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Reconsidering Fordyce's classic article, "Pain and Suffering: what is the unit?" to help make our model of chronic pain truly biopsychosocial

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Abstract

The biopsychosocial model (BPS) of chronic pain aspires to be comprehensive, incorporating psychological and social factors omitted from biomedical models. Although psychosocial factors are viewed as highly influential in understanding behavioral and psychological responses to pain, these factors are usually viewed as modifiers of biological causes of the experience of pain itself, rather than as equal contributors to pain. To further advance the BPS model, we re-examine a classic 1994 paper by Wilbert "Bill" Fordyce, "Pain and suffering: what is the unit?" In this paper, Fordyce suggested that pain-related disability and suffering should be viewed as "transdermal", as having causes both inside and outside the body. We consider Fordyce's paper *theoretically* important because this concept allows us to more fully break free of the medical model of chronic pain than customary formulations of the BPS model. It makes it possible to place psychological and social factors on an equal footing with biological ones in explaining pain itself and to remove distinctions between pain mechanisms and pain meanings. The brain's salience network now offers a platform on which diverse influences on pain experience—from nociception to multisensory indicators of safety or danger—can be integrated, bridging the gap between impersonal nociceptive mechanisms and personal meanings. We also argue that Fordyce's article is *practically* important because this concept expands the BPS model beyond the bounds of the clinical encounter, opening the door to the full range of social, psychological, and biological interventions, empowering patients and non-medical providers to tackle chronic pain.

Abstract

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In the classic 1994 paper, “Pain and suffering: what is the unit?”, Wilbert “Bill” Fordyce argues that pain-related disability and suffering must be understood as transdermal, as having sources both inside and outside the body.

> ”The ‘unit’, then, with which the practising health care provider is confronted in dealing with pain patients is a biopsychosocial entity which encompasses far more than what resides within the skin.” – Wilbert Fordyce, “Pain and suffering: what is the unit?”, *Quality of Life Research*, 1994[29]

Introduction: our current biopsychosocial model is biased toward biological causes of pain

Across the health professions, there is near universal endorsement of a biopsychosocial (BPS) approach to treating chronic pain. The 2016 U.S. National Pain Strategy explicitly endorses the BPS approach: “Chronic pain is a biopsychosocial condition that often requires integrated, multimodal, and interdisciplinary treatment, all components of which should be evidence-based.” The biopsychosocial approach is also a key feature of Australia’s National Pain Strategy and is explicitly endorsed as a component of national pain strategies by the International Association for the Study of Pain. [19; 72; 73] The BPS model aspires to be comprehensive, incorporating psychological and social factors often omitted from biomedical models. But the biological, the psychological, and the social factors incorporated into the BPS model are not treated equally in the model as it is usually conceptualized and implemented. Rather, psychological and social factors are generally investigated as modifiers of biological causes rather than as independent contributors to the experience of chronic pain. We propose that this “biology first” perspective has limited the applicability and effectiveness of the BPS model of chronic pain care.

Fordyce’s article: “Pain and Suffering: what is the unit?”

Our critique of the BPS model is inspired by a classic paper published in 1994 by one of the pioneers in pain psychology, Wilbert “Bill” Fordyce: “Pain and suffering: what is the unit?”[29] Fordyce is most famous for his conceptualization and treatment of pain behavior according to operant behavioral theory.[57] His 1994 paper continues his contention that chronic pain problems cannot be understood apart from their interpersonal and social context. In this paper, Fordyce argues that these problems are “transdermal,” meaning they have causes both inside and outside the boundary of the body. We consider Fordyce’s paper *theoretically* important because the transdermal concept allows us to more fully break free of the medical model of chronic pain, place psychological and social factors on an equal footing with biological ones in understanding pain itself, and remove distinctions between pain mechanisms and pain meanings. We also argue that Fordyce’s article is *practically* important because the transdermal concept opens the door to psychological and social as well as biological treatments for chronic pain, empowering patients and non-medical providers to tackle chronic pain. Fordyce was a master at condensing profound insights into pithy phrases like “transdermal,” but at present this term suggests a form of drug delivery, so we suggest the use of other terms like “contextual,” “relational,” or “ecological.”

