

The Kidney–Brain–Mind Connection

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

The kidney governs more than excretion of creatinine. It helps regulate plasma tonicity, acid-base balance, erythropoietin, mineral metabolism and the circulating concentration of numerous solutes. The speculative frontier is whether modest or early disruptions in renal physiology can shape cognition and mood before overt uremic encephalopathy occurs.

The biological route

Chronic kidney disease is associated with cognitive dysfunction through combinations of vascular injury, anemia, disturbed mineral metabolism, retained solutes, inflammation, sleep disorders and medication exposure. Certain uremic toxins have mechanistic plausibility as neural stressors. In contrast, dietary oxalate is not established as an independent cause of common psychiatric syndromes in people with normal renal function.

An illustrative scenario

A person with declining kidney function and cognitive complaints may also have vascular disease, anemia or sleep apnea. If cognitive performance improves after addressing one of those conditions, that finding matters, but it need not identify an unnamed retained solute. Conversely, a specific metabolite candidate should demonstrate biological access, sufficient concentration and a reproducible effect. The kidney–brain axis is a family of mechanisms, not a single switch.

Alternative explanations and limits

The co-occurrence of kidney abnormalities and depressed mood could reflect shared diabetes, hypertension, socioeconomic stress, chronic pain or illness burden. An association between eGFR and cognition does not identify a specific causal metabolite or show reversibility after its removal.

What would change the conclusion?

A useful conceptual investigation would compare filtration markers, urine measures, albuminuria, vascular biomarkers and time course of cognitive change. A particular solute mechanism needs exposure, dose, biological access to neural tissue and a disease-specific effect—not just statistical association.

The distinctive prediction

A convincing early kidney–brain pathway needs to predict cognition from an identified retained solute, mineral disturbance or vascular mechanism rather than eGFR alone. The strongest candidate should be measurable before overt encephalopathy and remain relevant after common confounders are controlled.

Clinical interpretation

People with acute confusion, seizures, marked renal dysfunction or electrolyte changes require timely clinical evaluation. This hypothesis should encourage detection of defined treatable conditions rather than the marketing of unproven renal-cleansing products.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Joshi R, Chou S-Y. Beer Potomania: A View on the Dynamic Process of Developing Hyponatremia. *Cureus*. 2018.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC6150768/>

Angleys H et al. The effects of capillary transit time heterogeneity on brain oxygenation. *J Cereb Blood Flow Metab*. 2015.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC4420854/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.