Chapter 4
Pain Perception in Mental Health: An Overview

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4.1 Introduction

Chronic pain and the apparent insensitivity (or indifference) to acute pain constitute two very different, yet often observed clinical phenomena in psychiatry—depending of course on the type of pathology being considered. The presence of such dichotomous response profiles demand that we pay close attention to the processing of nociceptive signals and to the presence of a possible change in the expression of subjective pain ratings among psychiatric patients. A better understanding of how psychiatric patients actually process pain holds the promise of better understanding psychiatric problems in their own right, and, of better understanding the neurobiological roots of psychiatric ailments. The current chapter provides an overview of issues concerning pain perception and mental illness. It is divided as a function of the clinical disorders most frequently associated with a change in perceived pain. Our review is based on the careful consideration of the most recent, and sometimes older but seminal, peer-reviewed scientific literature. Our classification of mental disorders is based on the multiaxial taxonomy of the Diagnostic and Statistical Manual of Mental Disorders–4th edition, text revision (DSM IV-TR) [2]. We begin our overview of pain and mental health by focusing on severe, or principal, psychopathological conditions (i.e., clinical disorders: Axis I of the DSM IV-TR). In this manner, schizophrenia, autism, anxiety disorders, mood disorders, and somatoform disorders will all be discussed. Our chapter then proceeds to review the impact of personality on the development and/or maintenance of painful conditions. Here, the

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S. Marchand et al. (eds.), *Mental Health and Pain*,
DOI 10.1007/978-2-8178-0414-9_4,

emphasis is placed on personality disorders (Axis II of the DSM IV-TR), but we will also consider the influence of personality or character traits (N.B., personality traits are not maladaptive, as is the case for personality disorders, but rather define enduring patterns of behavior and feelings that pervade most aspects of day to day life among healthy adults. Personality traits, taken together, define one's personality). Finally, it is worth noting that this chapter provides a general appraisal of issues and findings that concern pain and mental illness. It offers an introduction to this rapidly evolving field and prepares the reader for the more detailed chapters that follow.

4.2 Schizophrenia

Dating as far back as the beginning of the twentieth century, anecdotal and clinical reports have been accumulating to suggest that patients suffering from schizophrenia experience pain quite differently from unaffected individuals. In the course of defining the disorder, Kraepelin and Bleuler remarked, early on, that schizophrenia patients had a decreased ability to detect, or possibly report, pain [7, 22]. Kraepelin and Bleuler even provided several examples of apparent pain insensitivity among their own patients. The two authors were also among the first to propose the hypothesis that pain insensitivity among schizophrenia patients may reflect either the presence of sensory abnormalities or the presence of a change in non-sensory, affective processing.

A change in pain sensitivity among schizophrenia patients may explain why some patients present with serious traumatic injury, but demonstrate little or no pain complaints. Decreased sensitivity to pain among schizophrenia patients may also explain their limited use of—and reliance on—health services. It may also, partly, explain the near ubiquitous lack of treatment-compliance observed among this population. The limited consumption of health services can, in turn, contribute to the increased morbidity and mortality observed among schizophrenia patients. Despite the obvious clinical consequences of altered pain perception, few experimental studies have investigated how schizophrenia patients actually respond to painful experimental stimuli. To date, results collected on this issue can be, at best, qualified as equivocal. Whereas some authors report a decrease in pain sensitivity among schizophrenia patients, others report an increase in pain sensitivity, or no change at all (for a thorough review, see [8]). Current studies do not allow us to draw strong conclusions concerning the direction and cause of altered pain perception in schizophrenia because current studies vary immensely in their: (i) characterization and inclusion of pain threshold, pain tolerance, and, pain perception measures, (ii) inclusion of objective, pain-evoked physiological responses, (iii) use of subjective pain assessment scales, (iv) use and nature of painful stimuli (e.g., type of pain stimulus used and length of time applied), and, (v) inclusion of age- and sex-matched control groups.