In his paper, Fordyce claims “there appears to be inadequate understanding of the nature of pain complaints and their relationship to extant suffering and mood states of the complaining person.” He says this is based on “the conceptual basis from which pain and suffering complaints are interpreted and case management and treatment methods are derived.” Note that Fordyce is extending his operant analysis of pain behavior to the patient’s suffering. He proposes viewing this suffering as determined by psychosocial contextual factors as well as biological factors within the patient’s body. Nevertheless, he continues to distinguish pain (‘as a signal’) from suffering (‘as an emotional state triggered by anticipation of threat to one’s self or identity’). This follows the definition of suffering provided by internist Eric Cassell in his famous 1982 *New England Journal of Medicine* article, “The Nature of Suffering and the Goals of Medicine.”[17] Both Fordyce and Cassell, therefore, appear to operate within the traditional view that sees pain as a biologically-produced sensation that prompts an emotional and behavioral reaction, which is itself shaped by additional features of people and their environments. This approach focuses on pain as a cause of suffering (as pain-related suffering), but not on suffering as a cause for pain, which is encompassed by the new contextual approach.

The most important new concept introduced by Fordyce in this paper is the “transdermal” concept, which Fordyce borrowed from Vicki Lee’s 1992 paper: “Transdermal Interpretation of the Subject Matter of Behavior Analysis.” By “transdermal,” Lee meant something that has both organismic (internal, physiological, psychological) and environmental (external, interpersonal, social) determinants. For Fordyce, like Lee, the primary focus was behavior: “The most important implication of ‘transdermal’ is that it implies that a state of permanent disability of a person cannot be assigned without considering the ongoing environment in which he or she functions.” For Fordyce, disability was not an intrinsic property of the patient, but a characteristic of a patient-within-an-environment.

Fordyce went beyond Lee in extending the “transdermal” concept beyond behavior to include suffering as well: “The assignment of status as disabled is the rampant problem with which this paper is principally concerned. At present, disability determination fails to take into account the transdermal nature of suffering, particularly as it pertains to low back pain.” Fordyce also extended the transdermal concept to include the patients themselves: ”The ‘unit’, then, with which the practicing health care provider is confronted in dealing with pain patients is a biopsychosocial entity which encompasses far more than what resides within the skin.” This important claim has not been fully incorporated into pain medicine: patients with chronic pain are not the biological bodies that one sees in the clinic, but the full people with all of their histories, interpersonal relationships, and sociocultural contexts.

Although Fordyce focused on behavior and suffering, new studies of pain itself view pain as the product of influences both inside and outside the body as integrated within the brain. The fact that Fordyce did not explicitly focus on the contextual nature of the pain experience is consistent with his famous reluctance to ask patients about their pain levels as they progressed through treatment. For Fordyce, the pain experience was not the singular, or even the primary, cause of the suffering and disability associated with chronic pain. Personal pain experience was but one component of the complex social phenomenon of chronic pain.

Psychosocial causes distinct from biological causes

Pain scientists and clinicians broadly accept that psychosocial factors can modulate or modify the experience of pain, but such modulation occurs within a general theory focused on biological causes for pain. Even the Gate Control Theory of Pain, proposed as an alternative to a traditional biomedical model of pain causation, begins with nociception and then adds modulation from both peripheral and central sources. The “central factors” in the original Gate Control paper pointed toward an as yet undefined psychosocial modulation of pain experience transmitted through efferent fibers descending from brain to spinal cord.[63]

Clinical chronic pain care also begins with a biomedical evaluation, followed sometimes by a psychosocial evaluation. But these psychosocial evaluations are not given equal priority or weight in clinical care or medicolegal proceedings. Nowhere is this more apparent than in the independent medical examinations used by Worker’s Compensation evaluations in the U.S., where tissue pathology—but not psychosocial pathology—is allowed to legitimize disability due to pain. Similarly, the U.S. Social Security Administration also evaluates disability due to chronic pain in terms of medical impairments:

> “statements about your pain or other symptoms will not alone establish that you are disabled. There must be objective medical evidence from an acceptable medical source that shows you have a medical impairment(s) which could reasonably be expected to produce the pain or other symptoms…”[3]

In the past 20 years, neuroimaging research has begun to demonstrate that social and psychological factors can activate similar structures in the brain to those activated by nociceptive input. Neuroimaging research has increasingly emphasized overlapping neural circuits related to the experience of physical pain and socially painful experiences, including exclusion,[25] rejection,[47] and bereavement.[33; 69] Individuals with chronic pain reliably show higher rates of adverse life experiences [31] and traumatic exposures [16; 39] compared to the general population, and these factors appear to be predictors of chronic pain onset and severity [16; 39].

