

The Psychiatric Symptom as a Biological Signal: Toward an Integrated Medicine of Mind and Body

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Abstract

The distinction between psychiatric and medical disease reflects practical disciplinary history, not a separation within the patient. Thoughts, emotions, motivation, perception and behavior arise from neural circuits continuously influenced by endocrine, metabolic, immune and environmental conditions. Yet the existence of these interactions does not establish that every psychiatric symptom has a reversible peripheral cause. This concluding paper proposes a model of explanatory pluralism: use molecular causation where the evidence is specific, systems reasoning where interactions matter and direct psychiatric care where it is indicated.

From description to mechanism

Psychiatric diagnosis can be valuable even when its full mechanism is unknown. A diagnosis may summarize recurring symptom patterns, anticipated risks and treatments that help. It becomes incomplete when mistaken for a total causal account. A patient with hallucinations from autoimmune encephalitis, another with psychosis during severe hypothyroidism and a third with a primary psychotic disorder may share a descriptive symptom but not an underlying disease process. Precision begins by distinguishing phenotype from etiology.

A hierarchy of causal confidence

At one end are well-characterized syndromes such as electrolyte encephalopathy, Wilson disease, certain porphyrias and autoimmune encephalitis, where biochemical or pathological mechanisms can be specifically investigated. In the middle are complex associations such as inflammatory variation in depression, where causal effects may depend on phenotype and comorbidity. At the speculative end are proposals that ordinary dietary oxalate causes psychiatric disorders through unproven pathways. All three categories can motivate questions, but they cannot be written as though they share the same evidentiary status.

Why the oxalate question matters despite uncertainty

The oxalate series provides a test of intellectual discipline. It contains established intestinal chemistry, crystallization and renal injury; severe systemic oxalosis can involve extra-renal tissues; rare neurological consequences can occur through serious metabolic illness. These facts suggest a narrow inquiry into susceptible hosts. They do not prove that dietary oxalate is a common cause of psychosis, postpartum psychiatric illness, autism or depression. The Institute should treat that distinction as a virtue of frontier research rather than a concession to orthodoxy.

Treatment should follow the best causal account available

A patient with acute encephalopathy needs urgent medical care; a patient with confirmed endocrine disease needs appropriate treatment; a patient with severe bipolar disorder needs psychiatric care even if additional metabolic questions remain. Psychotherapy, pharmacology, sleep treatment, nutritional intervention and medical investigation are not philosophical rival camps. Their value depends on what they address in a particular patient's illness and how benefits and harms are measured over time.

What the Institute can contribute

An open-access research library can distinguish hypothesis, documented mechanism, contradictory evidence and practical implications more clearly than popular health commentary often does. The purpose of provocative papers is not to replace one dogma with another, but to show where current descriptions fail to explain and where new explanations could earn credibility. Each proposition should name the competing causal paths that might defeat it. An institute becomes trustworthy by preserving a record not only of promising hypotheses but of revisions and falsifications.

Conclusion

Mental illness is biological without being reducible to any one biological pathway. The path toward better explanation is neither wholesale rejection of psychiatric diagnoses nor uncritical acceptance of them as complete causes. It is disciplined integration: follow evidence where it is strong, investigate gaps where it is weak, and protect patients from the harms of both neglect and overclaiming.

Selected readings

- Pollak et al., Autoimmune psychosis consensus
- Bousman et al., CPIC pharmacogenetics
- Witting et al., Enteric Hyperoxaluria
- Mulay et al., NLRP3 renal injury