Schizophrenia is characterized by a mixture of unique signs and symptoms, including at least 30 days of active-phase symptoms (delusions, hallucinations,

disorganized speech, grossly disorganized or catatonic behavior, and/or negative symptoms). Patients typically present with distinct symptom-profiles, which are believed to be the reflection of distinct changes in the neurobiology or neurochemistry of the central nervous system (CNS). It is important to note that symptom profiles, together with the presence of measurable changes in subjective pain experiences, can provide useful clues regarding the neurobiology of pain processing alterations in schizophrenia. In recent years, several hypotheses have been advanced to explain why schizophrenia patients experience pain differently from healthy controls. For example, altered pain perception in schizophrenia has been linked to the severity of negative symptoms. Flattening of affect, in particular, is thought to be responsible for a disease-associated change in the processing of pain's objectionable properties (i.e., its immediate, disagreeable dimensions) [13]. Another hypothesis is that disease-associated changes in pain perception are caused by the presence of positive symptoms (e.g., hallucinations and delusions). Positive symptoms may increase the experience of pain among schizophrenia patients because they lead to an aberrant attribution of salience to afferent nociceptive information [18]. This particular hypothesis would elegantly explain why some authors have found schizophrenia patients to be more, not less, sensitive to acute painful stimuli [14, 17, 28, 38]. Cognitive deficits among schizophrenia patients are also advanced to explain why some patients experience pain differently from unaffected controls. Cognitive slowing is regularly noted among schizophrenia patients and can affect how readily, or fluently, pain is expressed. Cognitive slowing may also prevent the rapid and contextually appropriate matching between ongoing painful experiences and previous painful experiences. Poor contextual matching would prevent the adoption of suitable, pain-related reference behaviors, which may explain why schizophrenia patients sometimes display behaviors that can be interpreted as pain-insensitive [30]. Another hypothesis is that the presence of motor symptoms, such as psychomotor slowing, may interfere with the behavioral response demanded during experimental pain testing, and thus, be responsible for the false impression of hypoalgesia noted by researchers. Psychomotor slowing may affect reaction time to pain specifically [14], or, it may indiscriminately affect the processing of all incoming information [25]. This still needs to be verified. A final hypothesis proposes that mood alterations (depression, anxiety, stress) may be responsible for the change in pain expressed by patients [13]. All of these hypotheses are currently plausible and need to be formally tested before definitive conclusions can be drawn.

The dominant neurochemical hypothesis regarding the etiology of schizophrenia suggests an impairment in the organization and efficacy of dopaminergic circuits [15]. Since dopamine is thought to play an important role in both the pathophysiology of schizophrenia and the proper functioning of endogenous pain control systems (i.e., inhibitory pain circuits) [34], some authors (e.g., Potvin et al. [35]), have proposed that schizophrenia patients may be less sensitive to pain because their endogenous pain control systems are over-active. Interestingly, these authors found that schizophrenia patients and healthy controls show a comparable degree of pain inhibition. Despite the similar degree of pain inhibition observed between patients and controls, Potvin et al. [35] found that schizophrenia patients actually display

less pain sensitization than healthy controls. Pain sensitization refers to the progressive increase in subjective pain reported when stimulation intensity is kept constant but stimulation frequency increases. Given this finding, it is possible to argue that there may be truth to the idea that schizophrenia patients are less responsive to pain—at least when prolonged or tonic pain is tested. Results of this study have recently been replicated in our lab, and now confirm that the lack of pain sensitization displayed by patients is not due to spinal sensitization, but rather, to a probable change in the cortical processing of prolonged pain [27].

Notwithstanding the possible, and likely multiple, causes underlying the change in pain processing observed among schizophrenia patients, most authors would agree that altered pain perception can have important clinical consequences. It is essential, therefore, that we adapt our clinical practice when treating schizophrenia patients, including the exercise of greater attention to the possible presence of unreported chronic pain.

4.3 Autism

Autism is defined as a complex developmental disorder [1] and is associated with a multi-factorial etiology. The pathophysiological process of autism includes environmental and genetic factors. Neuroimaging studies report numerous changes in central nervous system (CNS) functioning and CNS neurotransmitter concentration (review: [23, 29]). Autism is a heterogeneous pathology more accurately defined as autism spectrum disorder (ASD). Patients suffering from ASD present at least three of the following impairments: (i) qualitative impairments in social interactions, (ii) qualitative impairments in communication, and, (iii) restricted, repetitive, and stereotyped patterns of behavior, interests, and activities [2, 42]. Moreover, autism is associated with various other clinical conditions, including mental retardation, hyperactivity, attention deficit disorder, motor impairments, sleep disorders, etc. [42, 47]. ASD is a severe condition affecting anywhere from 0.04 to 1 % of the population [9]. Despite a relatively weak prevalence, ASD has a profound impact on family members, with both emotional and economic costs. The chronic course of the disease, along with the presence of co-morbid conditions, makes ASD a serious and disabling condition.

