Energy Per Kilogram (EPK): A Physiology-Aware Framework for Human Energy Availability and Dietary Adherence

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Working Paper | Version 1.0 | 9 September 2026

Status: conceptual/hypothesis-generating working paper. Not peer reviewed.

Abstract

Traditional calorie-restriction approaches to weight management frequently demonstrate poor long-term adherence and limited maintenance of weight loss. Emerging evidence suggests that physiological and behavioural compensatory responses may contribute substantially to these outcomes. The present paper explores the relationship between energy restriction, compensatory behaviour, dietary adherence, and energy availability through both original MSc research findings and broader physiological literature.[1,2,3,4,5,6,7]

The original research investigated the relationships between calorie deficit targets, compensatory health beliefs (CHBs), autonomous motivation, dietary adherence, and dieting success in individuals following self-directed weight-loss diets. Although the primary psychological hypotheses were statistically non-significant, important behavioural patterns emerged. Participants consistently failed to achieve intended calorie deficits, with actual deficits significantly lower than targets. Greater intended calorie deficits were associated with greater fluctuations in calorie intake, suggesting oscillatory compensatory behaviour in response to restriction. Mean intake across the intervention period frequently drifted toward estimated maintenance requirements despite ongoing dieting intentions.[8,6,7,9]

The paper argues that these findings may be better explained through physiological compensation rather than simple failures of willpower or motivation. Adaptive thermogenesis, appetite regulation, hypothalamic energy sensing, and constrained energy expenditure models all suggest that the human organism actively resists prolonged energy restriction in an attempt to maintain equilibrium. These responses may manifest behaviourally as overeating episodes, calorie fluctuations, reduced adherence, and long-term weight regain.[1,2,3,10,11,4,5]

Building upon these observations, this paper introduces the EPK (Energy Per Kilogram) framework as a practical model for assessing sustainable human energy availability. Rather than focusing exclusively on static calorie deficits, EPK attempts to contextualise energy intake relative to body mass, physiological function, recovery capacity, and compensatory risk. The framework proposes that chronic low-energy states may contribute to behavioural and physiological

DOI: 10.5281/zenodo.22673106

instability, whereas more sustainable energy availability may improve adherence, recovery, appetite regulation, and long-term outcomes.[4,12,13,14,15,16,17]

The paper proposes that obesity and dieting research may benefit from shifting away from purely deficit-based models toward more physiology-aware approaches that account for metabolic individuality, adaptive responses, and constrained energy systems. Further research is required to validate EPK thresholds and determine its clinical utility across different populations and activity levels.[18,19]

Introduction

Obesity and weight-management difficulties remain among the most significant public health concerns globally. Although calorie restriction continues to represent the dominant approach to weight loss interventions, long-term success rates remain consistently poor. Numerous studies have demonstrated that while short-term weight reduction is often achievable, the maintenance of that weight loss is substantially more difficult, with many individuals regaining most or all of the weight lost within several years.[1,2,3,4,5]

Traditional dietary models frequently operate on the assumption that weight regulation is predominantly governed by conscious behavioural control over calorie intake. Within this framework, greater calorie deficits are typically expected to produce greater rates of weight loss. However, both clinical observations and experimental evidence increasingly suggest that the human organism does not respond passively to energy restriction. Instead, caloric deficits appear to trigger a variety of compensatory physiological and behavioural responses designed to restore equilibrium and protect energy availability.[20,21,22,23]

These compensatory responses may include:

increased hunger and food preoccupation,

reductions in metabolic rate,[10,11,24,25,26]

altered spontaneous activity levels,

reductions in exercise efficiency,

endocrine adaptations,

psychological distress,

and oscillatory eating behaviours characterised by periods of restriction followed by episodes of overconsumption.[8,9]

The author's original MSc research explored whether compensatory health beliefs (CHBs), autonomous motivation, and calorie deficit targets predicted dietary adherence and dieting success in individuals following self-directed weight-loss diets. Although the primary psychological hypotheses failed to achieve statistical significance, important behavioural patterns emerged throughout the data. Participants consistently failed to achieve intended calorie deficits, with actual deficits significantly lower than planned targets. Furthermore, larger intended calorie deficits were associated with greater fluctuations in calorie intake.[6,7]

These findings suggested that participants were not simply "failing" to follow dietary instructions through lack of willpower. Rather, the observed behavioural fluctuations appeared consistent

DOI: 10.5281/zenodo.22673106

with compensatory responses to energy restriction. Indeed, participants frequently alternated between periods of under-eating and over-eating in a manner suggestive of biological attempts to restore energy balance.

Modern physiological research provides several mechanisms that may explain these observations. Adaptive thermogenesis, appetite regulation through leptin and ghrelin signalling, hypothalamic regulation of energy availability, and constrained energy expenditure models all suggest that the body actively resists prolonged energy restriction. These responses challenge simplistic linear assumptions regarding calorie deficits and expected weight loss outcomes.[10,11,4,24,12,25,26]

In parallel with these physiological findings, growing criticism has emerged surrounding the accuracy of standardised calorie prediction equations and universal calorie prescriptions. Inter-individual variability in metabolic rate, non-exercise activity thermogenesis, spontaneous movement, endocrine status, body composition, and adaptive responses may substantially alter actual energy requirements between individuals of similar body size. Consequently, two individuals consuming identical calorie intakes may experience dramatically different physiological and behavioural outcomes.[10,11,24,25,26,27,28]

The present paper proposes that many dieting difficulties traditionally framed as behavioural failures may instead represent predictable compensatory responses to insufficient energy availability. Building upon the author's MSc findings, alongside modern constrained-energy and adaptive physiology literature, this paper introduces the EPK (Energy Per Kilogram) framework as a practical approach for assessing sustainable human energy availability.[20,21,22,23]

Rather than focusing exclusively upon static calorie deficits, EPK attempts to contextualise energy intake relative to body mass, physiological function, recovery capacity, behavioural stability, and compensatory risk. The framework proposes that chronic low-energy states may contribute to deteriorations in appetite regulation, sleep quality, mood, recovery, adherence, and overall physiological function, whilst more sustainable energy availability may support improved long-term behavioural stability and health outcomes.[4,12,13,14,15,16,17]

The purpose of this paper is therefore threefold:

To review evidence surrounding compensatory responses to energy restriction.

