

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

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Abstract

This paper asks whether EPK can help support physical function, recovery and sustainable activity. It integrates controlled low-energy-availability studies, sleep, ageing and tissue-recovery evidence while separating performance from metabolic rate and mitochondrial biomarkers. A wearable-informed baseline and an optional 15% increment are programme choices requiring validation. The proposed study measures reproducible function and tolerability rather than assuming that a calorie surplus produces useful growth. Age-related loss of strength and aerobic capacity is not reduced to a single energy 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. Function as a primary outcome

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 practical concern is whether a person can undertake daily activity, train appropriately and recover without persistent fatigue. Body mass and calorie expenditure are relevant but incomplete outcomes. A rise in either can reflect different processes; neither demonstrates improved work capacity.

An EPK intervention should specify what better function means in its population: walking capacity in less-active adults, a standardised time trial in trained athletes, or strength and mobility in older people. These outcomes should not be combined into a proprietary score before its measurement properties are established.

2. Controlled evidence and carbohydrate availability

Caldwell et al. studied 12 endurance-trained women in controlled crossover conditions. Low availability impaired time-trial performance while measured muscle respiratory capacity remained unchanged. This supports direct performance measurement, not a requirement that mitochondrial failure explain the result. The related Jeppesen report should not be treated as an independent replication. [2] [3]

Short-term ketogenic-diet research found impaired anaerobic performance in trained adults. This is not a demonstration that resting metabolism necessarily declines. Energy and carbohydrate availability should be recorded separately, including timing relative to training. [4]

A four-day study in 16 recreationally active adults compared higher and lower carbohydrate availability during low energy availability. Higher carbohydrate attenuated the reduction in IGF-1; this was not a uniform rescue across measured hormones. Its limited endpoint does not establish complete recovery or validate a fixed carbohydrate prescription for all athletes. [5]

3. The performance pathway

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. [6]

Helms et al. compared maintenance, 5% and 15% surplus prescriptions during resistance training. Greater mass gain was more clearly associated with skinfold gain than superior strength or hypertrophy. The small study does not establish a universal optimal surplus, and specifically does not demonstrate that 15% is necessary for growth. [7]

For testing, distinguish a prescribed 15% above an estimated baseline from a measured physiological surplus. Monitor training stimulus, protein, adherence, fat and lean tissue, and relevant performance. Avoid attributing glycogen-associated water changes or all fat-free-mass gain to new muscle.

4. Recovery, sleep and tissue context

Restricted sleep altered the composition of weight loss in a controlled experiment. This supports treating sleep as a potential modifier rather than assuming calories alone determine tissue preservation. [8]

Long-distance trail-running observations show red-cell oxidative changes following extreme exertion. The evidence was assessed at abstract level and cannot identify inadequate fuelling as the cause or show that ordinary exercise damages health. It motivates recording exercise dose and recovery. [9]

Fasting research implicates tissue-specific interorgan signalling in hair-follicle regeneration, predominantly through animal mechanisms with a small short human component. Meal timing and total energy reduction should be distinguished. Hair growth is not a validated general recovery marker for EPK. [10]

5. Ageing and movement

Longitudinal work documents loss of strength that is not explained fully by loss of muscle mass, and an accelerating decline in aerobic capacity with age. These findings argue for measuring both tissue quantity and function. [11] [12]

Structured physical activity reduced major mobility disability in the LIFE trial. Training research also demonstrates measurable adaptations in older adults. Such evidence supports modifiability of function, but does not show that ageing is mainly caused by insufficient calories or that a high RMR per kilogram is a treatment target. [13] [14]

Centenarian comparisons are selected observational samples. They cannot establish that increasing RMR or enforcing 25 kcal/kg/day intake produces longevity. Body composition and survivor selection complicate the comparison. [15]

6. A functional study design

Select one primary task with established repeatability, familiarise participants before baseline, and standardise testing time, food and exercise. Record training volume, illness, medication, sleep, body composition and baseline fitness. Use assessors unaware of allocation where feasible.

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.

In a pragmatic randomised trial, match coaching attention. Assess fatigue, enjoyment, adherence and adverse events alongside performance. A feasibility study should determine whether participants can follow the programme and complete testing, not use a small favourable effect to declare efficacy. [16]

For a later powered trial, prespecify a meaningful functional difference and use an appropriate variance estimate. Repeated days do not multiply the number of independent participants. A credible result could be improvement in function without higher expenditure; an increase in calories burned alone would be insufficient.

Conclusion

Adequate fuelling and effective training are plausible partners, but EPK must show that its guidance improves meaningful function. Performance, recovery, muscle preservation and tolerability require separate measurement, and the 15% pathway remains a testable option.

Version and disclosure

Version 1.2 adds controlled performance evidence, ageing and sleep outcomes, qualifies the performance increment, and distinguishes tissue biomarkers from real-world function. 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.
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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.

v1.1.1 - 11 September 2026: earlier licensing release. Archived DOI: 10.5281/zenodo.22710029. Earlier versions remain available through Zenodo.

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