

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

Billy Craig | Independent Researcher

Version 1.2 | 2 October 2026 | Conceptual working preprint - not peer reviewed

DOI: [10.5281/zenodo.23106398](https://doi.org/10.5281/zenodo.23106398)

Abstract

This paper examines whether hunger, stress, sleep and the difficulty of sustaining dietary targets are useful outcomes in EPK research. Restriction can provoke compensatory responses, but effects depend on dose, duration, context and individual circumstances. Selected successful programme members cannot establish population-wide maintenance rates; sustained losses in controlled programmes also require acknowledgment. The revision integrates Minnesota recovery evidence, human stress and sleep studies, and an explicit distinction between psychological burden and energy intake. It proposes a prospective behavioural study and a pragmatic intervention evaluation without assuming that behavioural variability proves a biological deficit.

Scope and evidence selection

This is a targeted narrative synthesis and hypothesis paper, updated through 2 October 2026. Sources were selected from the existing EPK evidence library, primary publications and the recent companion reviews. Selection was question-led and not systematic; no pooled effect, formal risk-of-bias score or new participant dataset is reported. Bibliographic records and available abstracts/full texts were used. Abstract-only access limits appraisal of allocation, missing data, confounding and adverse events. Supporting, contrary and null findings are retained.

1. Behavioural stability as a research question

EPK is defined as mean dietary energy intake in kcal/day divided by body mass in kg. It is an intake-to-total-mass ratio, not a direct measure of energy balance, ATP production or nutritional sufficiency. Sports energy availability instead subtracts exercise expenditure from intake and divides by fat-free mass. Those quantities must be recorded separately; sports thresholds cannot be transferred numerically to EPK. [1]

Behavioural stability should mean a specified, useful outcome such as reduced distress around eating or sustained engagement with an agreed plan. It should not mean identical intake every day: changing training, appetite, work and social circumstances can make flexible eating appropriate. Variability becomes clinically or practically meaningful only in relation to the participant's goals and function.

The hypothesis is that some restrictive prescriptions increase hunger and food preoccupation sufficiently to undermine sustainable adherence. This does not exclude motivation, cost, access, habits or social pressures. The original MSc account inspired the hypothesis, but unavailable source data cannot establish its causal explanation or effect size.

2. Starvation and recovery

Minnesota's prolonged semi-starvation experiment showed major behavioural and physiological changes in selected young men. Its severity limits extrapolation to supported modest weight loss. Later reanalyses link post-starvation hyperphagia and body-composition recovery, motivating attention to recovery rather than treating all increased eating as misconduct. [2] [3]

A participant's difficulty following a target is an outcome to understand, not evidence of moral failure. Conversely, the observation that a person eats above an app target does not establish overeating relative to actual expenditure: both the target and the food log are estimates. An apparently oscillating deficit can arise partly from measurement error.

3. Hunger, stress and social context

Sumithran et al. found persistent appetite-related hormonal adaptations following weight loss. Polidori et al. estimated compensatory appetite feedback using a model-based approach. These complementary findings support investigating the effort needed to sustain restriction, rather than assuming motivation alone determines adherence. They do not quantify an EPK-specific effect. [4] [5]

Restriction and monitoring affected different stress outcomes in Tomiyama et al.; a cortisol result and a self-reported stress score should not be collapsed into one variable. Sapolsky's work further suggests that predictability, control and social circumstances influence stress responses. Food insecurity and fear of weight regain should be measured rather than inferred from intake. [6] [7]

Macronutrient composition can also alter cortisol metabolism. Stimson et al. reported altered regeneration and clearance without higher measured circulating or salivary cortisol. Hormone production, local tissue action and a single blood value are different observations. [8]

4. Sleep and mood are separate outcomes

The human sleep literature contains differing results: restriction-related sleep fragmentation in a small study of women, changes in sleep architecture under brief severe restriction, and no general deterioration in CALERIE 2. Sleep timing, menstrual phase, baseline sleep and achieved energy exposure need recording. [9] [10] [11]

Induced nocturnal hypoglycaemia caused awakening responses in an experimental study, but ordinary dietary restriction is not equivalent to insulin-induced hypoglycaemia. Waking hungry does not retrospectively diagnose a glucose emergency. [12]

Mouse high-fat-diet research links inflammatory lipid signalling with depressive-like behaviour. This is a mechanistic lead, not evidence that a specified human fat intake causes depression or that a supplement should be prescribed. Human mood outcomes need direct assessment. [13]

5. What does dietary failure mean?

Separate failure to reach a target, treatment withdrawal, regain after loss and inability to tolerate the intervention. Lowe et al.'s successful-completer sample answers a conditional maintenance question. Heshka et al.'s randomised comparison and the longer Look AHEAD follow-up answer different questions about programme outcomes. None licenses a single failure percentage for all diets. [14] [15] [16]

EPK should report who enrolled, who declined, who stopped, why they stopped and how missing outcomes affect conclusions. A favourable completer analysis should not replace intention-to-treat reporting. Financial incentives, ongoing support and selection of successful users should be explicit.

6. Prospective design and falsifiable predictions

Use an initial exposure window and a subsequent outcome window. Calculating mean intake and intake variability from the same days creates mathematical coupling. Include validated measures of dietary distress, hunger, fatigue and sleep, and preregister one primary endpoint. Avoid calling all fluctuations instability or interpreting compliance as intrinsically beneficial.

