

Energy Availability and Human Functional Capacity: Extending Sports Physiology Concepts Into General Population Health

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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 Not peer reviewed.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

Modern functional decline is often characterised by fatigue, reduced movement, poor recovery and worsening exercise tolerance. This paper asks whether chronic low-energy availability may contribute to these changes outside sport. EPK conceptualises spontaneous movement and recovery not merely as behaviours but as outputs of physiological capacity.^{1,2,3,4,5,6} 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.

Human Function as an Energetic Process

Neurological activity, thermoregulation, immunity, tissue repair, endocrine signalling and movement all require energy. Under prolonged restriction, the organism can reduce expenditure and spontaneous activity. Athletes make these effects visible through performance loss, but similar changes in non-athletes may be labelled aging, burnout or lack of motivation.

Spontaneous Movement

NEAT is a substantial component of daily expenditure and is sensitive to energetic state. Starvation and weight-loss studies show that spontaneous activity can fall during restriction. EPK therefore treats reduced movement as potentially both cause and consequence of underpowered physiology. Movement is not merely a tool for burning calories; it can be a marker of energetic sufficiency.^{1,2}

Recovery and Resilience

Tissue repair, glycogen restoration, sleep and endocrine regulation carry energetic costs. When recovery is chronically inadequate, prescribing ever more exercise may increase fatigue and reduce adherence. EPK therefore prioritises restoration of sufficient energy and recovery before progressively increasing movement demand.

Aging and Turning the Curve

The phrase 'turning the curve' describes a shift from progressive functional decline toward increasing capacity. It does not claim to reverse chronological aging. Rather, it proposes that some decline attributed to aging may be modifiable through improved energetic sufficiency, muscle retention, movement and recovery.

Cellular capacity and human performance

The cardiac studies by Cluntun et al. and Kim et al. show why fuel transport, oxidation and membrane integrity merit consideration alongside energy intake [R1,R2]. Their disease models cannot establish why an otherwise healthy person is tired, nor whether raising EPK improves exercise tolerance. Translation requires direct functional measurements.

The fruit-fly sleep study by Sarnataro et al. adds a further distinction: sleep pressure need not mean a simple shortage of ATP [R3]. For EPK research, perceived fatigue, sleep quality and measured exercise capacity should remain separate outcomes rather than being combined into an unvalidated metabolic score.

Human evidence from the PubMed search

A randomized crossover study of 12 endurance-trained women found poorer cycling performance after 14 days of low energy availability, with increased fat oxidation but unchanged measured muscle mitochondrial respiratory capacity. This supports a functional effect while challenging mitochondrial impairment as its necessary explanation. [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.

Testing functional capacity

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.

A prospective cohort could use a prespecified exercise-tolerance or walking-performance endpoint, with grip strength, accelerometry, sleep and perceived recovery as secondary measures. Establish repeatability and clinically meaningful change before testing the exposure association. Measure body composition, cardiorespiratory fitness, illness, medications, sleep and habitual activity; all can influence both intake and performance.

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.

Assess fuel switching under standardised conditions in a mechanistic subset and test whether baseline physiology modifies the EPK association. Improvement in symptoms without change in the proposed mechanism would support a narrower interpretation. Exercise performance, resting expenditure and mitochondrial respiration are related measurements, but none can substitute automatically for the others.

Conclusion

Energy availability may be relevant to general human function as well as sport. Prospective studies should examine whether EPK predicts spontaneous movement, recovery and functional capacity across age groups.^{1,2,3,4,5,6}

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Primary studies added in version 1.1

- [R1] Cluntun AA, et al. The pyruvate-lactate axis modulates cardiac hypertrophy and heart failure. *Cell Metab.* 2021;33:629–648.e10. <https://doi.org/10.1016/j.cmet.2020.12.003>
- [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>
- [R3] 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/s41586-025-09261-y>

Human studies added in version 1.1

- [H1] Caldwell HG, et al. The whole-body and skeletal muscle metabolic response to 14 days of highly controlled low energy availability in endurance-trained females. *FASEB J.* 2024;38:e70157. doi:10.1096/fj.202401780R. <https://pubmed.ncbi.nlm.nih.gov/39530548/>

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.22678102>. 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.