248</sup>

This chronic enhancement of non-noxious signals is commonly described as “central sensitization,” and is widely accepted among pain researchers as the biological process underlying much enigmatic chronic pain.<sup>249</sup>

A variety of research strategies have also provided the long sought-after objective evidence that those suffering from chronic pain syndromes have “real” pain. First, numerous functional imaging studies of the central nervous system, such as SPECT scans and MRI studies, confirm abnormal activity during the processing of nociceptive stimuli in chronic pain sufferers.<sup>250</sup> Second, using measures of pain that are impervious to patient manipulation or control, several researchers have demonstrated that fibromyalgia sufferers have abnormally high pain sensitivity.<sup>251</sup> Third, a variety of researchers including Russell have

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247. *See id.*

248. *Id.*

249. *See* BERNE, *supra* note 11, at 92. As two leading researchers on chronic pain have observed, “pain is not a passive consequence of the transfer of a defined peripheral input to a pain center in the cortex, but an active process generated partly in the periphery and partly within the CNS [central nervous system] by multiple plastic changes that together determine the gain of the system.” *See* Clifford J. Woolf & Michael W. Salter, *Neuronal Plasticity: Increasing the Gain in Pain*, 288 *SCIENCE* 1765, 1768 (2000); *see also* Roland Straud, *Fibromyalgia Pain: Do We Know the Source?*, 16 *CURRENT OPINION IN RHEUMATOLOGY* 157, 157 (2004) (“There is strong evidence for abnormal central pain processing in FMS.”); Robert J. Schwartzman et al., *Neuropathic Central Pain*, 58 *ARCHIVES NEUROLOGY* 1547, 1547 (2001) (“Central sensitization is the pivotal physiologic phenomenon . . . .”); Jain et al., *supra* note 15, at 43 (“Findings of physiological and biochemical abnormalities provide compelling evidence that clearly identifies FMS as a biological and distinct clinical disorder.”).

250. *See, e.g.,* Jain et al., *supra* note 15, at 59–60 (discussing various imaging studies); Bennett, *supra* note 212, at 388 (discussing studies of electrophysiologic activity in response to peripheral stimulation); Russell, *supra* note 233, at 15 (discussing “SPECT” analysis of regional cerebral blood flow and functional magnetic resonance imaging of nociceptive activity); White, *supra* note 31, at 637 (discussing physiological abnormalities in fibromyalgia patients, including reduced cerebral blood flow); Crofford & Clauw, *supra* note 234, at 1136 (discussing altered cerebral blood flow in fibromyalgia patients); Bradley & Alarcón, *supra* note 23, at 1819–21 (discussing brain scans showing functional activity abnormalities).

251. *See, e.g.,* Borut Banic, *Evidence for Spinal Cord Hypersensitivity in Chronic Pain After Whiplash Injury and in Fibromyalgia*, 107 *PAIN* 7 (2004); Roland Staud et al., *Maintenance of Windup*

found that chronic pain sufferers have significant abnormalities in neurochemicals (pro-nociceptive and anti-nociceptive) important in the central processing of pain.<sup>252</sup> Prominent among these abnormalities is a dramatically increased concentration of a neurochemical aptly named “substance P,” which has been found to amplify pain signals.<sup>253</sup> The migration of substance P through the spinal cord helps explain the widespread, nonspecific pain that is the hallmark of fibromyalgia.<sup>254</sup> Research also confirms that fibromyalgia sufferers have significantly lower levels of important pain inhibitors like serotonin and noradrenaline.<sup>255</sup> These findings confirm that fibromyalgia sufferers are biochemically different from healthy persons.<sup>256</sup> As Russell observes, chemical abnormalities “speak eloquently in favor of a chemical pathogenesis for the pain of [fibromyalgia].”<sup>257</sup>

The specific pathogenesis leading to the “central sensitization” that produces chronic pain is highly complex,<sup>258</sup> and its full understanding will require intensive and costly research.<sup>259</sup> The predisposition to develop centralized pain can result from genetic factors,<sup>260</sup> adverse

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*of Second Pain Requires Less Frequent Stimulation in Fibromyalgia Patients Compared to Normal Controls*, 110 PAIN 689 (2004).

