

Mineral Balance and Neural Excitability: Could Oxalate Contribute Indirectly?

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Abstract

Calcium and magnesium participate in neuronal excitability, synaptic transmission and cellular signaling. This makes mineral physiology an attractive bridge between dietary oxalate and mental symptoms. Yet most popular arguments omit the crucial difference between binding calcium in food, changing urinary stone risk and changing ionized calcium in blood. We assess the plausible indirect mechanisms and the frequent inferential error that follows.

A calcium story with three compartments

Calcium in the intestinal lumen can bind oxalate and reduce absorption. Calcium in urine can combine with oxalate to form kidney stones. Ionized calcium in plasma is regulated by parathyroid hormone, vitamin D and the kidneys, and influences neuromuscular excitability. These processes interact through physiology but are not interchangeable. The body does not allow meal-by-meal fluctuations in intestinal free calcium to translate freely into corresponding changes in brain extracellular calcium.

What mineral disorders actually do

Marked hypocalcemia can cause paresthesias, tetany, seizures and neuropsychiatric changes. Hypercalcemia, especially in severe parathyroid or malignant disease, may produce fatigue, impaired concentration, mood changes or delirium. Significant magnesium abnormalities likewise affect neuromuscular and cardiac function. These examples establish that mental symptoms can have mineral causes. They do not establish that dietary oxalate regularly provokes these abnormalities. Severe ethylene glycol intoxication and advanced kidney failure are special clinical settings, not representative exposures.

The role of kidney function

Renal disease can disturb calcium, phosphorus, vitamin D and acid-base homeostasis. That offers a credible indirect path from severe oxalate-related nephropathy to altered neurological function. But the risk depends on the actual extent of renal impairment and mineral derangement, not on a theoretical dietary ratio alone. Symptoms without abnormal systemic physiology require competing explanations rather than an assumed hidden calcium depletion.

A useful boundary

The compelling hypothesis is that oxalate can change mineral handling in defined intestinal and renal contexts, which may influence systemic physiology if disease becomes sufficiently severe. The unsupported hypothesis is that normal oxalate intake continuously removes calcium from the brain and thereby causes anxiety or psychosis. Any persuasive account would require appropriately timed ionized calcium measurements, kidney assessment, exposure context and a neurologically coherent clinical presentation.

Conclusion

Mineral physiology links the intestine, kidney and nervous system through established mechanisms. Scientific rigor consists in identifying the circumstances in which the link becomes clinically active, not equating the presence of oxalate in food with a disturbed brain.

Selected readings

- Witting et al., Enteric Hyperoxaluria
- Primary Hyperparathyroidism and Psychiatric Sequelae