Because the ratio is determined entirely by intake and mass, it cannot contain information absent from those inputs. Its usefulness is a question of practical summarisation and predictive performance. Compare a prespecified EPK model with flexible models of intake, mass, fat-free mass and activity, including nonlinear terms where justified. Use the same held-out validation procedure, report calibration and uncertainty, and prefer the simpler model if performance is similar. A favourable comparison with a deliberately weak linear model would not establish a new biological quantity.

For an intervention, match contact time and monitoring burden across groups. Otherwise improved wellbeing might reflect greater support or less intrusive tracking rather than energy provision. Assess whether the proposed intervention changes actual intake and perceived burden before testing mediation. Define an improvement that participants regard as worthwhile and record adverse psychological responses.

A reduction in hunger without improved adherence would support only part of the hypothesis. Improved adherence with unchanged energy intake would favour a behavioural or support mechanism. No advantage over a credible comparator would count against claims of programme superiority. The 60-person feasibility proposal in Paper 1 is intended to refine these procedures, not establish effectiveness.

Conclusion

Behavioural outcomes give EPK an opportunity to test whether energy guidance can become easier to live with. Hunger and distress deserve respect, but neither validates an energy threshold. The programme must be judged by prespecified, participant-relevant outcomes and complete reporting.

Version and disclosure

Version 1.2 replaces unverified MSc inferences with a published-evidence rationale, adds dietary-maintenance denominators and stress/sleep counterevidence, and defines behavioural outcomes without equating flexible eating with failure. This version supersedes v1.1.1 for the current text; the earlier PDF and DOI remain an archive of that release. Reference numbering has been rebuilt for this version and must not be transferred from earlier editions.

The author develops EPK and has an intellectual and potential commercial interest in its evaluation. AI-assisted editorial drafting and source checking were used. This independent working paper has not undergone external peer review. No new human intervention or participant analysis is reported. Copyright 2026 Billy Craig. Author-owned text is licensed CC BY-ND 4.0; third-party material retains its own rights.

References

References are numbered in order of first citation. Companion EPK working papers provide context, not independent validation.

1. Mountjoy et al. (2023). IOC consensus statement on Relative Energy Deficiency in Sport.
<https://doi.org/10.1136/bjsports-2023-106994>
2. Kalm LM, Semba RD (2005). They starved so that others be better fed: remembering Ancel Keys and the Minnesota experiment. *Journal of Nutrition* 135:1347-1352.
<https://pubmed.ncbi.nlm.nih.gov/15930436/>
3. Dulloo, Jacquet and Girardier (1997). Poststarvation hyperphagia and body fat overshooting in humans: a role for feedback signals from lean and fat tissues.
<https://pubmed.ncbi.nlm.nih.gov/9062520/>
4. Long-term persistence of hormonal adaptations to weight loss
<https://pubmed.ncbi.nlm.nih.gov/22029981/>
5. How Strongly Does Appetite Counter Weight Loss? Quantification of the Feedback Control of Human Energy Intake
<https://pubmed.ncbi.nlm.nih.gov/27804272/>
6. Tomiyama et al. (2010). Low calorie dieting increases cortisol.
<https://pubmed.ncbi.nlm.nih.gov/20368473/>
7. Sapolsky (2005). The influence of social hierarchy on primate health.
<https://pubmed.ncbi.nlm.nih.gov/15860617/>
8. Stimson et al. (2007). Dietary macronutrient content alters cortisol metabolism independently of body weight changes in obese men. *JCEM* 92:4480-4484.
<https://doi.org/10.1210/jc.2007-0692>
9. Kim, Shekhar et al. (2025). Caloric Restriction, the Menstrual Cycle, and Sleep in Women Without Obesity.
<https://academic.oup.com/jcem/article/110/11/3108/8052714>
10. Collet et al. (2016). The Sleep/Wake Cycle is Directly Modulated by Changes in Energy Balance.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC4989258/>
11. Martin et al. (2016). Effect of Calorie Restriction on Mood, Quality of Life, Sleep, and Sexual Function in Healthy Nonobese Adults: The CALERIE 2 Randomized Clinical Trial.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC4905696/>
12. Schultes et al. (2007). Defective Awakening Response to Nocturnal Hypoglycemia in Patients with Type 1 Diabetes Mellitus.
<https://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.0040069>
13. Zhao et al. (2026). Aspirin alleviates long-term high-fat diet-induced depressive-like behavior in male mice via suppressing arachidonic acid-mediated microglial activation and neuroinflammation.
<https://www.nature.com/articles/s41380-026-03752-8>
14. Lowe et al. (2001). Weight-loss maintenance in overweight individuals one to five years following successful completion of a commercial weight loss program.
<https://pubmed.ncbi.nlm.nih.gov/11319628/>
15. Heshka et al. (2003). Weight loss with self-help compared with a structured commercial program: a randomized trial.
<https://pubmed.ncbi.nlm.nih.gov/12684357/>

16. Look AHEAD Research Group (2014). Eight-year weight losses with an intensive lifestyle intervention: the Look AHEAD study.
<https://pubmed.ncbi.nlm.nih.gov/24307184/>

Version history

v1.2 - 2 October 2026: scientific and editorial revision, refreshed reference mapping and integrated recent evidence. Unverified MSc quantitative claims are excluded from the evidential basis.

v1.1.1 - 11 September 2026: earlier licensing release. Archived DOI: 10.5281/zenodo.22709977. Earlier versions remain available through Zenodo.

Licence: Creative Commons Attribution-NoDerivatives 4.0 International. Third-party material is excluded.