Behavioural Stability and Energy Availability: Exploring the Relationship Between EPK, Compensatory Eating Behaviour, and Long-Term Dietary Adherence

Billy Craig, MSc
Independent Researcher

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Status: conceptual/hypothesis-generating working paper. Not peer reviewed. This revision adds primary-study appraisal and prospective tests; it reports no new participant data.

Abstract

Dietary relapse is commonly framed as a failure of motivation or restraint. This paper explores a complementary hypothesis: behavioural stability during dieting may itself be influenced by physiological energy availability. The hypothesis builds directly on the MSc observation that intake varied during attempted restriction, although the primary statistical analyses were nonsignificant. This revision separates dietary intake from cellular utilisation and proposes prospective comparison of EPK with its component measurements; the added mechanistic evidence does not validate EPK.

Behavioural Instability

Dietary relapse is often described in moral or motivational terms. These factors can matter, but they do not explain why restriction itself can increase hunger, food salience and preoccupation. The Minnesota starvation data demonstrate that profound behavioural changes can arise in motivated people when energy availability is reduced.5

The MSc pattern is better described as oscillation than simple failure. Participants repeatedly moved above and below targets. A large deficit may increase compensatory drive; increased intake reduces the deficit; concern about the increase then prompts renewed restriction. Over time, mean intake can drift closer to need while daily behaviour appears highly inconsistent.6

Appetite as Behavioural Pressure

Leptin, ghrelin and hypothalamic signalling provide a mechanistic bridge between energy state and behaviour. Weight loss can produce persistent hormonal changes that favour hunger. The subjective experience may be cravings, food thoughts or declining restraint, but the behavioural pressure has a physiological component. This does not remove agency; it recognises that physiological state changes the difficulty of the behavioural task.[7,8,9,10,11,12,13]

EPK Interpretation

EPK proposes that behavioural consistency itself may be an outcome worth monitoring. Lower EPK states may predict greater intake variability, stronger food preoccupation, poorer sleep and reduced recovery. If so, escalating restriction after overeating could amplify the cycle. Whether a strategy focused on intake and recovery improves adherence requires a controlled comparison.

Sleep as a candidate modifier

Sarnataro et al. linked sleep pressure to mitochondrial processes in fruit-fly sleep-control neurons [R1]. This is an indirect lead for behavioural research, not evidence that low EPK causes cravings or that sleep problems diagnose mitochondrial failure. Its value here is to motivate measurement of sleep alongside eating behaviour, while established appetite pathways remain a separate evidence base.

Human evidence from the PubMed search

The two-year CALERIE 2 trial reported improvements in some mood and quality-of-life outcomes with calorie restriction, rather than the predicted general deterioration. Structured moderate restriction in selected healthy adults should be distinguished from severe low availability in athletes or symptomatic undernutrition. [H1]

These studies test energy restriction or sports energy availability, not EPK as an intake-to-total-mass ratio. Their populations and intervention durations bound interpretation; none validates an EPK threshold. The human literature requires the framework to allow beneficial, adverse and null responses in different contexts.

A prospective test of behavioural stability

Does EPK predict functional outcomes beyond total energy intake, body composition, carbohydrate availability and activity, and could fuel switching or mitochondrial function explain any association?

For prospective testing, EPK is defined here as mean dietary energy intake in kcal per day divided by body mass in kg, expressed as kcal/kg/day. This intake ratio is not the sports energy-availability measure, which subtracts exercise energy expenditure from intake and divides by fat-free mass. Exercise expenditure, fat-free mass, carbohydrate intake and weight trajectory must therefore be recorded separately. No diagnostic EPK threshold is established by this revision.

Measure mean EPK during a baseline period and eating variability, food preoccupation and adherence in a separate subsequent period. Calculating both EPK and intake variability from the same days can create mathematical coupling; a future outcome window reduces that problem. Preregister one primary behavioural outcome and assess sleep, mood, medication, restraint, food access and intended deficit as potential confounders or modifiers.

EPK is calculated from intake and mass and therefore contains no information absent from those inputs. Its value must be judged as a useful summary, not an independent biological variable. Compare a preregistered EPK model with a flexible model of energy intake and mass, including relevant nonlinear terms and interactions, under the same validation procedure. Adding the ratio to a simple linear baseline would not by itself establish unique physiological information. Report out-of-sample prediction error, calibration and uncertainty; favour the simpler model if performance is comparable.

An association with subsequent behaviour would warrant replication. It would not show that mitochondrial changes mediate eating behaviour unless those changes were measured with the required temporal ordering. The MSc pattern is exploratory and its primary statistical tests were nonsignificant. A trial of an EPK-guided strategy would require its own design and cannot be inferred from this literature.

Conclusion

Eating behaviour remains multifactorial and the proposed relationship is not established causally. Nevertheless, the model creates testable predictions: prospective research can examine whether EPK predicts day-to-day intake variance and adherence independently of baseline restraint, body composition and intended deficit.

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Primary studies added in version 1.1

[R1] Sarnataro R, Velasco CD, Monaco N, Kempf A, Miesenböck G. Mitochondrial origins of the pressure to sleep. Nature. 2025;645:722–728. https://doi.org/10.1038/s41 586-025-09 261-y

Human studies added in version 1.1

[H1] Martin CK, et al. Effect of Calorie Restriction on Mood, Quality of Life, Sleep, and Sexual Function in Healthy Nonobese Adults: The CALERIE 2 Randomized Clinical Trial. JAMA Intern Med. 2016;176:743–752. doi:10.1001/jamainternmed.2016.1189. https://pubmed.ncbi.nlm.nih.gov/27 136 347/

Publication Note

This manuscript is part of the EPK (Energy Per Kilogram) paper series developed by Billy Craig, MSc. The paper should be cited as a working/preprint paper until peer-reviewed publication. EPK-specific propositions are presented as hypotheses where they extend beyond the cited evidence.

Revision record

Version 1.1 adds the mechanistic discussion and prospective study design above. The original numbered bibliography remains distinct from the new R-prefixed mechanistic and H-prefixed human-study citations. Studies discovered through haidut.me are cited to their original research publications; the blog commentary is not treated as primary evidence.

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