

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

Billy Craig | Independent Researcher

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Abstract

The possibility of inadequate energy provision outside sport is physiologically plausible, but nonspecific symptoms and low intake per total body mass do not establish a new diagnostic syndrome. This revision distinguishes low supply, impaired utilisation, adaptive responses and clinical disease. It integrates sleep and endocrine findings, nutritional guidance across life stages, and contrary evidence from calorie-restriction studies. EPK is presented as an exposure and programme to evaluate, not as a diagnostic substitute for REDs or clinical assessment.

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. Translating sports concepts carefully

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]

The updated IOC framework concerns problematic low energy availability in sport. It provides a reason to investigate endocrine, skeletal and performance outcomes, while exposure duration, severity and individual responses matter. Transferring a sports threshold to a non-athlete's intake divided by total mass would change both numerator and denominator. [1]

The phrase underpowered physiology is descriptive language in EPK, not an established disease category. Fatigue, reduced activity, poor sleep and cold intolerance have multiple possible causes. A dietary explanation must compete with illness, medication, sleep disorders and other explanations rather than being assumed.

2. Adiposity, supply and utilisation

Stored fat and present intake are distinct, but obesity alone cannot establish cellular energy deprivation. Greater adiposity lowers an intake-to-total-mass ratio at a fixed intake. Any proposed deficiency category must be evaluated independently of that arithmetic.

Experimental mitochondrial overload and genetic adipose dysfunction show that functional disturbance can arise in settings other than inadequate food supply. Adding calories is therefore not a general remedy for every metabolic phenotype. [2] [3]

Human restriction studies showing mitochondrial biogenesis or improved insulin sensitivity are necessary counterexamples to a universal metabolic-decline narrative. The target is to identify which exposures and participants experience harm, benefit or neither. [4] [5]

3. Restriction dose, endocrine responses and sleep

Minnesota provides a historical example of major consequences of severe prolonged restriction outside sport. Its selected population and protocol should not be presented as a model of every modern dietary intervention. [6]

Short experiments in exercising men report differing endocrine responses: leptin and insulin changed in one study without corresponding changes in several other hormones, while another manipulation in male runners found testosterone and fatigue effects in the most restricted condition. A single hormone cannot serve as an intake meter. [7] [8]

Sleep fragmentation after reduced availability in women and the contrasting CALERIE 2 findings illustrate the need to specify population, duration and dose. Symptoms alone cannot identify the mechanism. [9] [10]

4. Why 25 kcal/kg is not a universal floor

Hospital nutrition guidance uses weight-based energy estimates for particular clinical settings. This does not make 25 kcal/kg/day a universal RMR value, an EPK diagnostic boundary or a minimum adequate prescription for every adult. Guidance for cancer and geriatric care also requires individual adjustment. [11] [12] [13]

Pregnancy requirements concern maternal energy needs and tissue deposition; they are not obtained by dividing maternal intake by fetal mass. Infant and childhood needs change with growth and differ from adult maintenance. Those distinctions prevent a single lifespan cutoff. [14] [15] [16]

Critical illness requires its own assessment of expenditure and phase of feeding; an unvalidated app target must not override clinical nutrition care. The same caution applies to suspected malnutrition and refeeding risk. These groups require specialist study protocols rather than inclusion in an unsupervised general-population pilot. [17]

5. Distinguishing timing, balance and recovery

Fasting-window research should not be treated as interchangeable with chronic energy deficit. The hair-regeneration study raises a tissue-specific timing mechanism; it does not establish a general human diagnosis of inadequate calories. [18]

The developer describes an EPK programme using wearable-estimated total daily expenditure, a fallback estimate when data are absent, and an initial five-day learning period. It offers a target intended

to match the estimated baseline and an optional performance target approximately 15% above it, within a low-fat/high-carbohydrate eating pattern. These are programme specifications, not validated physiological thresholds. The intake ratio and the programme are distinct research objects. Device errors, missing data, the fallback equation and subsequent update rules must be specified before a reproducible trial. [19]

A baseline target that matches a wearable estimate is intended energy balance, not proof of it. Five days may not represent habitual demand during varying work, training or illness. Clinical validation requires recording these conditions and confirming that the intervention can be adjusted safely.

6. A discriminating general-population study

Recruit across activity and body-composition ranges, screen for conditions requiring clinical care, and record whether participants are weight-stable or losing weight. Measure dietary exposure, symptoms and function separately. Avoid recruiting only people already convinced that EPK helps them.

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.

The hypothesis would be weakened if lower EPK chiefly reflected greater adiposity, if it added no practical prediction, or if raising the target did not improve the prespecified functional outcome. Improvements attributable to additional support or treatment of an unrelated condition should not be assigned to energy restoration.

A first pilot should focus on feasibility and tolerability in an appropriately defined adult population. It should not aim simultaneously to validate a syndrome across pregnancy, children, frail older adults and disease. Those applications require separate designs and safeguards.

Conclusion

Energy adequacy deserves attention beyond sport, but the appropriate translation is a research question rather than a diagnosis. EPK must distinguish insufficient intake from impaired utilisation and account for life stage, body composition and clinical context.

Version and disclosure

Version 1.2 updates REDs context, removes universal threshold implications, adds lifespan and clinical boundaries, and specifies counterexamples and falsification tests. 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.

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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.

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