Mechanisms claimed to underlie the connection between adverse social environments and chronic pain include dysregulation of immune system function, [10] altered endocrine function, [94] deficiencies in emotional recognition and regulation abilities, [2; 78; 79] altered resting state connectivity, [34] and central nervous system sensitization.[77] These psychological and social influences on pain were formerly categorized within a dualistic framework as “psychogenic,” “functional,” or “somatized” pain that was contrasted with purely “somatogenic” or “physical” pain. But now these psychophysiological mechanisms and the brain’s salience network within which they operate offer us a non-dualistic framework for pain causation that integrates causes originating both inside and outside the body under the unifying concept of “danger,” which can encompass both physical and social threats.[83] Research to date suggests that when individuals are positioned in highly stressful, threatening, or invalidating circumstances, they can become vulnerable to sensitized and overprotective responses like pain.[24] For example, depression and anxiety are often interpreted as emotional responses to the aversive sensory experience of chronic pain. But there is good epidemiological evidence that depression and anxiety may also

precede the onset of chronic pain and increase the risk of the onset, severity, or chronicity of chronic pain.[35; 38; 82]

Epidemiological studies of psychological and social risks for chronic pain, however, cannot control all relevant variables and are subject to residual confounding. Efforts have been made to address these problems in epidemiologic research. In 1965, the English statistician Sir Austin Bradford Hill proposed nine criteria by which to evaluate epidemiologic evidence of a causal relationship between a presumed cause and an observed effect.[36] These included: Strength (effect size), Consistency (reproducibility), Specificity, Temporality, Dose-response relationship, Plausible mechanism, Coherence (with laboratory findings), Experimental evidence (if available), Analogy (with other causal processes). These criteria have been applied to putative psychological and social causes of pain as well as physical causes.[80]

In addition to the complexities of psychological influences on chronic pain, we must also address the complexities of of social influences. Social determinants of health include "the causes of health inequality" [59] and encompass sociodemographic and economic factors such as education, race, ethnicity, gender, employment status, income, and financial insecurity. These social factors have been highlighted recently as major contributors to non-communicable diseases including cardiovascular health, diabetes, addiction, and chronic pain.[14] Recent systematic reviews have indicated that low education status and low income or socioeconomic status are consistent risk factors for chronic pain prevalence, severity, and related disability [45] as well as predictors of poor surgical response in chronic pain. [92] Similarly, research has noted longstanding race-based disparities in chronic pain severity and pain-related disability, with Black Americans showing a disproportionately high burden of severe and disabling chronic pain. Importantly, these racial effects appear to contribute above and beyond the effects of socioeconomic disparity. [43; 62] Immigrant and minority populations have a higher prevalence and severity of chronic pain in many countries.[37] Social deprivation, such as poverty, unemployment, and living alone is also associated with chronic pain severity across countries. [42; 60; 91] It is important to note that the influence of social determinants is not confined solely to functional outcomes such as disability, but to the presence and severity of chronic pain itself. [23]

Controlled experiments examining the social and cultural influences on pain are difficult to execute. Most research on these factors is retrospective and correlational. The research reviewed by Princeton economists Anne Case and Angus Deaton in their *Deaths of Despair* study of the American opioid epidemic is typical of that found in economics and the other social sciences.[15] Their research explores the capacity of one variable to influence another. Trends in unemployment, pain prevalence, and opioid prescribing are compared and contrasted in different countries and counties. Mechanisms by which these social factors exert their influence are not often explored in detail. For example, an association between higher educational attainment and better health status has been repeatedly reported. But it is not possible to randomize people to receive different amounts of schooling, so the studies remain correlational and subject to the challenges of reverse causation and omitted variable bias. Sometimes, natural experiments can be exploited to minimize the risk of these biases. [46]