To reinterpret observed dieting behaviours through a physiological rather than purely behavioural lens.

To introduce the EPK framework as a physiology-aware model for understanding sustainable energy availability and dietary adherence.[6,7,20,21,22,23]

2. Background Literature

Calorie Restriction and Long-Term Weight Management[1,2,3,4,5]

Calorie restriction remains the dominant strategy within conventional weight-loss interventions. The central premise is that body mass is primarily governed by energy balance, whereby sustained calorie deficits produce reductions in body weight. Although this model is

thermodynamically correct at a basic level, practical human outcomes often diverge substantially from theoretical predictions.

Short-term weight loss during periods of caloric restriction is common; however, long-term maintenance remains poor. Meta-analyses demonstrate that many individuals regain significant proportions of lost weight within several years following dieting interventions. Furthermore, actual weight loss achieved during interventions is frequently lower than mathematically predicted based upon estimated calorie deficits.

The author's MSc research demonstrated similar findings. Participants engaged in self-directed calorie restriction strategies with the intention of achieving meaningful weight loss. However, actual calorie deficits achieved were significantly smaller than intended targets, and greater intended deficits predicted greater fluctuations in intake behaviour. These findings align with broader literature suggesting that human responses to restriction are dynamic rather than linear.

Traditional "3500 kcal per pound" models of weight loss have increasingly been criticised for oversimplifying the biological complexity of human energy regulation. Hall et al. demonstrated that weight loss trajectories are influenced by dynamic physiological adaptations, including reductions in energy expenditure and alterations in body composition over time. Consequently, predicted linear weight loss rarely occurs in real-world conditions.[10,11,24,25,26]

Adaptive Thermogenesis[10,11,24,25,26]

Adaptive thermogenesis refers to reductions in energy expenditure beyond what would be expected from changes in body mass alone. During caloric restriction, the body appears to actively reduce energy expenditure in an attempt to conserve energy and restore equilibrium.[10,11,24,25,26]

Classic evidence emerged from the Minnesota Starvation Experiment conducted by Keys et al., in which healthy male participants underwent prolonged semi-starvation. Participants experienced profound reductions in basal metabolic rate, decreased spontaneous movement, reductions in body temperature, psychological distress, food preoccupation, and binge-eating tendencies during refeeding. These findings demonstrated that energy restriction produces widespread physiological and behavioural adaptations rather than isolated reductions in body mass.[10,11,24,25,26,27,28]

Modern studies continue to support these observations. Leibel et al. reported that weight-reduced individuals exhibit persistent reductions in energy expenditure even after accounting for changes in body composition. Rosenbaum and Leibel further suggested that adaptive thermogenesis may represent an evolutionary survival mechanism designed to resist prolonged energy deficits.[10,11,24,25,26]

The implications for dieting adherence are substantial. If caloric restriction progressively reduces energy expenditure whilst simultaneously increasing hunger and food preoccupation, sustained adherence may become increasingly difficult despite strong motivation.[10,11,24,25,26]

Appetite Regulation and Energy Sensing[4,12,13,14,15,16,17]

Human appetite regulation involves complex interactions between peripheral hormones, central nervous system signalling, and hypothalamic energy sensing mechanisms.[4,12,13,14,15,16,17]

DOI: 10.5281/zenodo.22673106

Leptin, primarily secreted from adipose tissue, acts as a long-term signal of energy sufficiency. During caloric restriction and fat loss, leptin concentrations decrease substantially, signalling reduced energy availability to the hypothalamus. This reduction is associated with increased hunger, reduced satiety, decreased thyroid output, and reductions in energy expenditure.[10,11,4,24,12,25,26]

Conversely, ghrelin concentrations typically increase during caloric restriction. Ghrelin stimulates appetite and food-seeking behaviour, particularly during states of negative energy balance. Sumithran et al. demonstrated that hormonal adaptations promoting hunger may persist long after weight loss has occurred, potentially contributing to long-term regain.[4,12,13,14,15,16,17]

The author's MSc findings may reflect behavioural manifestations of these physiological processes. Participants frequently oscillated between periods of restriction and overconsumption, particularly when larger deficits were pursued. Such fluctuations appear consistent with compensatory responses driven by biological attempts to restore energy balance.[8,9]

Set-point and settling-point models further support the concept that body weight and energy intake are biologically regulated rather than exclusively behaviourally controlled. Although these theories remain debated, they provide useful frameworks for understanding why prolonged restriction frequently produces compensatory responses.

