

Could Neuroinflammation Distort the Sense of Self?

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The proposition we defend

The felt sense of being oneself may depend on immune and metabolic conditions. We argue that in a subset of patients, inflammatory signaling could change interoceptive network function enough to produce depersonalization, derealization or a loss of embodied familiarity. This is more precise than claiming “inflammation causes mental illness”: the predicted endpoint is alteration in self-experience, not every psychiatric symptom.

Depersonalization and dissociation are often discussed psychologically. Here the question is whether changes in inflammatory signaling, sleep and interoceptive network function can sometimes change the quality of embodied self-experience. This is a mechanistic hypothesis, not a claim that inflammatory biomarkers diagnose dissociative illness.

Why the mechanism deserves to be taken seriously

Cytokine activity can affect sleep, motivation and neural signaling; astrocytes modulate excitatory synapses; and interoceptive processing contributes to the brain's model of bodily state. A combined disturbance might alter the integration of bodily sensations, memory, emotion and agency. Severe autoimmune encephalitis or delirium demonstrate medical causes of altered self and awareness, but they are not direct models of ordinary depersonalization.

The causal argument

The bodily self depends on integration of internal signals, sensory predictions, affect and memory. Inflammatory mediators can modify sleep, motivation, sickness behavior and neural signaling; disturbed sleep can also destabilize perception and emotional salience. A plausible pathway runs from inflammatory signaling through interoceptive and salience-network processing to altered bodily familiarity. The challenge is that these intermediates are not usually measured together, and common blood markers do not directly reveal inflammatory activity in the relevant brain circuits.

Causal architecture under closer examination

The embodied self is constructed from ongoing integration of bodily sensation, spatial perception, memory and affect. Inflammation may disturb several of the conditions that support that integration: sleep quality, motivation, interoceptive salience and neural excitability. This supplies a route by which an immune event might change not only mood but the perceived relationship between self and body. Yet the route must be carefully specified. A raised peripheral inflammatory marker does not demonstrate central inflammatory activation or altered activity in networks that support self-experience. Moreover, depersonalization can accompany anxiety, trauma, seizures, medications and sleep deprivation. The neuroimmune thesis should identify a subgroup in which inflammatory activity reliably precedes and covaries with episodes of altered embodied self. It should ask whether sleep disruption mediates the effect rather than simply labeling sleep as a nuisance variable. In some patients the true causal sequence could be inflammation leading to poor sleep and then altered perception; in others panic and insomnia may explain the event without immune input.

A concrete physiological case

Consider someone whose episodes of depersonalization reliably follow an inflammatory illness and coincide with severe sleep fragmentation. A useful study would measure symptom timing, sleep architecture and inflammatory dynamics, rather than assume that one elevated C-reactive protein value proves the cause. If episodes occur independently of immune change but reliably after panic attacks, a different mechanism may be more persuasive.

Original conceptual illustration

A person reporting depersonalization after sleep deprivation might experience symptom relief with restored sleep without any immune intervention. Another patient with fever, seizures, disorientation and rapidly evolving behavioral change needs medical assessment for encephalitis or other acute conditions. The proposed neuroimmune model should not merge these distinct presentations simply because both involve altered consciousness or self-experience.

What established physiology already supports

Inflammatory states can influence affect, cognition and neural function; depersonalization is a clinically recognized subjective phenomenon. The frontier claim is a causal bridge from specific immune states to a specific alteration of self-modeling. Human evidence for that bridge is far thinner than the underlying facts, which makes good discrimination especially important.

The strongest counterargument—and the answer

The best alternative is dissociation driven by panic, trauma, medications, seizures or sleep deprivation independent of inflammation. These must not be erased by a broad neuroimmune story. The model strengthens when inflammatory events temporally precede the altered self-experience after the competing pathways are measured, and when responses depend on the predicted immune or sleep mediator.

Trauma history, panic, migraine, epilepsy, drug effects, sleep deprivation and primary dissociative disorders remain crucial alternatives. Inflammatory markers are nonspecific and may be elevated because of unrelated obesity, infection or stress. The presence of inflammation would not establish direction of causality.

The discriminating experiment

A prospective study could capture episodes with ecological symptom reports, repeated inflammatory measurements, objective sleep and appropriately selected neurocognitive or imaging tasks. Patients with inflammatory flares could be compared with those having similarly severe sleep loss without inflammation. The model predicts a subgroup-specific temporal mediation pattern; failure to observe one would weaken the direct neuroimmune version.

The hypothesis needs prospective coupling of inflammatory changes with validated dissociation measures and independent neural or interoceptive markers, controlling for sleep and medication exposure. If the symptoms persist without any measurable immune change, the claim should narrow to selected subgroups.

Decisive comparisons and limits of inference

A strong design would prospectively capture episodes instead of relying on distant recall. Participants could report changes in embodiment and derealization with time stamps while researchers assess sleep, inflammatory activity and other relevant medical states. Controls would include individuals with comparable sleep deprivation but without an inflammatory flare and individuals with inflammation who do not experience altered self. Tasks probing bodily ownership, interoceptive accuracy or multisensory integration may provide functional endpoints beyond a mood questionnaire, although their interpretation is still complex. To support a causal neuroimmune pathway, the proposed immune exposure must precede the predicted perceptual alteration and a measured mediator must connect them. If symptoms occur independently of inflammatory change, the thesis weakens for that subgroup. A

separate issue is magnitude: minor changes in ordinary cytokine variation should not be assumed sufficient to reorganize self-experience. The strongest version of the hypothesis challenges a narrow psychological interpretation of dissociation while preserving the reality that perception can be altered through different routes. The body's immune state may sometimes become one of the inputs from which the sense of self is constructed; demonstrating when it matters is the scientific task.

A second prediction from the original model

A biologically specific subgroup would require convergent evidence in immune measures, interoceptive processing and dissociative symptoms, not merely elevated C-reactive protein. Strong associations with sleep or trauma alone would favor narrower explanations.

Why the hypothesis matters if correct

The point of this paper is more unusual than saying inflammation worsens mood. It asks whether the very experience of being embodied—feeling present, familiar and situated in one's body—can change with inflammatory and sleep-related physiology. If supported, this would move a seemingly philosophical complaint toward specific interoceptive and neural mechanisms. The strongest phenotype would involve objectively characterized inflammatory episodes that precede distinctive alterations in self-experience, rather than a single elevated blood marker discovered after symptoms. The hypothesis must also compete with panic-related depersonalization, seizures, medication effects and ordinary sleep disruption. These alternatives are not embarrassments; they define what a neuroimmune account would have to explain better. A convincing study could compare inflammatory flare periods with noninflammatory periods in the same person and assess multisensory and bodily-ownership tasks alongside symptom reports. The novel causal claim would be limited but striking: some alterations of self-experience may partly reflect the physiological state of the networks constructing the bodily self.

The clinical and conceptual consequence

The unsettling idea is that the sense of "I" is partly a physiological achievement. If inflammation can disturb the bodily signals from which that achievement is constructed, some apparently existential symptoms would have a measurable somatic component. This remains a research claim, not a license to self-diagnose inflammation from dissociation.

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Source bibliography: verification pending

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- Seth AK, Critchley HD. Extending predictive processing to the body: emotion as interoceptive inference. Behav Brain Sci. 2013.

Editorial and clinical boundaries

This paper advances a causal thesis and identifies observations that would support or defeat it. It does not establish a new diagnosis or treatment, and it should not be used to discontinue medically indicated care. Bibliographic entries inherited from the original edition have not all been independently verified.