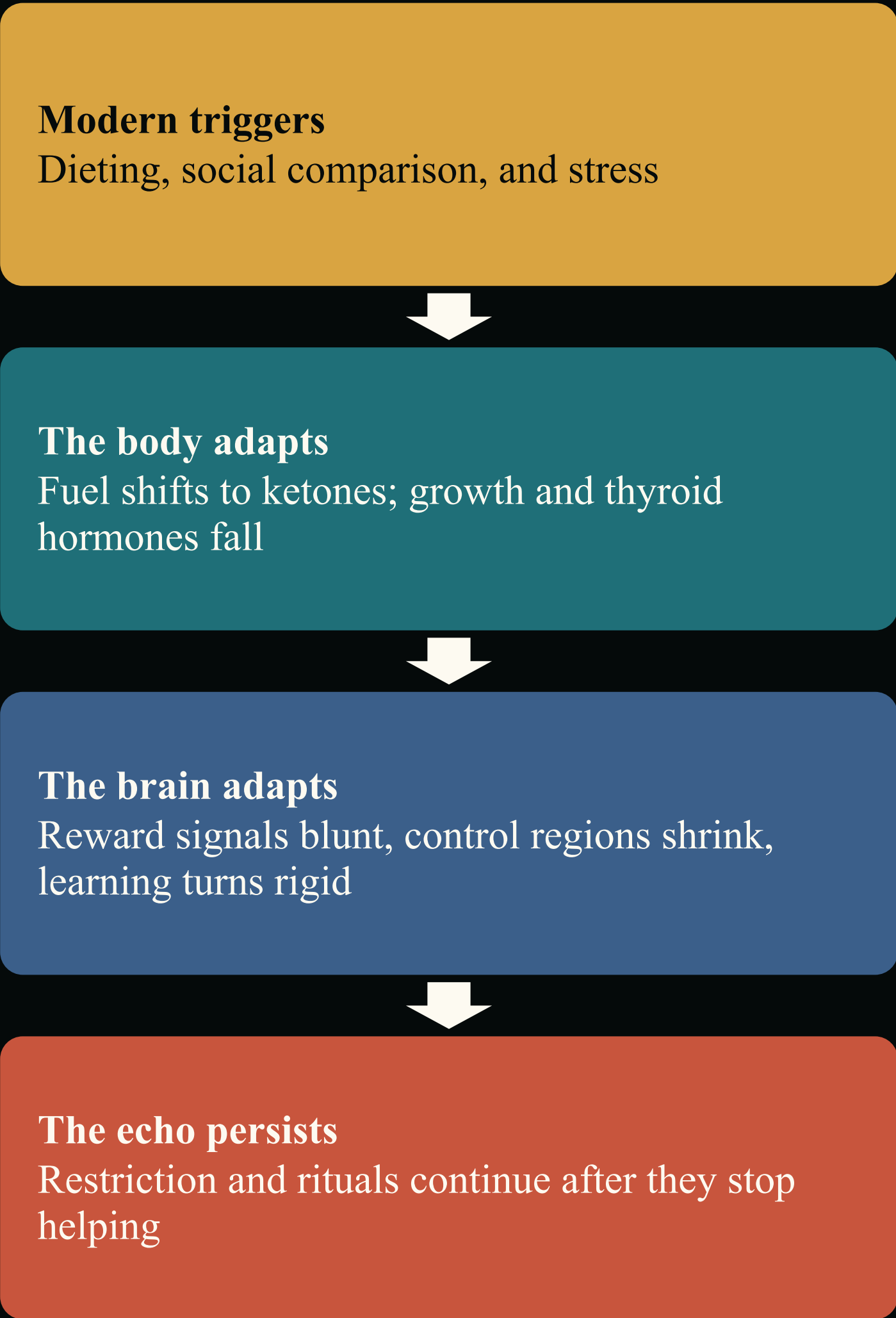


Abstract

Anorexia nervosa (AN) has one of the highest death rates of any psychiatric illness, and social and psychological explanations leave its persistence unexplained. This review asks how prolonged starvation, and the survival responses humans evolved for famine, shape the brain in AN. It proposes an “evolutionary echo”: ancient starvation responses, set off by modern pressures, that continue after they stop helping. Across metabolic, imaging, and learning studies, starvation changes brain fuel use, reward signaling, and flexibility, and many changes ease with refeeding.