Constrained Energy Expenditure[10,11,24,18,19,25,26]

Traditional energy balance models often assume that increases in physical activity proportionally increase total daily energy expenditure. However, constrained energy expenditure theory challenges this assumption.[10,11,24,18,19,25,26]

Pontzer et al. proposed that total energy expenditure is biologically constrained within relatively narrow ranges. According to this model, increases in physical activity may be partially compensated for through reductions in energy expenditure elsewhere within the organism. This compensation may occur through:[10,11,24,18,19,25,26]

reductions in spontaneous movement,[27,28]

hormonal adaptation,

immune modulation,

reproductive suppression,

or reductions in other physiological processes.

This theory may help explain why aggressive dieting combined with excessive exercise frequently produces diminishing returns over time. Rather than creating indefinitely increasing energy deficits, the body may compensate behaviourally and physiologically to preserve overall energy balance.

Importantly, constrained energy models align with observations from the author's MSc research, where participants appeared unable to sustain large intended deficits despite continued dieting intentions.[18,19]

Metabolic Individuality

One major limitation of standard dieting approaches is the assumption that energy requirements can be accurately estimated through generic equations and activity multipliers. Although predictive equations such as Mifflin-St Jeor are widely used, substantial inter-individual variability exists in actual energy expenditure.[10,11,24,29,30,25,26]

Differences in:

resting metabolic rate,[10,11,24,25,26]

spontaneous movement,[27,28]

body composition,

endocrine function,

thermic effect of food,

recovery demands,

stress physiology,

and adaptive responses

may substantially alter true energy requirements between individuals of similar size.

The author's MSc research discussed the limitations of standardised calorie calculations extensively, noting that inaccurate estimations may contribute directly to poor adherence outcomes.

Consequently, two individuals prescribed identical calorie intakes may experience dramatically different physiological and behavioural responses. This variability highlights the potential importance of individualised approaches to energy assessment and management.

Dietary Adherence and Compensatory Behaviour[6,7]

Dietary adherence consistently emerges as one of the strongest predictors of successful weight management outcomes. However, adherence itself may be heavily influenced by physiological state.[6,7]

Rather than viewing overeating episodes purely as behavioural failures, emerging evidence suggests that compensatory behaviours may represent adaptive biological responses to perceived energy insufficiency.

The Minnesota Starvation Experiment demonstrated that prolonged restriction increased food obsession, binge eating, emotional instability, and compulsive food-related behaviours even in highly motivated participants. Similarly, modern dieting studies frequently report increased cravings, reduced mood, fatigue, irritability, and episodes of overconsumption following restriction.[8,9]

The author's MSc findings appear consistent with these observations. Participants pursuing larger deficits demonstrated greater intake fluctuations and poorer adherence, suggesting that aggressive restriction may increase compensatory drive.

DOI: 10.5281/zenodo.22673106

Collectively, the literature suggests that many dieting difficulties traditionally interpreted as failures of discipline may instead reflect predictable physiological and behavioural responses to chronic energy restriction.

3. MSc Study Findings: Compensatory Behaviour During Energy Restriction

Study Overview

The author's original MSc research explored the relationships between calorie deficit targets, compensatory health beliefs (CHBs), autonomous motivation, dietary adherence, and dieting success in individuals engaging in self-directed dieting.[6,7]

Three primary hypotheses were investigated:

Whether calorie deficit targets and motivational orientation predicted CHBs.

Whether calorie deficit targets, CHBs, and motivational orientation predicted dietary adherence.[6,7]

Whether calorie deficit targets, CHBs, motivational orientation, and adherence predicted dieting success.

Participants were either:

actively attempting to lose weight,

or

interested in beginning a self-directed dieting intervention.

After exclusions for incomplete data and inadequate dietary logging, the final analysis consisted of 21 participants aged 18-67 years.

Dietary intake was monitored using MyFitnessPal, and participants attempted to follow self-selected calorie deficit targets over a one-month period.[31,32]

Primary Findings

The original regression analyses failed to demonstrate statistically significant relationships between the primary psychological variables and the measured outcomes. However, important behavioural findings emerged throughout the data which appeared highly relevant to dietary adherence and compensatory behaviour.[6,7]

The most striking finding was the discrepancy between intended calorie deficits and actual calorie deficits achieved.

Participants consistently failed to achieve the calorie deficits they intended to follow. Actual calorie deficits were significantly smaller than target deficits, indicating substantial adherence difficulties.

The mean intended calorie deficit was approximately 21.4%, whereas the actual mean deficit achieved was only 12.6%. Furthermore, the mean weight loss achieved during the study was modest relative to predicted outcomes based upon intended energy restriction.

DOI: 10.5281/zenodo.22673106

Importantly, participants did not simply fail uniformly. Instead, calorie intake fluctuated considerably above and below targets throughout the intervention period.

Participants consumed above their calorie target on approximately 62.3% of days and below target on 14.7% of days. Mean calorie fluctuation around targets reached approximately 1,697 kcal.

Perhaps most importantly, larger intended calorie deficits predicted larger fluctuations in calorie intake behaviour.

This observation suggested that increasingly aggressive restriction may destabilise eating behaviour rather than improve adherence.

Evidence of Compensatory Behaviour

Although the original CHB-related hypotheses were non-significant, behavioural patterns observed throughout the study suggested the presence of compensatory responses to energy restriction.

Participants frequently alternated between periods of excessive restriction and periods of increased intake. The data suggested that periods of under-eating were often followed by episodes of compensatory overconsumption, whilst periods of overconsumption were then followed by renewed restrictive attempts.

These oscillatory patterns appeared particularly evident amongst individuals pursuing larger calorie deficits.[8,9]

The author proposed that these fluctuations may reflect behavioural manifestations of physiological compensation rather than isolated failures of motivation or discipline.