252. See, e.g., Douglas A. Weigent et al., *Current Concepts in the Pathophysiology of Abnormal Pain Perception in Fibromyalgia*, 31 AM. J. MED. SCI. 405, 407 (1998); Jain et al., *supra* note 15, at 51–56; Russell, *supra* note 14, at 17–21; Russell, *supra* note 233, at 19–29; WALLACE & WALLACE, *supra* note 5, at 28–31; Pillemer et al., *supra* note 144, at 1929–30.

253. See, e.g., Jain et al., *supra* note 15, at 51–52. As the consensus reports notes, substance P levels in fibromyalgia patients are substantially higher than those in patients with depression. *Id.* at 45.

254. See Russell, *supra* note 14, at 28; Crofford & Clauw, *supra* note 234, at 1136 (“The most consistent biologic finding supporting aberrant central pain transmission in FM [is] the 3-fold higher concentrations of substance P in cerebrospinal fluid . . . of FM patients . . . compared with those . . . of normal controls . . .”).

255. See Nancy Julien et al., *Widespread Pain in Fibromyalgia Is Related to a Deficit of Endogenous Pain Inhibition*, 114 PAIN 295, 299–300 (2005). The deficiencies in these chemicals may also relate to the observed association between the onset of fibromyalgia and stress. There is considerable evidence indicating the body’s stress response mechanisms (e.g., the hypothalamic-pituitary-adrenal axis) are related to serotonin and noradrenaline. See Jain et al., *supra* note 15, at 56.

256. See Russell, *supra* note 233, at 8, 14; Crofford & Clauw, *supra* note 234, at 1136 (“Taken together, the data on pain processing in FM demonstrate that the central representation of pain correlates with patient reports of pain, and that purely behavioral or psychological factors are not primarily responsible for the pain and tenderness seen in FM.”).

257. Russell, *supra* note 233, at 13; see also *id.* at 45 (“Abnormalities in neurochemical mediators of central nervous system nociceptive function are clearly present in ways that are consistent with the patterns of symptom.”). See also Clauw, *supra* note 22, at 374 (discussing natural variation in individual “setpoints” for biological stress response that may be altered by, for example, trauma).

258. As one leading text on rheumatic diseases emphasizes, the study of the pathogenesis of all rheumatic diseases is “extraordinarily complex.” See PRIMER ON THE RHEUMATIC DISEASES, *supra* note 32, at 99.

259. See Russell, *supra* note 233, at 13.

260. A significant percentage of fibromyalgia sufferers report that a family member also has the illness. See Russell, *supra* note 14, at 22; Bennett, *supra* note 212, at 390. See generally PRIMER ON

environmental factors,<sup>261</sup> or some combination of both. The process leading from predisposition to central sensitization is often triggered by injury, repetitive trauma, inflammation, or illness,<sup>262</sup> resulting in excessive stimulation of the nervous system.<sup>263</sup> For reasons not yet fully understood, this stimulation leads to an increased excitability of neural circuits, referred to as “wind-up,”<sup>264</sup> which permanently alters the neurochemistry of persons who develop chronic pain.<sup>265</sup> As pain researchers observe, “it is not the duration of pain that distinguishes acute from chronic pain but, more importantly, the inability of the body to restore its physiological functions to normal homeostatic levels.”<sup>266</sup> Once central sensitization has occurred, only minor input is required “to maintain the sensitized state and clinical pain.”<sup>267</sup>

This new model of chronic pain has important implications for the law. Most importantly, this model presents a powerful challenge to the view that enigmatic chronic pain is a psychosomatic symptom of unresolved psychic distress. The premise of psychosomatic pain in both the DSM-IV-TR and popular literature is that pain is “medically unexplained.” This premise is increasingly difficult to sustain.<sup>268</sup> As with earlier claims about the psychosomatic origins of rheumatoid arthritis, multiple sclerosis, and AIDS, psychogenic explanations of chronic pain are becoming increasingly obsolete in light of biological

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THE RHEUMATIC DISEASES, *supra* note 32, at 110 (observing that most of the common rheumatic illnesses “result from the interaction of genetic and nongenetic factors”). A recent study by Karen Raphael suggests that certain women may have a genetic predisposition to develop both fibromyalgia and major depressive disorder. See Karen G. Raphael et al., *Familial Aggregation of Depression in Fibromyalgia: A Community-based Test of Alternate Hypotheses*, 110 PAIN 449 (2004).