Figure 1. The evolutionary echo model in four steps



Restriction deepens the starved state, which feeds the cycle again.

Introduction

Anorexia nervosa (AN) has one of the highest death rates of any psychiatric illness [1]. Social pressure and perfectionism can explain why someone starts dieting, but not why the illness lasts [2]. Human physiology evolved under fluctuating food availability, and starvation-response signaling is conserved across species [3, 4]. Responses that help conserve energy during famine may become harmful when restriction is sustained in a modern environment of food availability, social comparison, and weight-related pressure. The main question is how prolonged starvation, and the responses evolved to survive it, shape the brain in AN. The hypothesis is an “evolutionary echo”: ancient famine responses, triggered by modern pressures, that persist after they stop being useful.

Methods

PubMed, Google Scholar, and ScienceDirect were searched to find peer-reviewed human and mammal studies (2000–2024) using terms related to starvation neurobiology, anorexia nervosa and the brain, dopamine, and evolutionary explanations. Foundational studies were also identified through reference-list tracking. Studies of only psychological or social causes were excluded.



Table 1. Two evolutionary explanations tested against the evidence

Idea	Core claim	Fit with our findings
Adapted to flee famine [2]	Food restriction, denial of hunger, and restlessness helped ancestral foragers leave depleted land.	Fits blunted hunger and reward signals. Restlessness was not examined in the studies reviewed.
Evolutionary mismatch [1, 4]	Famine-era responses clash with modern food abundance and constant social comparison.	Explains why the responses can persist, and even start, where food is plentiful.

Reproductive suppression and sexual competition hypotheses were considered but not developed here.

Results

Brain fuel use changes rapidly. After 3.5 days of fasting, healthy volunteers’ brains used 26% less glucose and took up 13 times more ketones. Ketones provide an alternative fuel during prolonged fasting [5].

Brain-volume changes are associated with undernutrition. Structural MRI studies report lower gray-matter volume in underweight AN. Much of the volume difference improves with weight restoration, supporting a substantial state-related component [6, 7].

Starvation can alter costly learning in model organisms. Experimental work in flies found that starvation can suppress neural processes involved in long-term memory formation [10, 11]. This supports the plausibility that energy scarcity changes which neural processes are prioritized.