When considering the consequences that social and communication difficulties can have on patient well-being, it is not surprising that the rate of injury observed among ASD patients is twice as high as the rate of injury observed among the general population [9]. Some authors even report a high rate of self-mutilating behaviors among patients [37]. Self-injurious behaviors, together with a decreased predisposition to report pain, raises numerous questions regarding the true nature of the hypoalgesia observed among ASD patients. Is the purported indifference to pain observed among autistic patients truly the result of a different mode of expression to pain, or, is it the reflection of a real change in the processing of nociceptive afferents?

The presence of a possible change in the processing of pain among ASD patients has been the focus of only a few clinical and experimental studies. Some clinical observations do indeed suggest the presence of a legitimate form of endogenous analgesia among autistic patients—evidenced by a lack of nociceptive-specific reflex activity, an inability to initiate protective behaviors, and a failure to adopt analgesic postures in response to painful stimuli [46]. Despite these clinical observations, experimental studies (which remain relatively rare) do not suggest the presence of an intrinsic form of hypoalgesia among ASD patients, but rather, suggest that ASD patients express pain differently from unaffected individuals [31, 33]. At this junction, it is important to point out that some authors have, indeed, documented a decrease in pain reactivity among ASD patients [19, 32, 45, 46]. These studies, however, used parent impressions and ratings as proxy scores for their ASD children's pain experiences. This is unfortunate since the scientific literature clearly indicates that parents (in general) tend to underestimate the pain felt by their child [10]. Third-person reports in ASD pain studies are greatly influenced by the relationship maintained between the observer and the ASD child, causing biases that depend directly on the nature of the relationship held with the child. In order to document the effects of relationship status on third person pain assessments, a recent study compared how parents, doctors, and nurses rated the pain experienced by ASD patients [45]. Results revealed the presence of varying degrees of pain sensitivity *for the same ASD patient*—ranging anywhere from normal, to hypoalgesic, to hyperalgesic, depending on the observer. An external observers' evaluation of the pain felt by an ASD patient, therefore, should be considered very cautiously. Finally, it is worth noting that facial expressions suggesting the presence of pain during venipuncture in ASD children are comparable to those of age-matched control children [31]. It seems obvious, therefore, that more research is needed before we can fully understand how pain is experienced and expressed in this population.

As is generally the case with multi-factorially determined disorders, numerous neurotransmitter systems are thought to be involved in the pathophysiology of ASD. Nevertheless, the most strongly supported evidence to date concerns a change in serotonergic neurotransmission [23]. Unfortunately, serotonin's role in pain perception has not yet been studied among ASD patients. Experimental pain studies published to date mostly concern β-endorphins (a type of endogenous opioid). Pain researchers have found evidence of elevated β-endorphin levels in ASD patients exposed to painful stimuli [45]. Unfortunately, elevated β-endorphin levels in response to pain are not easy to interpret. Such an elevation could be responsible for hypoalgesic responses among ASD patients, or, it could be the result of a stress-evoked response that is unrelated to pain processing. Evidence of a possible change in endogenous opioid system function among ASD patients currently remains tenuous [23].

Finally, many of the neurotransmitter system alterations observed in autism also relate to the neurotransmitter systems involved in the modulation of pain signals. Since ASD varies greatly from patient to patient, and since this variability reflects neurobiological changes that are unique to each patient, there is little doubt that a substantial amount of inter-individual variability in pain reactivity dominates the

pain sensitivity profile of ASD patients in general. Moreover, communication difficulties among ASD patients add another layer of complexity to the evaluation of pain and suffering in this population. Thus, if we re-examine our previous discussion concerning the high rate of self-mutilating behaviors among ASD patients, one possible, but yet unstated explanation for these behaviors, may be that these behaviors reflect an attempt to deal with chronic unrelieved pain, rather than the manifestation of strong endogenous analgesia. It is imperative to entertain this possibility when trying to make sense of the pain response profile of ASD patients, and possibly even question the inclusion of hypoalgesia as part of the set of sensory symptoms associated with ASD in the DSM-IV-TR [2].

4.4 Anxiety Disorders

During the last few decades, the influence of anxiety on pain perception has been abundantly studied, and anxiety is now accepted as one of the most influential psychological factors determining the final, subjective experience of pain [44]. The impact of anxiety on pain perception is supported by several psychophysical studies, which show that a high degree of anxiety is associated with an increase in subjective pain and a decrease in pain perception threshold and pain tolerance threshold [44].