This interpretation aligns with modern physiological research demonstrating that energy restriction produces:

increased hunger,

elevated food preoccupation,

reductions in energy expenditure,[10,11,24,25,26]

altered endocrine signalling,

and increased drive toward energy restoration.

The findings therefore challenged the assumption that adherence failures are predominantly behavioural in nature.

Physiological Interpretation of Adherence Failure

The study discussed several physiological mechanisms that may help explain the observed adherence difficulties.

Firstly, inaccuracies in estimating energy requirements may have contributed to inappropriate calorie targets. Standard predictive equations cannot fully account for inter-individual variability in metabolic rate, activity expenditure, recovery demands, or adaptive responses. Consequently,

DOI: 10.5281/zenodo.22673106

prescribed deficits may have been substantially larger than intended for certain individuals.[10,11,24,29,30,25,26]

Secondly, adaptive physiological responses to restriction may have progressively increased compensatory drive throughout the intervention.

The author discussed evidence relating to:

reductions in metabolic rate,[10,11,24,25,26]

adaptive thermogenesis,[10,11,24,25,26]

increased appetite signalling,[4,12,13,14,15,16,17]

leptin suppression,[4,12,13,14,15,16,17]

ghrelin elevation,[4,12,13,14,15,16,17]

and hypothalamic regulation of energy balance.[4,12,13,14,15,16,17]

These mechanisms collectively suggest that the body actively resists prolonged energy restriction in an attempt to maintain survival and physiological function.

The study therefore proposed that many observed “adherence failures” may represent predictable biological responses to insufficient energy availability rather than simple deficits in willpower.[20,21,22,23]

This interpretation is supported by classic starvation research. Keys et al. demonstrated that prolonged energy restriction produced:

food obsession,

binge eating,[8,9]

emotional instability,

irritability,

reduced movement,

and compulsive food-related behaviours even in highly motivated individuals.

Similarly, modern dieting studies consistently report increased cravings, mood disturbances, and compensatory overeating following prolonged restriction.

The findings of the present study appear consistent with these broader observations.

From Behavioural Observation to Physiological Framework

Although the original MSc research focused primarily upon compensatory health beliefs, the behavioural patterns observed during the intervention increasingly suggested a broader physiological explanation for dieting difficulties.

Participants did not behave as passive mathematical systems responding linearly to calorie prescriptions. Instead, intake behaviour appeared dynamic, compensatory, and heavily influenced by physiological state.

DOI: 10.5281/zenodo.22673106

These observations contributed conceptually to the later development of the EPK (Energy Per Kilogram) framework.

Rather than focusing exclusively upon calorie deficits, the EPK model attempts to assess:

sustainable energy availability,[20,21,22,23]

physiological stability,

behavioural consistency,

and compensatory risk.

The MSc findings therefore represent an early observational foundation for the argument that many dieting difficulties may emerge from insufficient energy availability and physiological compensation rather than purely behavioural failure.[20,21,22,23]

4. From Compensatory Behaviour to Energy Availability

Reframing Dieting Failure

Traditional dieting models frequently interpret poor adherence as a behavioural or motivational problem. Within this framework, individuals are often viewed as failing because they lack discipline, self-control, or sufficient commitment to dietary goals. However, the findings from the author's MSc research, alongside broader physiological literature, suggest that this interpretation may be incomplete.

The observed intake fluctuations during the study did not appear random. Instead, participants frequently oscillated between periods of restriction and compensatory overconsumption, particularly when larger calorie deficits were pursued. These patterns appeared consistent with physiological attempts to restore equilibrium rather than isolated behavioural lapses.[8,9]

Modern energy regulation research increasingly supports the concept that the human organism actively defends energy availability. When energy intake falls below physiological requirements, compensatory mechanisms emerge to restore sufficient energy for survival, reproduction, repair, immune function, thermoregulation, and neurological activity.[20,21,22,23]

This perspective fundamentally alters the interpretation of dieting behaviour.

Rather than asking:

"Why do people fail diets?"

the more appropriate physiological question may be:

"What compensatory responses emerge when energy availability becomes insufficient?"[20,21,22,23]

Energy Availability as a Central Physiological Variable

Energy availability has traditionally been discussed primarily within sports science and relative energy deficiency literature. However, the concept may have broader implications across the general population.[20,21,22,23]

Low energy availability occurs when energy intake becomes insufficient to support:[20,21,22,23]

DOI: 10.5281/zenodo.22673106

basal metabolic requirements,

movement demands,

tissue repair,

endocrine function,

neurological activity,

and other physiological processes.

Importantly, low energy availability does not necessarily require starvation-level calorie intake. Rather, it may emerge whenever intake becomes insufficient relative to total physiological demands.[20,21,22,23]

The author's MSc findings suggested that individuals attempting larger calorie deficits experienced greater behavioural instability and adherence difficulties. This may indicate that aggressive restriction increases the likelihood of entering physiologically unstable states characterised by elevated compensatory drive.

Research on adaptive thermogenesis further supports this concept. During restriction, the body progressively reduces:[10,11,24,25,26]

resting metabolic rate,[10,11,24,25,26]

spontaneous movement,[27,28]

thermogenesis,

reproductive function,

thyroid output,

and other energetically expensive processes.

Simultaneously, appetite signalling increases through alterations in:[4,12,13,14,15,16,17]

leptin,[4,12,13,14,15,16,17]

ghrelin,[4,12,13,14,15,16,17]

neuropeptide-Y,

and hypothalamic energy sensing.[4,12,13,14,15,16,17]

Collectively, these responses appear designed to restore energy sufficiency rather than facilitate continued restriction.