Figure 2. The brain’s fuel switch after 3.5 days of fasting

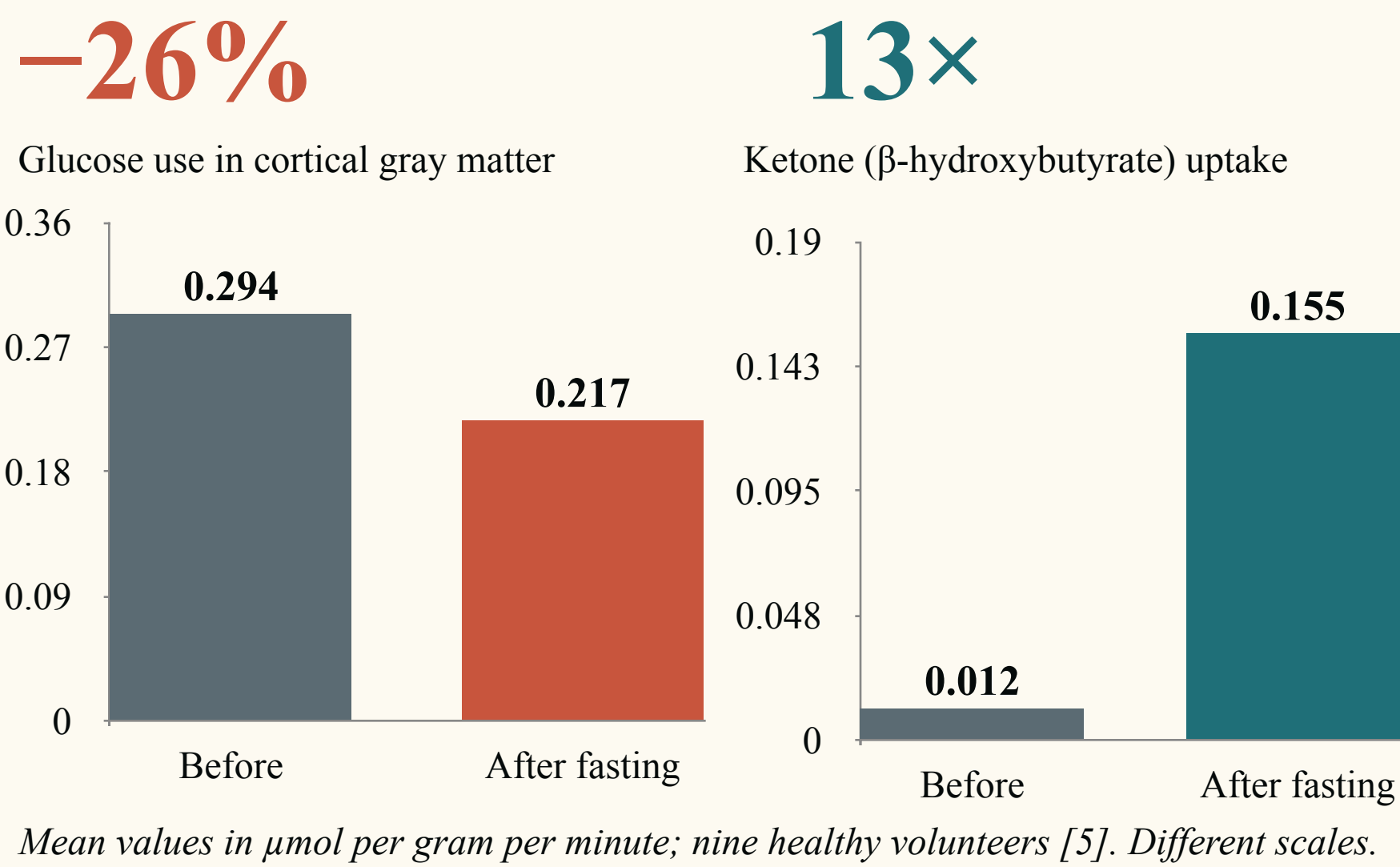


Table 2. Brain regions that change in underweight AN

Region (job)	Underweight	After refeeding
Insula (senses hunger and body signals)	Less gray matter	Mostly recovers
Cingulate (catches errors and conflicts)	Less gray matter	Mostly recovers
Prefrontal cortex (plans, self-control)	Less gray matter	Mostly recovers
White matter (wiring between regions)	Findings vary	Unclear

Some well-controlled studies find lasting differences after long-term recovery
Sources: [6, 7].