Although a substantial number of pain studies have considered the influence of anxiety, very few studies have focused on anxiety disorders (AD)—such as generalized anxiety disorder (GAD) and panic disorder (PD). The majority of pain studies conducted with patients suffering from AD are, in fact, cross-sectional epidemiological studies. These studies support the existence of a link between AD and pain by demonstrating, for example, that the prevalence of AD is higher among people who suffer from chronic pain than among the general population [3]. Interestingly, the effects of AD on pain seem stronger for GAD than for all other types of AD [6].

Although cross-sectional association studies provide interesting information pertaining to the association between AD and pain, these studies provide no information regarding the nature, or even the direction of causal influences. It remains difficult, therefore, to determine if AD predispose patients to the development of chronic pain given the right set of stressors, or, if alternatively, chronic pain predispose patients to the development of AD. Although the ensemble of influences is most probably bidirectional, the first hypothesis is more strongly supported. For example, a retrospective study by Knaster et al. [20] found that the diagnosis of AD precedes the onset of pain for more than 75 % of individuals (!). Similarly, Shaw et al. [41] observed that men consulting for an initial complaint of low back pain were 2.45 times more at risk of developing chronic low back pain when they also suffered from GAD than when they did not. Importantly, the association between AD and pain is not without consequences for clinicians working with this population. According to a recent survey of psychiatrists practicing in different parts of the world, pain is one of the most difficult symptoms to treat among patients who have

been diagnosed with AD [4]. This situation leads to significantly higher treatment costs for pain among people who suffer from both psychiatric and pain problems than for people who suffer only from pain [51].

From an epidemiological standpoint, the relationship between AD and pain is well established. But what of the evidence collected from experimental pain studies? As mentioned earlier, far fewer of these studies have been conducted and the results published to date remain far less convincing. Two exceptions concern well-known experimental pain studies carried-out in the 1980s, which showed that participants diagnosed with AD (PD, agoraphobia, GAD) and healthy controls had comparable electrical pain thresholds [21, 36]. The absence of a group effect is not limited to electrical pain. In 1999, a study by Lautenbacher et al. [26] confirmed that patients with PD had the same mechanical and thermal (heat and cold) pain thresholds as healthy controls. Critically, Lautenbacher et al. noted a very weak association between experimental pain measures (pain thresholds) and clinical pain measures (number of painful sites and intensity/unpleasantness of clinical pain over the past 6 months), suggesting that experimental pain thresholds are a poor proxy of the clinical pain felt. As a result, we cannot assume that the absence of effect observed using experimental pain data translates readily to an absence of effect for clinical pain data. If well-controlled experimental pain studies do not confirm a change in pain sensitivity among patients suffering from AD, how then, do we explain the high population prevalence of clinical pain among patients presenting with AD? Perhaps the answer can be found in the mutual maintenance model proposed by Sharp et al. [40]. This model suggests that AD and chronic pain share common pathological components (whereby the circuitry and components of one may piggy-backed on top of the other). These components include avoidance behaviors, reduced activity levels, as well as a tendency to dwell on current and past traumas. According to the mutual maintenance model, people suffering from AD report pain more frequently because they are inclined to focus more on their experience of pain, not because their pain threshold is somehow lower. In other words, the difference between chronic pain patients who present with co-morbid AD and chronic pain patients who do not may depend more on psychological processes than on neurophysiological ones.

4.5 Depression

A significant number of patients suffering from chronic pain also suffer from depression. Although the co-occurrence of depression and pain vary greatly from study to study, it is generally estimated that depression rates are twice as high among patients suffering from chronic pain than they are among the general population [24]. Epidemiological studies, therefore, point to a close link between pain and depression. Unlike what can be observed for anxiety, however, depression generally develops following the onset of pain. This brings many researchers and clinicians to believe that depression is more likely to be the result of chronic pain than it is to

serve as a predisposing factor. Notwithstanding the apparent development of depression from pain, it is important to remember that a close association between factors does not entail the automatic (or necessary) presence of a causal relationship, even if one factor precedes the other in time.

From a neurophysiological point of view, the association between pain and depression is not surprising. Several neurotransmitter systems (e.g., serotonergic, dopaminergic) and CNS structures (e.g., anterior cingulate cortex, amygdala, brainstem) are known to play a role in the development and/or maintenance of both mood disturbances and pain. This gives biological plausibility to the presence of a shared etiology for chronic pain and depression. It is also not surprising to note that many therapeutic approaches originally intended to relieve symptoms of depression (e.g., antidepressants, psychotherapy) are now also extensively used to treat chronic pain.