The Development of the EPK Concept

The EPK (Energy Per Kilogram) framework emerged conceptually from the observation that absolute calorie values alone may inadequately describe physiological energy status.

Traditional dieting approaches frequently prescribe calorie targets using:

standardised equations,[29,30,33,25]

fixed calorie deficits,

or universal calorie recommendations.

However, these methods may fail to adequately account for:

body size,

lean mass,

activity variability,

recovery demands,

adaptive responses,

metabolic individuality,

and constrained energy expenditure.[10,11,24,18,19,25,26]

The author's MSc research discussed these limitations extensively, noting that estimated energy requirements may differ substantially from true physiological requirements.[29,30,33,25]

EPK therefore attempts to contextualise energy intake relative to body mass and physiological function.

Rather than focusing exclusively on:

"How large is the calorie deficit?"

the EPK model instead asks:

"Is sufficient energy available to support stable physiological function?"

This distinction is important because two individuals consuming identical calorie intakes may experience dramatically different physiological states depending upon:

body mass,

activity levels,

recovery demands,

metabolic rate,[10,11,24,25,26]

endocrine function,

and adaptive responses.

Physiological Stability Versus Restriction Severity

The central proposition of the EPK framework is that physiological stability may be more important than aggressive restriction when considering long-term adherence and health outcomes.

Low-energy states may contribute to:

increased food preoccupation,

fatigue,

sleep disruption,

DOI: 10.5281/zenodo.22673106

reduced recovery,

elevated stress physiology,

reduced spontaneous activity,

mood instability,

and compensatory overeating behaviours.

These outcomes are widely documented within starvation and restriction literature.

The Minnesota Starvation Experiment demonstrated that prolonged restriction produced profound behavioural and psychological changes despite high participant motivation. Participants became increasingly obsessed with food, emotionally unstable, socially withdrawn, and prone to binge eating during refeeding.[8,9]

Importantly, many modern dieters may unknowingly recreate milder versions of these physiological states through repeated cycles of aggressive restriction.

The author's MSc findings suggested that larger calorie deficits produced greater behavioural instability and reduced adherence. This aligns with the hypothesis that physiological compensation intensifies as energy availability decreases.[20,21,22,23]

The EPK framework therefore proposes that maintaining more sustainable energy availability may:[20,21,22,23]

reduce compensatory drive,

improve behavioural consistency,

support recovery,

improve appetite regulation,[4,12,13,14,15,16,17]

and enhance long-term adherence.

Compensatory Behaviour as a Physiological Signal

Within the EPK framework, compensatory behaviours are not viewed purely as failures of discipline.

Instead, behaviours such as:

binge eating,[8,9]

intense cravings,

oscillatory restriction,[8,9]

food obsession,

fatigue,

reduced movement,

and increased appetite

may represent physiological signals indicating insufficient energy availability.[20,21,22,23]

DOI: 10.5281/zenodo.22673106

This interpretation is consistent with modern constrained-energy and appetite-regulation research.

Importantly, this does not imply that energy balance principles are invalid. Rather, it suggests that human energy regulation is dynamic, adaptive, and biologically defended.

The body does not appear to passively tolerate prolonged deficits without compensation.

Consequently, dietary strategies that ignore compensatory physiology may inadvertently increase:

behavioural instability,

metabolic adaptation,

and long-term regain risk.

Toward a Physiology-Aware Framework

The EPK framework therefore represents an attempt to integrate:

adaptive thermogenesis,[10,11,24,25,26]

appetite regulation,[4,12,13,14,15,16,17]

constrained energy expenditure,[10,11,24,18,19,25,26]

metabolic individuality,

and behavioural compensation

into a more physiology-aware model of dietary adherence and energy management.[6,7]

Rather than viewing weight management solely through the lens of restriction severity, EPK proposes that sustainable human function may depend upon maintaining sufficient energy availability relative to physiological demand.[20,21,22,23]

Further research is required to determine:

optimal EPK thresholds,

relationships between EPK and compensatory behaviour,

associations with metabolic health markers,

and long-term adherence outcomes across different populations.

However, the observational findings from the author's MSc research suggest that compensatory behaviour during dieting may represent an important physiological phenomenon worthy of further investigation.

DOI: 10.5281/zenodo.22673106

5. Energy Availability Per Unit Mass: Lessons From Sports Physiology and Human Performance

Energy Availability in Performance Research

Within sports physiology, the concept of energy availability has become increasingly important in understanding human performance, recovery, endocrine health, and long-term physiological function. Research in athletes consistently demonstrates that insufficient energy availability negatively affects multiple biological systems, even when body weight appears relatively stable.[20,21,22,23]

Low energy availability (LEA) is typically defined as a state in which dietary energy intake becomes insufficient to support both exercise expenditure and the energetic requirements necessary for normal physiological functioning. Importantly, the consequences of LEA extend far beyond athletic performance alone.[20,21,22,23]

Research demonstrates that chronic LEA may contribute to:

endocrine disruption,

reductions in metabolic rate,[10,11,24,25,26]

impaired recovery,

reductions in bone health,

decreased immune function,

impaired reproductive function,

increased fatigue,

mood disturbances,

and impaired cognitive performance.