Discussion

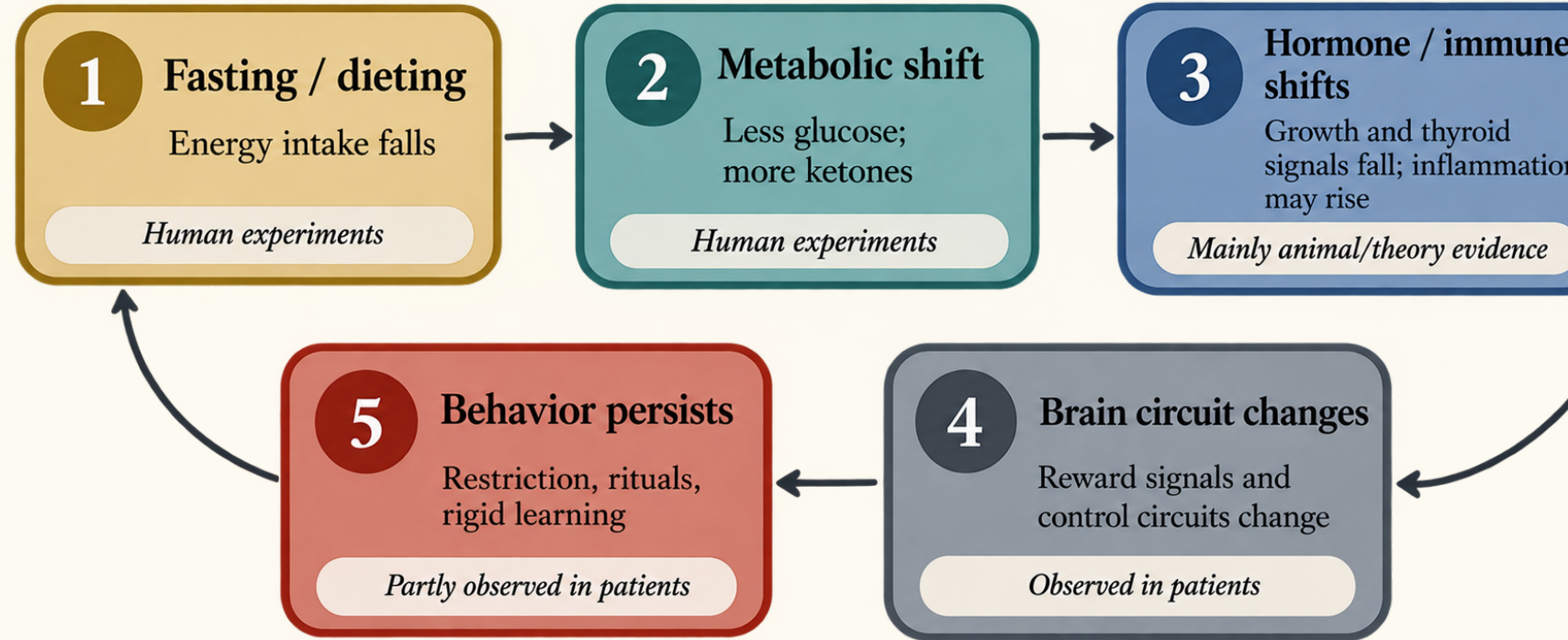
One story across studies. The results fit a brain that cuts costs during famine. Fasting changes its fuel [5], and in fruit flies, starvation switches off the costly formation of some long-term memories [10]. A brain on a tight budget may favor fixed, low-effort rules over flexible learning. In Figure 1, only the first link has been tested by experiment; the rest are inferred.

Where the evidence is firm. Fuel use, brain shrinkage, and its recovery come from human imaging [5, 6, 7]. Hormone, immune, and evolutionary links rest mostly on animal work and theory [3, 1].

Two evolutionary readings. Under the flee-famine idea, hunger denial and restlessness once helped people leave depleted land [2]. Under the mismatch idea, dieting and social comparison trigger the same responses where food is plentiful [1].

What the echo model adds. The model links starvation-related metabolic changes, brain differences, and altered learning into a feedback loop. This framework connects findings across studies, but remains a hypothesis: the evidence for each link varies in strength, and the full sequence has not been established experimentally.

Figure 3. The evolutionary echo, graded by evidence



Conclusion

The literature supports a multidimensional account of AN in which starvation-related metabolic changes, neural differences, and altered learning may interact. The evolutionary-echo model organizes these findings into a testable framework, but should be treated as a hypothesis rather than proof that AN is an adaptation. The next step is longitudinal research that distinguishes direct effects of undernutrition from pre-existing vulnerability and modern triggers.

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