

Type V — Somatic-Sensitization, Pain-Dominant PTSD

Absurd Health Institute · Medical Concept Papers, Volume Three · Third editorial pass, 17 October 2026

Article type: Mechanistic research perspective · **Status:** Conceptual argument, not clinical guideline

Pain may become a principal maintaining loop after trauma. Trauma-related distress and persistent pain frequently interact, but the connection cannot be explained simply by saying psychological suffering creates physical pain. Nociceptive injury, central sensitization, sleep disturbance, avoidance, and changes in movement may all contribute. The Institute's pain-dominant PTSD profile proposes that, in a subset of patients, persistent pain becomes an active mechanism perpetuating threat and disability, rather than a passive comorbidity that can safely be ignored while addressing traumatic memories.

Consider someone who avoids activity because pain predicts danger. Reduced movement contributes to deconditioning, poor sleep raises pain sensitivity, and persistent bodily vigilance reinforces apprehension. This is a plausible feedback cycle. But inflammatory arthritis, nerve injury, spinal pathology, and other medical causes must be evaluated in their own right. The existence of psychological amplification does not nullify physical disease; nor does a normal imaging study establish that pain is psychogenic.

A profile that must outperform ordinary comorbidity coding. A prospective test would distinguish nociceptive, neuropathic, and nociplastic pain features while recording PTSD symptoms, sleep, activity, and medication use. The proposed mechanism predicts that changes in pain interference and movement confidence will precede or predict changes in trauma-related functioning in some participants. Investigators should compare these dynamics with those among chronic-pain patients without PTSD. If the pattern is fully explained by pain severity and depression, a separate “type” offers little diagnostic information.

The case for a pain-maintained loop could motivate coordinated care across trauma-focused psychotherapy, rehabilitation, and condition-specific pain treatment. It cannot justify withholding established treatments or assuming that one symptom must be cured before another can improve. The scientific opportunity is to identify bidirectional pathways and show when treating both delivers a durable functional benefit that treating one alone does not. A careful theory of somatic adaptation is more controversial—and more useful—than simply saying pain lives in the mind.

Pain creates a different obstacle from intrusive memory A person may learn to tolerate traumatic memories while physical pain still prevents work, sleep and movement. In another person, improved sleep and reduced threat monitoring may lower pain interference even when tissue pathology remains. This two-way relationship makes the pain-dominant profile worth testing, but it should not be taken to mean pain is imaginary or that psychotherapeutic progress necessarily eliminates tissue injury.

The research needs carefully assessed pain mechanisms and control groups with pain but no PTSD. Longitudinal changes in movement, sleep, pain sensitivity and trauma symptoms can reveal whether a proposed maintaining loop has temporal direction. The strongest clinical outcome is restored participation in valued activities, not the eradication of one symptom scale. The theory deserves recognition only if its cross-domain prediction adds something beyond routinely diagnosing and treating both pain and PTSD.

The necessary comparison group A pain-focused trauma profile cannot be tested only against PTSD patients who have little pain. It must also be compared with patients who have comparable chronic pain without PTSD, and ideally with patients who have similar trauma histories but different pain mechanisms. Only then can investigators ask whether trauma-linked threat and avoidance create a genuinely distinctive maintenance loop. Standard pain assessment matters because neuropathic pain, active inflammation and nociplastic symptoms are not interchangeable. An intervention that helps movement confidence may not treat a structural lesion, and the theory should not suggest otherwise. The goal is predictive integration, not replacing either pain medicine or trauma care.

Causation can run in both directions between pain and trauma symptoms An injury may cause pain that intensifies threat vigilance; persistent vigilance may then amplify pain interference and avoidance even as tissue healing progresses. The feedback can be clinically important without implying that the pain was “all in the mind.” A useful study would measure condition-specific pain mechanisms, sleep and trauma symptoms repeatedly, looking for lead-lag patterns rather than relying on one cross-sectional correlation.