The development of Relative Energy Deficiency in Sport (RED-S) further expanded understanding beyond the original Female Athlete Triad model, recognising that low energy availability affects multiple systems in both males and females.[20,21,22,23]

Although these concepts emerged within elite sport, the physiological principles involved are not exclusive to athletes. All humans require sufficient energy availability to support:[20,21,22,23]

neurological function,

immune activity,

movement,

tissue repair,

endocrine signalling,

thermoregulation,

and psychological stability.

DOI: 10.5281/zenodo.22673106

The difference between athletes and the general population may therefore be one of visibility rather than physiology.

Athletes often notice reductions in performance rapidly when energy availability becomes insufficient. In contrast, non-athletic populations may experience:[20,21,22,23]

fatigue,

poor sleep,

reduced motivation,

increased hunger,

reduced recovery,

reduced spontaneous movement,[27,28]

and gradual metabolic decline

without recognising low energy availability as a contributing factor.[20,21,22,23]

Energy Per Unit Mass as a Physiological Concept

Sports physiology research frequently assesses energy availability relative to body size or fat-free mass rather than relying solely on absolute calorie intake.[20,21,22,23]

This distinction is critical because:

a fixed calorie intake does not represent the same physiological state across different individuals,

larger individuals generally require greater total energy availability,[20,21,22,23]

and activity expenditure substantially alters energetic requirements.

For example, a daily intake of 1800 kcal may represent:

severe restriction for one individual,

moderate restriction for another,

or approximate maintenance for a smaller sedentary individual.

The author's EPK framework extends this principle beyond athletic populations by proposing that energy intake relative to body mass may provide a more physiologically meaningful indicator than absolute calorie values alone.

This approach aligns conceptually with existing sports performance literature where:

energy availability,[20,21,22,23]

body mass,

lean tissue preservation,

and recovery status

are all considered critical determinants of performance and health outcomes.

DOI: 10.5281/zenodo.22673106

The present framework proposes that similar principles may apply across broader human physiology.

Human Function Requires Energy Sufficiency

Many modern dieting approaches normalise chronic low-energy states as desirable or necessary for body composition change. However, from a physiological perspective, the human organism appears designed to function optimally under conditions of sufficient energy availability.[20,21,22,23]

Research consistently demonstrates that low-energy states impair:

thyroid function,

reproductive function,

mood,

recovery,

sleep quality,

exercise tolerance,

and spontaneous activity.

The Minnesota Starvation Experiment provided perhaps the clearest demonstration of the systemic consequences of prolonged energy restriction. Participants experienced:

reductions in metabolic rate,[10,11,24,25,26]

reduced movement,

emotional instability,

food obsession,

depressive symptoms,

and profound behavioural changes.

Importantly, many of these symptoms mirror complaints commonly observed in chronic dieters and modern low-calorie interventions, albeit often to lesser degrees.

The present paper argues that these outcomes may not represent isolated psychological weakness, but rather expected biological responses to insufficient energy availability.[20,21,22,23]

From this perspective, humans may function best when physiologically “fully powered” rather than chronically energy depleted.

This does not imply unlimited energy intake or rejection of body composition goals. Rather, it suggests that:

stable physiology,

behavioural consistency,

and sustainable energy availability[20,21,22,23]

may represent more appropriate long-term targets than aggressive restriction alone.

DOI: 10.5281/zenodo.22673106

Compensatory Physiology and Functional Decline

One major implication of chronic low-energy availability may be gradual functional decline.[20,21,22,23]

As energy availability decreases, the organism appears to progressively down-regulate energetically expensive processes in order to preserve survival. This may include:[20,21,22,23]

reductions in spontaneous movement,[27,28]

reductions in reproductive signalling,

suppression of thyroid activity,

reductions in immune activity,

decreased recovery capacity,

and reductions in exercise output.

Within athletes, these effects are often recognised rapidly because performance deteriorates measurably. In non-athletic populations, however, these same adaptations may emerge more gradually and become normalised as:

“aging,”

“burnout,”

“lack of motivation,”

or “poor discipline.”

The EPK framework proposes that many individuals may unknowingly exist in chronically underpowered physiological states whilst attempting to sustain restrictive dietary behaviours.

The author’s MSc findings suggested that larger calorie deficits produced greater behavioural instability and poorer adherence. This observation aligns with the hypothesis that insufficient energy availability progressively increases compensatory drive and destabilises behaviour.[20,21,22,23]

From Weight-Centric Models to Function-Centric Models

Traditional dieting models frequently prioritise body weight reduction as the primary marker of success. However, sports physiology literature often places greater emphasis on:[20,21,22,23]

functional output,

recovery,

tissue preservation,

endocrine health,

and long-term performance sustainability.

The EPK framework attempts to integrate these principles into a broader physiology-aware model applicable beyond athletic populations.

Rather than asking exclusively:

“How much weight was lost?”

DOI: 10.5281/zenodo.22673106

the framework instead asks:

Is physiological function improving?

Is behavioural stability improving?

Is recovery improving?

Is spontaneous movement increasing?[27,28]

Is sleep quality improving?

Is food preoccupation decreasing?

Is energy availability sufficient to support sustainable function?[20,21,22,23]

This represents a conceptual shift away from purely weight-centric approaches toward function-centric physiology.

EPK as a Practical Energy Availability Framework

The EPK model therefore proposes that energy intake relative to body mass may serve as a practical marker for:

physiological sufficiency,

compensatory risk,

recovery status,

and behavioural stability.

The framework does not reject energy balance principles, but instead proposes that:

human energy regulation is adaptive,

physiology actively defends energy availability,[20,21,22,23]

and chronic low-energy states may destabilise both biology and behaviour.