261. See Pillemer et al., *supra* note 144, at 1932 (discussing possible contribution of abuse, neglect, and childhood illness to fibromyalgia).

262. See, e.g., Bennett, *supra* note 212, at 389–90; Clauw, *supra* note 22, at 374; Russell, *supra* note 233, at 15; Pillemer et al., *supra* note 144, at 1929.

263. See Clauw, *supra* note 22, at 375; Pillemer et al., *supra* note 144, at 1929–30.

264. See, e.g., Staud et al., *supra* note 251, at 689–90; Bennett, *supra* note 212, at 387. As Bennett explains, “wind-up” was first identified by researchers in 1965 and reveals how repetitive stimulation of peripheral nerves causes a progressive buildup in the magnitude of the nervous system’s response. “Investigators now appreciate that the phenomenon of wind-up is crucial to understanding chronic pain.” *Id.*

265. One possible mechanism is the immune system’s production of cytokines in response to infection, inflammation, or trauma. Cytokines are known to contribute to neuropathic pain. See Staud, *supra* note 249, at 160.

266. See John D. Loser & Ronald Melzack, *Pain: An Overview*, 353 LANCET 1607, 1609 (1999).

267. See Staud, *supra* note 249, at 158.

268. It is worth emphasizing that the biopsychosocial interpretation of pain is not inconsistent with the emerging evidence that chronic pain results from dysfunction in the central nervous system. As noted earlier, psychological factors can contribute to the physiological processes involved in pain by, for example, altering the concentrations of important neurotransmitters that modulate pain. See *supra* note 142.

discovery.<sup>269</sup>

One thing is difficult to explain: Why is there virtually no discussion of the emergent biology of chronic pain in case law?<sup>270</sup> Something is amiss when evidence of the biological bases of chronic pain is now featured regularly in *Science*, *Scientific American*, and even the *New York Times*, and yet goes unmentioned in judicial proceedings.<sup>271</sup> This is a disquieting misapplication of the dictum, “law lags science” in the courtroom.<sup>272</sup> The failure of lawyers representing pain claimants to provide biological context for “enigmatic” pain is especially troublesome, since this context both enables courts to make biological sense of chronic pain and calls into question the key diagnostic element of somatization—that pain symptoms have no plausible medical explanation.

One expects that proponents of psychosomatic pain will not concede easily that chronic pain has, in fact, been “medically explained.” The complex research that underlies the discovery of chronic pain mechanisms has yet to produce a lab test or brain imaging technique that can be routinely used to verify a particular patient’s centralized pain.<sup>273</sup> Clinicians fond of psychosomatic explanations can still point out that individual pain claims are “subjective” and unverified, and the ghost of malingering is always present.

That said, contemporary biological understanding of chronic pain has the potential to check the over-diagnosis of mental disorder in chronic pain sufferers. There is far better evidence supporting the concept of centralized pain than somatized pain.<sup>274</sup> While proponents of psychosomatic pain urge that psychogenesis is “one of nature’s basic

269. This has important implications for treatment of chronic pain. While psychotherapies and psychotropic medications might help patients “cope with or modify the intensity and aversiveness of their pain through reduction of emotional distress,” they “should not be expected to greatly modify” pain generated in the central nervous system. See Weigent et al., *supra* note 252, at 411. Or as one physician observes, “Depression or stressful factors can in turn enhance pain . . . . But to make stress reduction a primary strategy for pain treatment is trying to repaint the walls of a crumbling house.” See Thernstrom, *supra* note 143.

270. As noted earlier, the sole reported decision even mentioning the view that chronic pain results from dysfunction in the central nervous system is *Hocraffer v. Secretary Health & Human Services*, No. 99–533V, 2004 WL 627777 (Fed. Cl. Mar. 12, 2004).

271. See *supra* note 96.

272. See *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996).

273. Some pain researchers have recently expressed hope that future brain imaging techniques will permit “a case definition of a particular chronic pain condition that allows us to give it a signature.” See Marx, *supra* note 17, at 329.

