

The Liver That Looks Normal but Cannot Keep Up

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Routine aminotransferases are mainly markers of hepatocellular injury, not complete readouts of transporter activity, enzymatic flux, biliary excretion or metabolic reserve. This essay asks whether significant functional bottlenecks may exist despite unremarkable enzyme measurements—and when that possibility is clinically established versus conjectural.

The biological route

Bile formation requires canalicular transporters, hepatocyte polarity, energy-dependent transport and coordinated uptake/excretion systems. Drug metabolism involves multiple enzymes whose capacity can change with genotype, inflammation, interactions and liver disease. Albumin, bilirubin, INR and disease-specific tests offer different information from AST and ALT. The proposed systems lens emphasizes flux and throughput, not vague “liver congestion.”

An illustrative scenario

Consider a patient taking a drug cleared by a hepatic transporter who has normal AST and ALT. Drug exposure can still vary because of interactions, transporter genetics or changes in organ function. That is a tractable pharmacological question. It is very different from asserting that the patient has unspecified “liver congestion” because of a headache or food intolerance. Good terrain reasoning must preserve that difference.

Alternative explanations and limits

Normal enzymes with symptoms are common and rarely prove occult hepatic disease. Symptoms attributed to poor detoxification might instead reflect gastrointestinal, endocrine, neurological or psychological conditions. Claims about unidentified circulating toxins without analytical measurements cannot substantiate the mechanism.

What would change the conclusion?

Discriminating evidence would be objective abnormalities in relevant transport, bile formation or drug metabolism pathways plus clinical response to correction of the demonstrated problem. Where tests and physiology remain normal, the hypothesis should not be used as a catch-all diagnosis.

The distinctive prediction

The claim becomes medically meaningful only when the suspected bottleneck is named: which enzyme, transporter, synthetic function or excretion route is impaired? Vague “congestion” without a measured pathway offers no testable explanation.

Clinical interpretation

The value is refined test interpretation: understand what a measurement assesses and what it does not. It does not justify liver flushing, indiscriminate supplements or stopping effective medication.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Jansen A et al. Increased levels of persistent organic pollutants in serum one year after a great weight loss in humans. Sci Total Environ. 2018.

<https://pubmed.ncbi.nlm.nih.gov/29890598/>

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