By incorporating concepts from:

sports energy availability research,[20,21,22,23]

adaptive thermogenesis,[10,11,24,25,26]

constrained energy expenditure,[10,11,24,18,19,25,26]

and compensatory behavioural physiology,

EPK attempts to provide a more integrated framework for understanding long-term dietary adherence and human function.[6,7]

Further research is required to validate:

specific EPK thresholds,

relationships with metabolic markers,

associations with recovery and behavioural stability,

and long-term clinical outcomes.

DOI: 10.5281/zenodo.22673106

However, parallels between sports physiology and the present findings suggest that energy sufficiency may represent a central determinant of sustainable human performance, health, and behavioural regulation.[20,21,22,23]

6. The EPK Framework

Introduction to the Framework

The EPK (Energy Per Kilogram) framework was developed as a practical attempt to assess human energy availability in a more physiologically meaningful manner than static calorie prescriptions alone.[20,21,22,23]

Traditional dietary approaches frequently utilise:

fixed calorie targets,

standardised calorie deficits,

or generic predictive equations.[29,30,33,25]

However, these approaches may fail to adequately account for:

metabolic individuality,

adaptive physiology,

body size differences,

activity variability,

recovery demands,

constrained energy expenditure,[10,11,24,18,19,25,26]

and compensatory behavioural responses.

The author's MSc findings demonstrated that participants frequently failed to sustain intended deficits, with larger calorie deficits associated with greater fluctuations in intake behaviour. These observations suggested that aggressive restriction may destabilise physiological and behavioural regulation.

The EPK framework therefore attempts to shift the focus from:

"How aggressively can energy be restricted?"

toward:

"Is sufficient energy available to support stable physiological function?"

Energy Per Kilogram

At its core, EPK contextualises energy intake relative to body mass.

The framework proposes that absolute calorie values alone may provide limited insight into true physiological state because identical calorie intakes can represent dramatically different energetic conditions across individuals.

DOI: 10.5281/zenodo.22673106

For example:

2000 kcal/day may represent substantial restriction for a highly active larger individual,

approximate maintenance for another,

or surplus intake for a smaller sedentary individual.

EPK therefore attempts to standardise energy intake relative to body mass in order to better estimate:

physiological sufficiency,

compensatory risk,

recovery status,

and behavioural stability.

This concept parallels existing sports physiology literature, where energy availability relative to body size or fat-free mass is already considered critical for maintaining endocrine function, recovery, and performance.[20,21,22,23]

Physiological States and Functional Capacity

The EPK framework proposes that chronic low-energy states may progressively impair physiological function.

Rather than viewing the body as a passive calorie equation, the framework conceptualises the organism as a dynamic energy-regulating system attempting to maintain equilibrium.

When energy availability becomes insufficient, compensatory adaptations may emerge, including:[20,21,22,23]

increased appetite,

food preoccupation,

fatigue,

reductions in spontaneous movement,[27,28]

impaired recovery,

reductions in metabolic rate,[10,11,24,25,26]

endocrine alterations,

mood instability,

and oscillatory eating behaviour.[8,9]

The author's MSc findings suggested that greater restriction intensified behavioural instability.

The EPK model therefore proposes that many individuals attempting chronic restriction may unknowingly exist in physiologically underpowered states.

Importantly, the framework does not suggest that all individuals should remain in chronic surplus intake. Rather, it proposes that:

DOI: 10.5281/zenodo.22673106

severe and prolonged low-energy states may impair sustainable function,

and that improving physiological sufficiency may stabilise behaviour and improve long-term outcomes.

Traffic Light Readiness States

One practical application of the EPK framework is the use of traffic-light readiness states to categorise physiological stability and compensatory risk.

These states are not intended as rigid medical classifications, but rather as practical educational tools integrating:

energy availability,[20,21,22,23]

recovery,

behavioural stability,

movement capacity,

appetite regulation,[4,12,13,14,15,16,17]

and subjective biofeedback.

Red State

Represents severe physiological strain or low-energy availability.[20,21,22,23]

Characteristics may include:

chronic fatigue,

high food preoccupation,

poor sleep,

mood instability,

reduced recovery,

low spontaneous movement,[27,28]

compensatory overeating,

and poor training tolerance.

Individuals in this state may demonstrate high compensatory drive and poor adherence capacity.

Amber State

Represents partial improvement but continued instability.

Individuals may demonstrate:

inconsistent appetite regulation,[4,12,13,14,15,16,17]

fluctuating energy levels,

intermittent cravings,

moderate recovery impairment,

DOI: 10.5281/zenodo.22673106

and continued behavioural oscillation.[8,9]

Green State

Represents relatively stable physiological function.

Characteristics may include:

improved recovery,

more stable appetite,

improved behavioural consistency,

greater spontaneous movement,[27,28]

improved sleep quality,

and reduced compensatory drive.

Gold State

Represents high functional capacity and resilience.

This state may include:

strong recovery capacity,

stable behavioural regulation,

robust movement tolerance,

high spontaneous activity,

stable appetite signalling,[4,12,13,14,15,16,17]

and improved physiological flexibility.

The framework proposes that many individuals attempting chronic restriction remain trapped within red or amber states whilst pursuing further deficits, potentially worsening compensatory physiology.

Biofeedback and Physiological Interpretation

A key distinction within the EPK framework is the emphasis placed upon physiological biofeedback rather than weight change alone.

Traditional dieting models often prioritise:

scale weight,

weekly deficits,

and calorie adherence.