274. See, e.g., Crofford & Clauw, *supra* note 234, at 1136 (“Taken together, the data on pain processing in FM demonstrate that the central representation of pain correlates with patient reports of pain, and that purely behavioral or psychological factors are not primarily responsible for the pain and tenderness seen in FM.”).



mechanisms in mobilizing the body to cope with mental distress,”<sup>275</sup> no one has yet proposed a psychophysiological explanation of how this mechanism operates.<sup>276</sup> The psychoanalytic engine once thought to drive somatization—repressed psychic conflict—has been abandoned.<sup>277</sup> Before concluding that mental distress can cause widespread debilitating pain for such a substantial portion of the population, proponents should carry the burden of explaining this extraordinary effect. They have not.

A dismaying inconsistency lingers in the willingness of medical experts to proffer psychogenic explanations of chronic pain while insisting that its biological causes are too speculative. It is worth recalling that the validity of psychiatric classification of the somatoform disorders remains unproven.<sup>278</sup> As with much mental illness, very little is known about their actual pathogenesis.<sup>279</sup> Law and mental health expert Christopher Slobogin has observed that more demanding application of the *Daubert* standard could “herald the end of much behavioral science testimony.”<sup>280</sup> Surely no one advocates such a result. But, in the case of chronic pain, courts should recognize that experts’ casual diagnoses of somatization are often as subjective as the symptoms they purport to explain.<sup>281</sup>

Recent biological research into chronic pain may also help elucidate another mystery courts have confronted since the dawn of modern personal-injury practice: How can minor trauma or illness produce widespread chronic pain? Abundant evidence suggests that trauma or

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275. See SHORTER, *supra* note 43, at x.

276. A useful comparison is “post-traumatic stress disorder.” There appear to be plausible physiological explanations for the development of symptoms in PTSD. See Edgar Garcia Rill & Erica Beecher-Monas, *Gatekeeping Stress: The Science and Admissibility of Post-traumatic Stress Disorder*, 24 U. ARK. LITTLE ROCK L. REV. 9, 11–12 (2001) (discussing the psychophysiological processes that give scientific validity to the diagnosis of PTSD).

277. See *supra* note 165 and accompanying text.

278. See, e.g., Sharpe & Mayou, *supra* note 178, at 465 (“Neither the overall category of somatoform disorder nor its sub-categories satisfy the accepted criteria for validity or reliability.” (citing Kendell & Jablensky, *supra* note 10)).

279. See Kendell & Jablensky, *supra* note 10, at 8 (noting that the etiology of most psychiatric disorders is “still largely unknown”).

280. Christopher Slobogin, *Pragmatic Forensic Psychology: A Means of “Scientizing” Expert Testimony from Mental Health Professionals?*, 9 PSYCHOL. PUB. POL’Y & L. 275, 287 (2003); see also Christopher Slobogin, *Doubts About Daubert: Psychiatric Anecdotes as a Case Study*, 57 WASH. & LEE L. REV. 919 (2000).

281. An example is found in *Riccio v. S&T Contractors*, Nos. 98-07740, 98-07741, 99-05984, 99-06295, 99-06359, 2001 WL 1334202 (Pa. Ct. Com.Pl. 2001). In *Riccio*, one of the leading medical critics of fibromyalgia, Dr. Frederick Wolfe, was willing to testify to a “reasonable medical certainty” that the plaintiff’s chronic pain was exacerbated by “pre-existing psychological and behavioral factors that can be seen to be developing in the decade before the accident” rather than from accident trauma. *Id.* at \*5. Dr. Wolfe is neither a psychiatrist nor a behavioral scientist but rather a professor of internal medicine and family and community medicine at the University of Kansas School of Medicine.

illness often precedes the onset of chronic pain,<sup>282</sup> yet the ensuing pain usually seems disproportionate to the precipitating event.<sup>283</sup> Proponents of psychosomatic pain have eagerly dismissed enigmatic trauma symptoms as “somatoform disorder,” “accident neurosis,” and “traumatic hysteria.”<sup>284</sup> Courts have, understandably, been skeptical of pain claims that make no sense under the prevailing model of acute pain.