In contemporary discussions, pain causation is generally understood in terms of pain mechanisms. Natural science tends to use a mechanical notion of causation that originated with Descartes and contemporaries. This perspective postulates that a cause is fully separate from its effect, temporally precedes this effect, and operates through a specified physical process.[89] Psychological and social sciences borrow this sense of mechanism, but expand the definition of causes and effects to include beliefs and behaviors as well as physical processes.[5] This raises the question: what is a psychological or social mechanism? Articles in the social sciences often include diagrams of causal models that depict psychological or social mechanisms with a series of boxes and arrows. The boxes depict concepts or beliefs and the arrows depict relations of influence. But the boundaries of these concepts are fluid (e.g., where does catastrophizing end and depression begin?). And the relations of influence are not mechanical in the classical sense (e.g., how does catastrophizing promote physical disability?). Social science models may, therefore, dispense with explicit causation claims in favor of a description of the statistical relationships among various factors.[40] The humanities (such as history and literature studies) tend to talk of relations among meanings rather than mechanisms. This raises the question: can meanings interact mechanically? More concretely, we might ask: do social and cultural causes of pain always operate by means of psychological mechanisms inside the individual? Answering these questions is beyond the scope of this paper. But overcoming the “bio bias” of our current BPS model of chronic pain means not only recognizing psychological and social factors as potential causes of pain, it means accepting different types of causation (e.g., interactions between meanings and mechanisms).

The treatment implications of “biology first”

There is a “treatment-prevalence paradox” [71] for many chronic pain conditions: the number of interventions that have been provided—both biomedical and psychological—has increased over the years, yet we continue to have an “epidemic” of chronic pain problems. This suggests that our current interventions [21; 22] are not reducing the population burden of chronic pain and poses questions about the efficacy of these interventions. It also raises the uncomfortable possibility that some of our interventions produce iatrogenic harms—worsening rather than ameliorating chronic pain problems. The use of imaging to identify the biological “cause” of the pain can produce iatrogenic harms. When spinal or other abnormalities are discovered on imaging in the absence of ‘red flags’ that suggest serious disease, patients and providers can too easily interpret these abnormalities as causes of pain and intervene accordingly. This has been shown to increase surgery rates and costs without an improvement in patient outcomes.[44] This “biology first” approach to clinical pain, which looks for psychosocial influences only after medical causes have been excluded, inappropriately supports the view that pain is fundamentally a bodily problem, and contributes to the idea that pain must be addressed medically.

More broadly, one wonders whether the heightened sociocultural emphasis on the medical evaluation and treatment of pain results in an increase in the prevalence of pain? A long view of the history of medicine and the evolution of people’s symptoms across eras suggests that somatic experiences—such as pain and fatigue—that are unclear in their etiology but validated by scientific and political interests, rise in prevalence, whereas those that can

be disproven through clinical testing—like conversion paralysis—diminish in prevalence.[81] Cross-national differences in the incidence of chronic pain stemming from injury (e.g., whiplash) [70; 74] might also be attributable to differences in symptom expectation and attribution, which are themselves shaped by sociocultural as well as socioeconomic processes. [26]

Consider what qualifies as a cause of chronic low back pain. Up to 84% of adults may have an episode of back pain in their lifetimes, with 26% reporting at least a full day of back pain in the past 3 months.[32] McGuirk and Bogduk report in the latest edition of *Bonica's Management of Pain, "*Although the possible sources of back pain have been demonstrated [through experimental stimulation studies], its causes [in clinical practice] have been more elusive. Conventional methods of assessment and investigation typically fail to identify the cause of chronic low back pain in the majority of patients."[61] They go on to explain that "many conditions traditionally considered to be possible causes of chronic low back pain *are actually not causes*." Degenerative changes in the back are common in people with back pain, but they also are common in persons with no back pain.[8] Tumors and infections can cause back pain, but they are rare. Back pain is often attributed to "muscle sprain, ligament sprain, segmental dysfunction, and trigger points," but these are difficult to verify, and it is unclear how they can cause pain to be chronic. Dysfunction of sacro-iliac and facet joints can produce pain that can be blocked with nerve blocks, but this blocking rarely lasts very long. Herniated intervertebral lumbar disks can cause back pain and sciatic nerve pain, but they are also seen in persons with no back pain. Surgery can speed recovery of sciatica, but without surgery, pain decreases in approximately 87% of patients within 3 months.[20] Thus the discovery of causes that would allow us to base therapy on the pathophysiological mechanisms of back pain has been elusive.