According to some researchers, the link between pain and depression can be explained by a patient's expectations regarding treatment efficacy or regarding the evolution of their condition. That is, for the same pain condition, a depressed patient may tend to anticipate greater pain than a patient who does not suffer from depression, thus partly explaining why the first patient eventually experiences more pain than does the second. This 'expectation-based hypothesis' was supported in 2001 by Sullivan et al. [43] who found that pain expectancies mediate the relationship between depression and pain. By negatively influencing expectations of relief or expectations of therapeutic success, depressive thoughts maintain patients in a vicious cycle of perpetual treatment failure and continued pain. The powerful link between expectation and pain is now well-documented, even among people who do not suffer from depression [48].

Although clinicians often observe the relationship between pain and depression, this relationship is much harder to document when formally tested in laboratory settings. Instead of finding increased pain sensitivity among depressed patients, experimental studies have largely reported decreased pain sensitivity (i.e., increased pain thresholds) [39], suggesting the presence of hypo- rather than hyperalgesia. In an attempt to explain this apparent paradox, Hall and Stride proposed, as early as the 1950s, that depressed patients show decreased pain sensitivity scores because depression: (i) is linked to a general state of non-responsiveness, and/or, (ii) produces affective indifference to artificial (experimental) stimulations [16]. In support of this hypothesis, Bär and collaborators [5] showed that the change in pain threshold observed among depressed patients actually depends on the type of stimulation used. Specifically, the authors report *increased* pain thresholds (hypoalgesia) when thermal and electrical stimulations are used, and *decreased* pain thresholds (hyperalgesia) when ischemic stimulations are used. The observations of Bär and collaborators suggest that while some pain stimuli might easily be perceived as artificial and thus trivial, others, such as ischemic pain, are much more difficult to ignore. As a whole, the data reviewed above help us to better understand how depression (and possibly other mood disorders) can bias pain perception, while also emphasizing the important role played by patient expectations, patient interpretations, and inner cognitive discourse.

4.6 Somatoform Disorders

The common feature to all somatoform disorders is the presence of physical symptoms that suggest a general medical condition but where no diagnosable general medical condition can be found. Somatoform disorders differ from psychological factors affecting a medical condition in that no medical condition is actually present in the former. Complaints of pain that cannot be fully substantiated from an organic standpoint are a central feature of somatoform disorders, in particular of somatization and pain disorders. Since the biological causes of chronic pain are often very difficult to prove, we should not be surprised that somatization and/or pain disorders be associated with the presence or development of chronic pain [12]. In this context, the relevant question is not whether or not there is a link between somatization and pain (since the presence of pain is a defining feature here), but whether or not somatization (and/or pain disorder) should even be considered as a valid diagnosis in the first place. This formulation stems from the fact that the biological origins of pain are typically missed when tested with the basic diagnostic armamentarium available to the general practitioner. In fact, short of having access to the neuroimaging tools available almost exclusively in laboratory settings, the biological origins of chronic pain often remain invisible. If we are to accept this, then differentiating chronic pain (in particular idiopathic chronic pain) from a somatoform complaint is nearly impossible. Moreover, the reader should remember that the link between injury and pain is not always obvious. In other words, the patient's readout of pain from injury is not always linear. Thus, ascribing a somatoform affliction remains very difficult. A great deal of professionalism and a thorough understanding of chronic pain syndromes are necessary to ensure a correct differential diagnosis, not to mention patient welfare. Given these caveats, it is worth noting that very few clinicians actually adhere to the attractive, but dated, dualist view of mind and body originally proposed by René Descartes (see Damasio [11] for a cogent argument against Cartesian dualism and for the embodiment of the mind). Rather most clinicians understand that chronic pain is largely the result of complex interactions between psychological and physiological pressures. This less dogmatic vision of pain and psychological suffering can be appreciated in the new DSM IV-TR [2] diagnostic criteria for pain disorder, which now allows psychiatrists to choose between pain disorder associated with psychological factors and pain disorder associated with both psychological factors and a general medical condition.

