

Relative Energy Deficiency Beyond Sport: Energy Availability, Compensatory Physiology, and Functional Decline in the General Population

Billy Craig, MSc
Independent Researcher

Working Paper | Version 1.1.1 | 11 September 2026

© 2026 Billy Craig. CC BY-ND 4.0. <https://creativecommons.org/licenses/by-nd/4.0/>

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 Not peer reviewed.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

RED-S demonstrates that inadequate energy availability can impair multiple systems in athletes. This paper asks whether analogous, milder energetic insufficiency may occur in the general population. The proposed term 'underpowered physiology' describes adaptive reductions in metabolic and behavioural output under chronic energetic stress.^{1,2,3,4} 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.

From RED-S to General Physiology

The tissues affected by low energy availability in athletes also exist in non-athletes. The unresolved question is not whether general humans need energy, but whether sports-derived thresholds and syndromes translate outside sport. EPK does not assume direct translation; it uses RED-S as physiological precedent for investigating energy sufficiency in the general population.^{1,2,3,4}

Underpowered Physiology

Repeated dieting can reduce expenditure, spontaneous movement and recovery while increasing hunger. Underpowered physiology describes the resulting pattern of reduced output and diminished reserve. Fatigue, low movement tolerance, poor sleep and behavioural inconsistency are nonspecific, but when they cluster during prolonged restriction they may justify considering energetic state rather than assuming poor motivation alone.^{5,6}

Obesity and Physiological Availability

A central challenge is that obesity appears incompatible with energy deficiency. EPK distinguishes stored energy from the current capacity to mobilise and use energy while maintaining normal physiological output. It does not call obesity starvation. It proposes that substantial adipose stores can coexist with low spontaneous movement, poor recovery, impaired metabolic flexibility and strong compensatory responses to further restriction.^{5,6,7,8,9}

Public Health Implications

If low energetic availability can contribute to declining movement and recovery outside sport, then repeated prescriptions to eat less and move more may become self-defeating in some already underpowered individuals. The alternative is not unrestricted intake, but restoration of physiological sufficiency followed by progressive rebuilding of movement capacity.

Distinguishing insufficient intake from impaired utilisation

Koves et al. studied mitochondrial overload in experimental insulin resistance [R1]. This is a useful counterexample to interpreting every functional impairment as insufficient intake: metabolic dysfunction can accompany excess substrate delivery. The cardiac model of Kim et al. similarly illustrates a specific utilisation defect without validating a dietary energy threshold [R2].

The EPK hypothesis must allow low dietary supply, excessive supply and impaired utilisation to occur in different combinations. Adiposity does not diagnose low energy availability, and a low intake-to-total-mass ratio can partly reflect the denominator. Fat-free mass, energy expenditure and weight trajectory are necessary for interpreting the exposure. Symptoms such as fatigue also have many causes and cannot establish RED-S or a general-population equivalent.

Human evidence from the PubMed search

In a six-month randomized study of 36 adults, calorie restriction increased muscle mitochondrial DNA content while key mitochondrial enzyme activities remained unchanged. The result separates mitochondrial quantity from measured functional capacity and challenges a universal decline-with-restriction model. [H1]

In a small 16-week comparison of weight loss (7 participants) and exercise (10), insulin sensitivity improved similarly while mitochondrial adaptations differed. This shows why insulin sensitivity and mitochondrial capacity should not be treated as equivalent outcomes. [H2]

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 discriminating study design

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.

Recruit across body-composition and activity ranges and distinguish weight-stable participants from those losing weight. Measure sports-style energy availability separately where feasible rather than importing its thresholds into EPK. Analyse whether any EPK association remains across strata and whether it is explained by fat-free mass or energy balance. Prespecify alternative hypotheses, including lipid oversupply and illness-related reductions in both intake and function.

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.

A study that finds low EPK only among people with greater adiposity, with no additional functional association after appropriate modelling, would not validate energy deficiency. Mechanistic measurements can test whether utilisation differs, but cannot by themselves identify the dietary cause of that difference. No clinical diagnostic category or general-population treatment threshold is established here.

Conclusion

The concept of relative energy deficiency beyond sport is hypothesis-generating. Direct research is required before clinical thresholds or diagnostic language can be justified.

References

1. Loucks AB, Kiens B, Wright HH. Energy availability in athletes. *J Sports Sci.* 2011;29(Suppl 1):S7-S15. doi:10.1080/02640414.2011.588958.
2. Mountjoy M, Sundgot-Borgen J, Burke L, et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. *Br J Sports Med.* 2018;52(11):687-697. doi:10.1136/bjsports-2018-099193.
3. Logue DM, Madigan SM, Delahunt E, et al. Low energy availability in athletes: a review of prevalence, dietary patterns, physiological health, and sports performance. *Sports Med.* 2018;48(1):73-96. doi:10.1007/s40279-017-0790-3.
4. Melin A, Tornberg ÅB, Skouby S, et al. Energy availability and the female athlete triad in elite endurance athletes. *Scand J Med Sci Sports.* 2015;25(5):610-622. doi:10.1111/sms.12261.
5. Levine JA. Non-exercise activity thermogenesis (NEAT): environment and biology. *Am J Physiol Endocrinol Metab.* 2004;286(5):E675-E685. doi:10.1152/ajpendo.00562.2003.
6. Speakman JR, Selman C. Physical activity and resting metabolic rate. *Proc Nutr Soc.* 2003;62(3):621-634. doi:10.1079/PNS2003282.
7. Goodpaster BH, Sparks LM. Metabolic flexibility in health and disease. *Cell Metab.* 2017;25(5):1027-1036. doi:10.1016/j.cmet.2017.04.015.
8. Randle PJ, Garland PB, Hales CN, Newsholme EA. The glucose fatty-acid cycle: its role in insulin sensitivity and the metabolic disturbances of diabetes mellitus. *Lancet.* 1963;1(7285):785-789. doi:10.1016/S0140-6736(63)91500-9.
9. Hue L, Taegtmeyer H. The Randle cycle revisited: a new head for an old hat. *Am J Physiol Endocrinol Metab.* 2009;297(3):E578-E591. doi:10.1152/ajpendo.00093.2009.

Primary studies added in version 1.1

- [R1] Koves TR, et al. Mitochondrial overload and incomplete fatty acid oxidation contribute to skeletal muscle insulin resistance. *Cell Metab.* 2008;7:45–56. <https://doi.org/10.1016/j.cmet.2007.10.013>
- [R2] Kim CW, et al. Unrestrained fatty acid oxidation triggers heart failure in mice via cardiolipin loss and mitochondrial dysfunction. *J Clin Invest.* 2026;136:e202528. <https://doi.org/10.1172/JCI202528>

Human studies added in version 1.1

- [H1] Calorie restriction increases muscle mitochondrial biogenesis in healthy humans. *PLoS Med.* 2007. PMID:17341128. <https://pubmed.ncbi.nlm.nih.gov/17341128/>

[H2] Calorie Restriction-induced Weight Loss and Exercise Have Differential Effects on Skeletal Muscle Mitochondria Despite Similar Effects on Insulin Sensitivity. 2017. PMID:28 158 621.
<https://pubmed.ncbi.nlm.nih.gov/28158621/>

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.

Original v1.0: <https://doi.org/10.5281/zenodo.22673566>. Version 1.1.1 (11 September 2026) changes licensing only; scientific content is unchanged from v1.1. CC BY-ND 4.0 applies to author-owned text; third-party material is excluded. No new DOI is assigned in this file.