If we look at the risk factors for developing low back pain, especially chronic low back pain, a much broader set of issues is relevant. Physical factors such as older age, female sex, obesity, and smoking are important. But also important are psychological factors (e.g., anxiety, depression), social factors (e.g., low education, life stress), and occupational factors (e.g., physically or psychologically strenuous work, sedentary work, low social support, job dissatisfaction).[32] Case et al. provide evidence that the effect of educational attainment on pain prevalence is currently increasing in the U.S. population.[13] A recent review of the social determinants of low back pain synthesized 41 studies with data from over 2 million adults in 17 countries. Strong evidence for independent social effects was found, including place of residence, race, occupation, education, socioeconomic status, and social capital.[45] Education and socioeconomic status showed the strongest associations. Social forces not only contribute to pain but frame painful experiences. Kugelmann has described how the individual subjective experience of pain as well as our concepts of pain are framed by social narratives.[49] Social forces also affect symptoms (e.g., fatigue and nausea) and other chronic diseases (e.g, diabetes, asthma) beyond pain.[53; 88]

Fordyce argues that the medical approach to back pain is flawed. He aligns himself with Allan and Waddell who "provide a powerful reason for seeing backache as a problem which is not particularly medical." He explains that care-seeking for low back pain is not driven by medical features of the patient's back: "ambiguities inherent in low back pain

indicate clearly that something more than nociceptive stimulation is involved in the problem. Only a small percentage of those who report onset of back pain in work settings perceive the problem as warranting medical care." Fordyce argues that other factors are relevant: "negatively toned mood state makes care-seeking more likely." He cites the findings of the large prospective study of back pain in the workplace that he directed: "Biomechanical and ergonomic factors did not prove to be predictors of later back injury report. Measures of job happiness at the time of entry into the study and personality measures from the MMPI did."

In clinical pain medicine, we tend to separate pain (as produced by impersonal mechanisms originating within the body) from suffering (as arising from personal and interpersonal meanings originating between bodies). In the dominant version of the BPS model, pain may cause suffering through psychological mechanisms like fear-avoidance and catastrophizing.[24] Personal and interpersonal processes of meaning-making involving the relation between patients' identity, agency, and social status and their suffering do not receive as much attention.[27] Both the biomedical model and the dominant version of the BPS model focus on the former mechanistic questions about "how" pain is produced and modulated rather than the latter meaning-focused "why" questions about personal reasons for pain and suffering.[84] Our contemporary interpretation of Fordyce's model challenges this separation of mechanisms and meanings and these distinct approaches to pain versus suffering. Pain not only produces suffering, but can result from suffering.[82] A truly biopsychosocial model of chronic pain will need to explain how impersonal mechanical processes and personally meaningful processes can interact to cause pain.

New neuroscience of pain offers a means of integrating mechanisms and meanings

Recent functional neuroimaging research may point to new ways to synthesize the biological, psychological, and social contributors to the pain experience. Multiple authors have proposed that what was formerly called the "pain matrix" of brain centers specific to pain perception are more properly considered a multisensory "salience network"[7; 48; 51] activated by sensory events of various modalities that indicate or predict a threat to the body's integrity. These events include not only nociceptive stimuli but also non-nociceptive stimuli (e.g., visual, auditory) that contribute to the brain's determination of the salience or relevance of input to an organism's survival. Non-nociceptive threatening stimuli, if salient, may produce a pattern of brain activity grossly identical to that produced by nociceptive stimuli.[41] For example, visual information of approaching threats can increase nociceptive responses in somatosensory centers. Therefore, the activity in the brain areas that respond to nociceptive stimuli is not a reflection of pain intensity, but of threat salience. Intensity has been favored as the most important feature of noxious stimuli because intensity is an objective property of the stimulus. According to psychophysical experiments, stimulus intensity is mirrored in the report of pain intensity.[75] But these are controlled experiments, not actually threatening clinical situations. Threat and salience cannot be studied with psychophysical techniques. Salience is a measure of the importance of the stimulus for the organism and its survival. In moving from stimulus *intensity* to stimulus *salience*, we have

begun to synthesize biological *mechanisms* and psychosocial *meanings* in the etiology of pain.

The brain's salience network expands the influences on pain experience from nociception to multisensory indicators of safety or danger, and thus offers a pathway by which personal meaning (such as the relevance to survival) interacts with impersonal nociceptive mechanisms. According to Legrain and colleagues, "Indeed, salience detectors represent neural mechanism by which selective attention is captured and oriented towards the most salient stimuli in order to prioritize their processing over background stimuli, to improve their perception and to prompt appropriate action."[52] The salience network, therefore, may integrate internal and external factors—nociception and psychosocial influences on pain—to promote survival. The central role played by salience in pain perception means, as Legrain explains, "that the purpose of pain is not merely to induce and to associate the feeling of unpleasantness to a somatosensory sensation, but it also to warn the body about potential physical threats."