4.7 Personality Disorders

According to the DSM IV-TR, personality disorders are an enduring pattern of behaviors that: (i) deviate markedly from those expected from the individual's culture, (ii) are pervasive and inflexible, (iii) develop during childhood, (iv) crystallize during early adulthood (usually in the early 1920s), and, (v) lead to distress or

impairment. Personality disorders affect at least two of the following areas: cognition, affectivity, interpersonal functioning, or impulse control. In most cases, personality disorders are present well before the onset of pain syndromes. The prevalence of personality disorders among patients suffering from chronic pain varies between 31 and 81 %. This proportion is substantially larger than what is observed among unaffected adults, which does not exceed a population prevalence of 8 % [49]. Unfortunately, studies that have look at the type of personality disorder most associated with the development of chronic pain do not provide definitive answers. Histrionic, dependent, paranoid, and borderline personalities have all been described as being the type of personality disorder most frequently associated with a co-morbid pain condition. This lack of agreement stems, in part, from a substantial amount of inter-study variation in methodology, notably in the way personality disorders are diagnosed (either via structured interview or via the administration of multidimensional questionnaires). However, this result also reflects a true heterogeneity in the distribution of personality disorders among patients suffering from pain. Thus, even if personality disorders are more frequently observed among pain patients, no single type of personality disorder is especially associated with the development of pain. In other words, variability in the overall profile of personality disorders is the same among chronic pain patients as it is among the general population. Thus, there is currently no evidence to support the concept of a pain-prone personality. Nevertheless, it is important to note that there are enduring patterns of inter-relational styles that appear to predispose patients to the development of chronic pain. For example, patients who evolve from acute to chronic pain often display sub-clinical features that evoke the presence of a possible personality disorder. These sub-clinical features worsen to constitute a noticeable personality disorder when an acute stressor is present, such as pain. Character traits most frequently associated with the development of chronic pain are those associated with anxious or fearful behaviors (such as those denoting neurotic, paranoid, dependent, or passive aggressive traits) as well as those associated with dramatic, emotional or erratic behaviors (such as those denoting histrionic, borderline, or narcissistic traits). The idea that a simple personality trait can become overtly pathological following the development of chronic pain constitutes a defining feature of the diathesis-stress model of chronic pain [49]. According to this model, biological and/or psychological predispositions amplify a patient's vulnerability to environmental stressors. The interaction between pre-existing vulnerabilities (e.g., a poorly adapted personality trait) and a stressful event (e.g., pain) can promote regressive defenses, which accentuate personality traits, resulting in the expression of a personality disorder (as defined by Axis II of the DSM IV-TR). Although diathesis-stress models were originally advanced to explain the development of schizophrenia, Weisberg and Keefe modified them in the late 1990s to account for the pain-induced expression of personality disorders [50]. Today, the diathesis-stress model of chronic pain is no longer understood as a rigorously linear model, but rather as a model where diatheses and stressors influence one another. In this manner, an acute stressor can provoke the decompensation of maladaptive coping styles into observable pathology, in much the same way that a maladaptive coping style can increase an individual's risk

of being exposed to unnecessary stressors. By viewing personality disorders in this fashion, clinicians are less likely to attribute maladaptive coping styles to the patient, and more likely to attribute them to an underlying interaction between the patient and his/her environment.

4.8 Conclusion

In clinical settings (psychiatric wards and pain clinics alike), the association between mental health and pain is well established and well appreciated, but remains poorly understood from a neurobiological standpoint. The result is a misunderstanding of the developmental trajectory of pain (as it evolves into chronic states) among psychiatric patients, and, an escalation of treatment costs due to the complicated control of chronic pain. Poor pain control among psychiatric patients is unfortunate since psychiatric patients are particularly vulnerable and in need of continued compassionate care. The instant we ignore, or under-appreciate, subjective complaints of pain among psychiatric patients, we run the risk of: (i) interfering with all of our clinical efforts, (ii) undermining our therapeutic alliance, and, (iii) maintaining our patients in a vicious cycle where psychopathology and pain negatively influence one other. A better understanding of the neurobiological systems linking pain and psychopathology will help us to better appreciate and treat our patients' legitimate pain complaints. Although the last few decades have offered numerous answers to lingering questions concerning pain and mental health, many questions remain unanswered. For example, we currently ignore the exact association linking pain and psychopathology for many disorders, as well as the reasons why this link varies so much within and between disorders. We also have a poor understanding of the risk factors (genetic, environmental, cognitive, affective, etc.) that predispose otherwise pain-free psychiatric patients to developing chronic pain. Are some risk factors uniquely relevant to people who suffer from a psychiatric condition or are they relevant to all? Finally, we must continue to explore the specifics of nociception; uncoupling peripheral, spinal, and, cortical contributions to the change in subjective pain expressed by psychiatric patients. Given the growing interest for this particular field of research, the next few years should provide new and clinically relevant discoveries.

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