

The Biological Differential Diagnosis of Psychosis

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Abstract

Psychosis describes disturbances in perception, belief or thought; it does not by itself establish etiology. This paper proposes an etiologically layered differential that includes primary psychiatric disorders alongside endocrine crises, delirium, autoimmune encephalitis, medication effects, intoxication, neurologic disease and rare inborn metabolic defects. The point is clinical precision, not dismissal of the reality or severity of psychiatric illness.

The Clinical Question and Its Context

The first distinction is between acute delirium with fluctuating attention and a primarily psychiatric psychosis with relatively preserved awareness. Fever, seizures, autonomic instability, new focal signs and rapid cognitive decline create a different workup from slowly progressive isolated symptoms. Thyroid disease, severe glycemic disturbances, electrolyte disorders, vitamin deficiencies and medication effects may contribute in selected patients. Autoimmune encephalitis is especially important when psychiatric change clusters with seizures, dyskinesias or abnormal consciousness.

Proposed Biological or Explanatory Model

A terrain perspective can be operational if it asks about exposures, time course, neurologic examination and objective organ dysfunction rather than attributing the experience to an undefined “toxic burden.” A structured approach maps syndrome, onset, medication chronology, accompanying neurological features, laboratory abnormalities and imaging/EEG relevance. Each proposed explanation should account for the observed pattern more successfully than alternatives.

Mechanism-Specific Interpretation

The critical distinction is between a thorough initial medical assessment and indiscriminate “root cause” testing. For an acutely altered person, orientation, attention, vital signs, substance exposure, medication chronology and neurological findings may be more urgent than an elaborate micronutrient panel. Autoimmune encephalitis enters the differential when the syndrome includes characteristic temporal progression and neurological phenomena, but isolated anxiety or depressive symptoms do not justify labeling a patient with encephalitis. Rare metabolic disorders become more plausible when symptoms cluster with systemic findings. This is a probabilistic process, not a hunt for one universal overlooked toxin.

Competing Explanations and Limits of Inference

Overtesting every patient can create false-positive findings, delay appropriate psychiatric treatment and increase cost. The reverse error is premature closure after assigning a psychiatric label. Neither all psychoses nor all psychiatric medications are manifestations of hidden metabolic failure; evidence supports multiple causal pathways and meaningful benefit from established psychiatric treatments.

Discriminating Observations and Evidence Thresholds

The model would improve clinical practice if it increased recognition of treatable organic conditions without postponing appropriate psychiatric care. Its failure modes are equally important: overtesting, incidental abnormalities, false diagnoses and delay of treatment for mania, psychosis or suicidal risk. The integrity of the framework is measured by what it helps rule out as well as what it helps discover.

Implications for Interpretation and Future Inquiry

The question for publication is how to preserve urgent psychiatric care while recognizing biologically specific exceptions. Clinicians require red-flag algorithms and referral thresholds, not a new universal diagnostic panel. When self-harm, dangerous behavior, catatonia or impaired self-care occurs, emergency assessment takes precedence over speculative root-cause exploration.

Selected Evidence and Further Reading

1. Psychiatric signs and symptoms in treatable inborn errors of metabolism
2. Recommendations for detection and diagnosis of Niemann-Pick disease type C

Source lineage: Covenant Psychiatry; Depression research; complex psychiatric differential

Editorial note: Conceptual scientific commentary; not an original clinical trial, systematic review, peer-reviewed publication, or medical advice. Hypotheses are not established treatment effects.