Importantly, this detection of threats is not limited to damage within the body. Legrain et al. explain, "Responding adequately to events that threaten the body's integrity constitutes an action whose achievement requires close interaction with systems that are able to localize threatening information in the proximal space of the body." For example, salience network neurons may respond to visual objects when they are *approaching* the body, but not when they are *moving away* from the body. This salience detection system will react to a wasp approaching my hand even before it actually stings my hand. Salience detection includes the "peripersonal space," which includes the body and the environment within grasping distance. There is a close relationship between visual, proprioceptive, and tactile processing of threats in this peripersonal space.

This salience model was not available to Fordyce in 1994, but it offers us a model for pain that bridges the boundaries of the body from "inside the skin to outside the skin."[7] A salience-focused approach means that the brain is changed by events inside and outside of the body. The brain is thus a crossroads of interacting causal influences, not an ultimate cause for pain. It has long been clear that evaluating threats both external and internal to the body is critical to survival. Now it is becoming clear that it is also essential to understanding the pain experience and its variable relationship to tissue damage. This variability includes feeling pain in the absence of a noxious stimulus, reporting minimal pain in the setting of major tissue damage, having an 'analgesic' response when no analgesic has been administered, or producing no pain relief after administration of a potent analgesic medication. The salience network idea helps explain why psychological trauma that produces no tissue damage can be associated with dramatic increases in pain.[54] It is also compatible with the concept of allostasis, which is the ability to adapt to pain and stress and maintain homeostasis. As psychosocial stressors become chronic, allostatic load can be come overwhelming, with negative long-term pain outcomes.[56] For example, patients with chronic pain who also report PTSD symptoms have not only greater psychological distress, but greater pain intensity and pain interference.[50]

The path forward to a truly biopsychosocial model of chronic pain

In considering how to implement our view of the biopsychosocial model, it is important to acknowledge both the relative successes and limitations of extant models. The concept of social and emotional contributors to the experience of chronic pain is not new but is, in fact, among the oldest in psychology.[28] However, the clinical explanation of pain has shifted away from socioemotional factors, in part, because these have been considered invalidating and stigmatizing for patients. Whereas older models of psychogenic pain assumed that psychogenic and somatogenic pain excluded each other, newer models strive to integrate these factors to make the BPS model more inclusive or patient-centered.[83] Carr and Bradshaw have advocated a "sociopsychobiological" model that identifies "emotional upset, family dysfunction, and economic stress" as drivers of pain rather than just effects.[11] Quintner et al. argue that the BPS is unable to overcome dualism in its implication that pain is a thing separate from the body.[76] Fordyce adds to these efforts by encouraging us to look at causes of the pain experience both inside and outside the body.

Psychological explanations of pain that are restricted to patients' reactions to biologically-generated pain, and the possibility of treating those reactions to improve functioning, may be more acceptable to patients than addressing psychosocial contributors to pain. Whereas established psychotherapeutic models have targeted primarily cognitive, affective, and behavioral responses to a given pain experience, more recent psychological interventions have borrowed from contemporary neuroimaging research and framed pain as a signal that arises *directly from social, cognitive, and emotional influences*. Perhaps the most salient example of this shift concerns pain neuroscience education (PNE)-based interventions, which have arisen over the past 15 years and offer a reconceptualization of pain as a brain-based response to perceived danger, rather than as a reliable indicator of bodily damage.[65] Educational interventions utilizing PNE improve maladaptive beliefs and fears about pain but show more modest effects on pain and pain-related indicators of function.[90] Trials implementing PNE in concert with more active retraining interventions show promise in reducing pain and may be a key component of unlocking greater treatment effects via a fundamental and experientially-based reconceptualization of chronic pain as a non-dangerous brain signal.

