

The Hormones That Govern Bile

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Abstract

Bile physiology and endocrine physiology influence each other, but claims that impaired bile directly causes generalized hormone deficiency go beyond current evidence. This paper maps known feedback between bile-acid synthesis, estrogen-related cholestasis, progesterone effects on motility, thyroid status and metabolic signaling. The aim is to identify precise directions of causality rather than invoke a single unexplained liver-hormone blockage.

The Clinical Question and Its Context

Bile acids are synthesized from cholesterol; steroid hormones also derive from cholesterol, but they do not require dietary animal fat to arrive as a special intact substrate. The liver regulates steroid metabolism and excretion, while estrogen-related states can alter hepatobiliary transport and gallstone risk. Thyroid function affects lipid metabolism and gastrointestinal physiology. FXR/FGF19 feedback coordinates bile-acid synthesis, while intestinal bile acids help absorb fat-soluble nutrients. These pathways intersect but operate through distinct enzymes and control loops.

Proposed Biological or Explanatory Model

The strongest hypothesis concerns people with measurable hepatobiliary dysfunction in whom endocrine signs coexist. Associations could reflect altered clearance of hormone metabolites, intestinal nutrient absorption, hepatic inflammation, or third factors such as obesity. Correct analysis requires distinguishing plasma hormone concentration, receptor activity, hormone production and metabolic elimination. A low measured hormone is not evidence of failure to digest dietary fat.

Mechanism-Specific Interpretation

The liver and endocrine glands share cholesterol-related chemistry but use it differently. Cholesterol is the precursor of steroid hormones through enzymatic pathways that operate in endocrine tissues, while bile-acid synthesis converts cholesterol into amphipathic compounds used for fat handling and signaling. Both processes are regulated, and the liver can clear hormones through conjugation and excretion. Thus a patient may have sufficient endogenous cholesterol for hormone synthesis despite consuming no cholesterol, or may have significant dyslipidemia while producing normal sex steroids. This distinction defeats simplistic analogies equating low dietary animal fat with hormone starvation.

Competing Explanations and Limits of Inference

The idea that vegan diets inherently prevent steroid hormone synthesis is inaccurate: humans can synthesize cholesterol and steroid hormones without consuming cholesterol. A vegan diet can still create nutrient-deficiency risks if poorly planned, while animal-fat excess can raise cardiovascular risk in some dietary contexts. Seed oils do not become an undifferentiated toxic class merely because they contain polyunsaturated fats; oxidation, food processing, energy balance and fatty-acid substitution matter.

Discriminating Observations and Evidence Thresholds

A clinically useful analysis should specify whether the hypothesized abnormality concerns hormone production, receptor signaling, tissue conversion, transport or elimination. Measurements that cannot distinguish these possibilities should not be presented as definitive. Pregnancy-associated cholestatic syndromes and thyroid-related changes in lipid metabolism offer concrete starting points, but cannot establish a universal nutrition–psychosis causal chain.

Implications for Interpretation and Future Inquiry

This paper should make the hormone–bile axis comprehensible through diagrams of directionality and compartments. It can legitimately argue against dismissing liver and intestinal disease in endocrine assessment, without claiming that supplementation with saturated fats treats psychiatric illness or corrects all hormone disorders.

Selected Evidence and Further Reading

1. [Recent Advances in the Digestive, Metabolic and Therapeutic Effects of FXR and FGF19](#)
2. [Bile acids: regulation of synthesis](#)

Source lineage: Bile–fat–hormone investigation; Absurd Diet

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