The proposed type is worthwhile if those patterns predict who benefits from coordinated rehabilitation and established trauma treatment. A strong null result would be that ordinary assessment of pain and PTSD separately predicts outcomes just as well. The Institute should welcome that finding because it would prevent creation of a redundant diagnostic category. Its real target is overlooked interaction, not the multiplication of labels.

One trajectory can contain two disorders and a shared maintaining loop
Chronic pain and PTSD may coexist because a traumatic event caused both physical injury and threat learning, because each aggravates the other, or because a third condition contributes to both. The model must distinguish these possibilities through history, clinical pain assessment and repeated symptoms. An apparent improvement in both domains after one treatment does not prove that a single underlying circuit caused them. Nevertheless, a reproducible bidirectional relationship could explain why isolated care sometimes produces incomplete functional recovery. The proposed profile should be judged by whether it predicts that interaction and changes treatment outcomes, not by whether its name sounds mechanistic.

Detailed considerations from the original investigation

Trauma can coexist with persistent pain, gastrointestinal symptoms, headaches and heightened attention to bodily sensations. The source book proposes a somatic-sensitization phenotype that gives such presentations explicit status rather than treating physical symptoms as incidental. Its central question is whether changes in pain processing and interoception sometimes constitute a dominant maintenance pathway in post-traumatic illness.

Pain is produced by peripheral inputs, central processing, prior learning, inflammation and context. Threat-associated vigilance may increase attention to innocuous sensations; ongoing pain disrupts sleep and elevates threat appraisal; avoidance and deconditioning can exacerbate disability. This does not imply that the pain is imaginary. Structural disease, neuropathy, inflammation and visceral pathology may be present simultaneously. Interoceptive amplification is only one possible mechanism and should not become an explanatory shortcut when diagnostic work is incomplete.

A person with trauma and severe abdominal pain needs appropriate gastrointestinal evaluation even when symptom attention and stress appear important. The presence of central sensitization never establishes the absence of organic disease. A responsible somatic-trauma paper shows how layered explanations can coexist without demoting a patient's physical complaint to a mere emotional expression.

The greatest hazard is diagnostic overshadowing: attributing new chest pain, neurological deficits, GI bleeding or disabling symptoms to trauma without appropriate medical evaluation. A history of abuse does not make a person less likely to have ordinary medical disease. Nor can central sensitization be inferred from pain intensity alone. Psychological mechanisms and structural pathology often overlap.

A discriminating analysis would use validated pain interference, sensory testing, interoceptive measures and longitudinal functional outcomes to see whether a reproducible pattern predicts response to specific integrated care. If the phenotype does not add beyond pain severity and comorbid diagnoses, the extra label may be unnecessary.

The conceptual contribution is to treat pain and trauma together without confusing the absence of an obvious lesion with the absence of genuine suffering or cause.

Pain and trauma can mutually reinforce vigilance, avoidance and poor sleep, but structural pain generators still require proper diagnosis.

A patient with widespread sensitization may also have inflammatory arthritis, neuropathy or spinal pathology.

Combine pain phenotyping and PTSD measures while testing directionality and independent contributions to function.

Selected scientific reading

1. McEwen (1998), Protective and damaging effects of stress mediators, New England Journal of Medicine.
<https://doi.org/10.1056/NEJM199801153380307>
2. Lanius et al. (2010), Emotion modulation in PTSD: evidence for a dissociative subtype, American Journal of Psychiatry.
<https://doi.org/10.1176/appi.ajp.2009.09081168>
3. Campbell et al. (2020), Identifying PTSD symptom typologies: a latent class analysis, Psychiatry Research.
<https://doi.org/10.1016/j.psychres.2020.112779>

Additional evidence checked for this edition

VA/DoD, *Management of Posttraumatic Stress Disorder and Acute Stress Disorder*, 2023.
<https://healthquality.va.gov/HEALTHQUALITY/guidelines/mh/ptsd/>

Editorial source note: The inherited selected readings are retained from the source manuscript and have not all been independently verified. Newly checked evidence is identified separately. The mechanistic extensions are hypotheses except where explicitly described as established physiology.