One recent trial described markedly improved outcomes combining PNE with motor training in chronic low back pain, which yielded clinically significant reductions in pain, disability, and catastrophic appraisals related to pain compared to a standard exercise and education condition. [58] Further, there was a recent trial of a pain neuroscience-based intervention employing tracking techniques for somatically-based sensations in concert with graded exposure principles for individuals with non-specific low back pain (Pain Reprocessing Therapy). Central to this intervention was the idea that chronic pain itself is an automatic brain-based misinterpretation of sensory signaling that can be changed via higher-order cognitive and affective regulation efforts. This intervention yielded highly positive results, with 66% of participants enrolled in the active condition reporting 0/10 or 1/10 pain intensity at the end of treatment with treatment effects largely maintained 1 year later.[4] These interventions demonstrate both the possibility that a reconceptualization of pain is

needed in order to reduce chronic pain, and that this reconceptualization involves more active, experiential relearning to yield larger and more durable pain relief.

Similarly, evidence is mounting from clinical trials over the past 15 years that trauma or emotion-focused interventions can yield superior analgesic effects in some pain conditions. We note that trials of Emotional Awareness and Expression Therapy (EAET) conducted in both civilian fibromyalgia [55] and Veteran chronic musculoskeletal pain samples [93] have shown superior effects compared to cognitive-behavioral therapy (CBT) in reducing some measures of pain severity. A key tenet of this therapy, like the aforementioned Malfliet [58] and Ashar [4] trials, teaches that chronic pain is derived primarily from interpretations of bodily threat and alarm, rather than as an accurate reflection of bodily damage or nociceptive signaling. There is also promising evidence that the well-established benefits of CBT approaches can be augmented through implementation of emotion-focused exposure protocols. [6] The results of these various trials demonstrate a crucial point: *social and emotional factors can directly increase pain intensity,* and *addressing these factors may yield superior effects in pain reduction*.

Discussion: looking beyond the bounds of our clinical model

Fordyce's view that pain-related disability and suffering is "transdermal" or contextual was prescient, but also limited. He focused on the contingencies provided by the social environment and the resultant impact on patients' pain behavior and suffering. This view shaped not only operant treatment but also cognitive-behavioral interventions for the ensuing three decades. Fordyce could not have known what neuroscience has taught about the brain's ability to integrate multiple sources of threat into the pain experience. But research now suggests that the sociocultural context may drive the onset, severity, and maintenance of pain itself. It is widely recognized that social factors affect access to care, including the likelihood of being evaluated for one's pain and offered various treatments, as well as the effectiveness of those treatments. Beyond these health care system effects, social factors may also help determine the onset or severity of pain. Just as individual patients' past experiences with childhood trauma can activate the brain's salience-danger/threat system and thereby pain pathways, current and historical social experiences that indicate danger may also activate this pain alarm. Some of these social dangers are to the integrity of one's body and life—such as physical threats from food insecurity, [64] community violence, or disproportionate law enforcement—resulting in pain activation that reflects the biological purpose of the pain-danger alarm, which is to promote survival. There also are myriad psychosocial threats, such as employment loss or instability, being demoted on the social hierarchy, social rejection, and social injustice.[9; 12; 18; 87] Such threats to one's social status may activate the pain-alarm as well, resulting in disproportionate pain prevalence and severity for marginalized groups. One might also view the presence or severity of pain as a declaration of inequity, unfairness, and harm. "I hurt" is a personal cry that reflects one's body, one's psyche, and one's social situation.

Problems with our assessment of pain begins with medical training that is focused on biological disease and damage. The medical student will be held accountable for missing a cancer diagnosis or a broken bone, but not held accountable in the same way for missing a

psychosocial determinant of pain or illness such as post-traumatic stress disorder. Validation, support, and reassurance about the absence of serious bodily damage can themselves be therapeutic, and can sometimes be all that patients seek or need. But the forces favoring a bodily explanation for pain within the practice of modern medicine can be overwhelming. The medical practitioner is expected to find an explanation and make a diagnosis so that something can be fixed. Insurance coverage is based on diagnostic coding that leaves few opportunities for either the recognition or the treatment of contributors to pain that are outside the body. The penalty for missing a bodily cause of pain during clinical practice could be even greater for the practitioner than for the medical student who faces reprimand, but will not be sued or lose a license to practice.