Contemporary research provides important clues about how chronic pain can evolve from seemingly minor insults to the body. Pain researchers are coming to appreciate how “injuries can trigger specific and long-lasting biological changes throughout the body’s pain-detection system” leading to a chronic condition where “ordinarily benign stimulus can be agonizing.”<sup>285</sup> For persons who have suffered an alteration in central mechanisms regulating the pain process, common-sense understanding of acute pain is no longer a reliable guide. Chronic pain cannot be immediately related to specific tissue injury, is disproportionate to any identifiable injury, and can spread widely beyond the site of original injury.<sup>286</sup> Yet, these seeming anomalies make sense under the developing model of chronic pain. Those persons predisposed to develop central sensitization may present a particularly challenging class of “eggshell” plaintiffs for the tort-law system. However, they can no longer be summarily relegated to the suspect class of neurotics, somatizers, and malingerers.

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282. See, e.g., Lily Neumann & Dan Buskila, *Epidemiology of Fibromyalgia*, 7 CURRENT PAIN & HEADACHE REP. 362, 364 (2004); Jain et al., *supra* note 15, at 44–45; WALLACE & WALLACE, *supra* note 5, at 16–19.

283. See, e.g., Kirmayer et al., *supra* note 42, at 663–64 (2004) (“Contrary to common clinical assumptions, there is a poor correlation between the nature and extent of an injury and the resulting pain and suffering.”)

284. See *supra* note 215 and accompanying text.

285. See Marx, *supra* note 17, at 326. See also Staud, *supra* note 249, at 158–59 (discussing how trauma or infectious illness can produce “windup” and central sensitization leading to chronic pain); WALLACE & WALLACE, *supra* note 5, at 28 (discussing how repeated, noxious stimulation of the nervous system can lead to central sensitization); Bradley & Alarcón, *supra* note 23, at 1823, 1825 (discussing how physical injury may lead to increases in pro-nociceptive chemicals like substance P and eventually central pain); Pillemer et al., *supra* note 144, at 1929 (discussing how injury produces hyperexcitability and central sensitization of CNS neurons leading to perception of “more intense and prolonged pain”).

286. See Bennett, *supra* note 212, at 387–88 (discussing how in chronic pain “the description of pain seems inappropriate in comparison with the degree of tissue pathology . . . noxious stimuli result in a pain experience that is greater and more unpleasant than would normally be expected . . . normally nonnoxious stimuli may now result in painful . . . and the extent of the pain boundary is greater than would be expected on the basis of the site of the original tissue pathology”).

## VI. CONCLUSION

Accepting the popular criticism that chronic pain is nouveau hysteria or some form of malingering is increasingly difficult. Little scientific evidence supports this criticism and much good science reveals the biological basis for previously enigmatic pain. One hopes this science is introduced into the judicial process before psychosomatic theories of chronic pain become an established part of legal folklore.

Research elucidating the biological basis for chronic pain does not necessarily lead to a happy ending. Scientific insight into the nature of chronic pain comes at an inauspicious time when pain is increasingly conceived of as a public policy concern rather than a form of private suffering. Significant issues loom: How should the health care system respond to the enormous costs imposed by chronic pain;<sup>287</sup> can medical interventions sufficiently ameliorate chronic pain to justify their subsidization; and is pain too ephemeral to be fully valued in the tort-law system?<sup>288</sup> Most of these issues are ultimately “trans-scientific.”<sup>289</sup> But, their resolution should be informed by contemporary scientific insight into the nature of chronic pain and not predicated on fictionalized accounts of human suffering.

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287. See Lisa Melton, *Aching Atrophy*, SCI. AM., Jan. 2004, at 22, 24 (reporting that chronic pain costs U.S. employers an estimated \$61.2 billion each year in lost productivity).

288. See, e.g., Joseph H. King, Jr., *Pain and Suffering, Noneconomic Damages, and the Goals of Tort Law*, 57 SMU L. REV. 163 (2004).

289. See Phillip H. Schuck, *Multi-Culturalism Redux: Science, Law and Politics*, 11 YALE L. & POL’Y REV. 1, 12–13 (1993) (defining “trans-scientific issues” as those formulated in scientific terms but unanswerable by science alone).