Our problem continues within the walls of the medical office. The biopsychosocial model for pain and its treatment has long been accepted, but it has been squeezed into a medical office that is set up for poking and prodding, testing and diagnosing patients' bodies. The very nature of the clinical encounter was built to focus attention within the skin. It may not be within the scope of practice for the medical practitioner to tackle the societal ills that cause or contribute to pain, yet recognition of the limits of the medical office by health systems, payors, and public health officials could be an important step forward. Fordyce acknowledged these limits in 1996 when, as chair of an IASP task force on pain in the workplace, he wrote a report recommending that after 6 weeks of rehabilitation, back injured workers who were unable to return to work should no longer be offered medically-disabled status.[30] He was declaring, in a sense, that medicine no longer had anything to offer once people with pain who had been offered appropriate diagnostic, curative and rehabilitative services. This recommendation was poorly received by many in the pain community because it was interpreted as a statement that persistent pain was no longer real.[85] But hopefully we have moved beyond that, and can understand that pain can be real even if its causes and solutions lie outside the medical office. The difficulty is how to have an exit strategy from the biomedical model that can frame the psychosocial contributors to pain as primary without invalidating the patient's pain and suffering.

We can no more turn our back on medical advances that have at once made medicine more technical and better at prolonging life, than we can extend medicine beyond the medical office into society itself. Medical care is not the best answer for social problems. Gone are the days of the family physician who lived in the village and knew all its inhabitants from birth to grave, through adversity and triumph, offering kindness and wisdom, but rarely a cure. However, we are not helpless. Our knowledge extends to a newfound understanding of how the brain integrates the factors that would today be considered outside the control of medicine. With that understanding, we can offer new treatment approaches that help reverse the damage caused by adverse social and societal factors. This requires reconceptualizing pain as an alarm signal stemming from a perception of danger. Patients can be taught this model and can begin to understand and address these sources of danger. Maybe patients can make changes in their lives, but even if they cannot, understanding the sources of their pain can still help. More importantly, the person can be taught safety. There are new treatments emerging with this focus, many with promising results.[4; 66; 67]

What are clinicians to do? Patients will continue to present with a complaint of pain. The first role of the physician is to find a bodily cause and treat it, if there is one, remembering that most cases of chronic pain lack valid biomedical explanations. Making an accurate medical diagnosis remains the physician's primary role, but validation, reassurance, and safety messaging are equally important skills in chronic pain treatment. This is especially true for the pain we might now call chronic primary pain by ICD 11 definition.[68; 86] This is the common type of pain for which conventional medical approaches have been found lacking.[1] This is the pain that begs for a complete reconceptualization, a new understanding of what such pain is, and one that necessarily involves patients themselves, often supported by specialty behavioral approaches. Here clinicians are not powerless because they can provide reassurance and a sense of safety, relieving fear that may be a driving force for the development of refractory pain.

Investigating and addressing the social determinants of pain may be more challenging within a medical system focused on the individual patient. At a minimum, clinicians should, in the explanatory models given to patients, include social factors as contributors to both pain and disability. Clinicians can also advocate for reform in the policies and regulations based on this assumption. We also need to address social system-wide changes in the factors that elicit a sense of danger and that activate the salience network, including sociopolitical changes to counter racism, sexism, income inequality, and the many other social determinants of pain.

Conclusion

A truly biopsychosocial model of chronic pain challenges not only the biomedical model, but more broadly points out the limitations of the traditional clinical model of pain care. It challenges not only the biomedical idea that pain is caused by disease and damage, but the BPS idea that prioritizes biological factors and relegates psychological and social factors to "modulators" of pain. Clinical care usually limits itself to intervention targets that can be addressed with individual patients in the clinical setting. Chronic pain, however, is not just a clinical problem, and we should not consign our conceptualization and treatment of pain to what is addressable in the clinic. Clinical work risks not only "biomedicalizing" people in pain but also "psychologizing" them as well—reducing them to individuals whose bodies—or whose thoughts, emotions, and behaviors—are the cause of their pain and needing to be changed. Throughout his career, Fordyce strove to understand patients with chronic pain in their social context. In his later years, his criticism of the traditional clinical understanding of pain and emphasis on the social determinants of pain resulted in a break with some of his colleagues. But Fordyce was right in pointing toward the idea that broader sociocultural factors play a fundamental role in the experience of chronic pain. This is what he meant in asking: Pain and Suffering: what is the unit? The one who experiences pain and suffering is not the body in the exam room, but the person embedded in their life. Life in its whole—biological, psychological, social, and contextual—as well as past, present, and future matters to pain. We do not need to be defeated by this complexity, but we do need to incorporate it into the way we understand, assess, and treat pain.

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