In contrast, the EPK model proposes that important physiological markers may include:

sleep quality,

spontaneous movement,[27,28]

recovery,

DOI: 10.5281/zenodo.22673106

mood,

training tolerance,

libido,

appetite stability,

warmth,

cognitive function,

and behavioural consistency.

These markers may provide insight into whether energy availability is sufficient to support sustainable function.[20,21,22,23]

The framework therefore attempts to integrate:

objective measures,

subjective physiological feedback,

and behavioural patterns

rather than relying solely upon body weight changes.

Progressive Restoration Rather Than Aggressive Restriction

The EPK model proposes that gradual restoration of energy availability may improve:[20,21,22,23]

behavioural stability,

recovery,

adherence,

and long-term outcomes.

This position aligns with evidence demonstrating that aggressive restriction frequently increases compensatory drive and long-term regain risk.

The framework therefore favours:

gradual adjustments,

sustainable intake progression,

and stabilisation of physiological function

rather than repeated cycles of severe restriction.

This concept is supported by starvation and adaptive thermogenesis research demonstrating that the body actively defends against prolonged energy insufficiency.[10,11,24,25,26]

Integration With Constrained Energy Theory

The EPK framework also aligns conceptually with constrained energy expenditure models.[10,11,24,18,19,25,26]

DOI: 10.5281/zenodo.22673106

If total energy expenditure is biologically regulated within constrained ranges, aggressive attempts to increase deficits may produce compensation through:[10,11,24,25,26]

reduced movement,

reduced metabolic activity,

endocrine adaptation,

or behavioural compensation.

Under this model, chronic restriction may not simply continue producing linear weight loss. Instead, the organism may progressively adapt in order to preserve energy availability.[20,21,22,23]

The EPK framework therefore proposes that:

improving physiological sufficiency,

movement capacity,

and behavioural stability

may ultimately represent more sustainable long-term strategies than escalating restriction severity.

EPK as a Practical Clinical and Educational Tool

The framework is not intended to replace established nutritional science or energy balance principles. Rather, it attempts to provide a more physiology-aware model integrating:

energy availability,[20,21,22,23]

adaptive thermogenesis,[10,11,24,25,26]

constrained energy expenditure,[10,11,24,18,19,25,26]

behavioural compensation,

and metabolic individuality.

EPK may therefore serve as:

an educational framework,

a behavioural interpretation model,

and a practical tool for assessing compensatory risk and physiological sufficiency.

Further validation research is required to determine:

optimal EPK ranges,

clinical applications,

predictive utility,

and long-term health outcomes.

DOI: 10.5281/zenodo.22673106

However, the present framework offers a potential explanation for why many individuals struggle to sustain aggressive dietary restriction despite strong motivation and repeated behavioural effort.

7. Discussion

Reconsidering Dietary Adherence

The findings presented throughout this paper suggest that poor dietary adherence may be more physiologically driven than traditionally assumed.[6,7]

Conventional dieting models frequently frame adherence failure as a behavioural problem characterised by:

weak motivation,

poor discipline,

low self-control,

or insufficient commitment.

However, both the author's MSc findings and broader physiological literature suggest that many compensatory behaviours may emerge as predictable biological responses to insufficient energy availability.[20,21,22,23]

Participants within the MSc research consistently failed to achieve intended calorie deficits, and larger deficits were associated with greater fluctuations in intake behaviour. Importantly, these patterns did not appear random. Rather, they reflected oscillatory attempts to restrict intake followed by periods of compensatory overconsumption.[8,9]

This interpretation aligns closely with:

adaptive thermogenesis literature,[10,11,24,25,26]

appetite regulation research,[4,12,13,14,15,16,17]

constrained energy expenditure models,[10,11,24,18,19,25,26]

and starvation physiology.

Collectively, these findings challenge simplistic notions that humans respond linearly to calorie prescriptions.

The Body Appears to Defend Energy Availability

One of the central arguments emerging from this paper is that the human organism appears to actively defend energy availability.[20,21,22,23]

During periods of restriction, numerous compensatory adaptations emerge, including:

reductions in metabolic rate,[10,11,24,25,26]

increased hunger,

altered endocrine signalling,

DOI: 10.5281/zenodo.22673106

food preoccupation,

reduced spontaneous movement,[27,28]

and behavioural instability.

The Minnesota Starvation Experiment remains one of the clearest demonstrations of this phenomenon. Despite high participant motivation and controlled laboratory conditions, prolonged energy restriction produced profound behavioural and psychological alterations, including:

binge eating,[8,9]

emotional instability,

compulsive food thoughts,

irritability,

and reduced physical activity.

Importantly, many modern dieting approaches may unknowingly recreate milder versions of these same physiological states through repeated cycles of aggressive restriction.

The present findings therefore support the hypothesis that many “diet failures” may represent physiological compensation rather than moral or behavioural inadequacy.

The Limitations of Static Calorie Models

Another major implication of the present work concerns the limitations of static calorie prescription models.

Traditional dietary interventions frequently utilise:

fixed calorie targets,

standardised calorie deficits,

and predictive equations based upon population averages.[29,30,33,25]

However, substantial inter-individual variability exists in:

resting metabolic rate,[10,11,24,25,26]

movement behaviour,

adaptive thermogenesis,[10,11,24,25,26]

recovery demands,

endocrine status,

and constrained energy responses.[18,19]

Consequently, identical calorie prescriptions may create dramatically different physiological conditions across individuals.

The author’s MSc findings highlighted this issue directly, noting that inaccuracies in estimated energy requirements may contribute to adherence failure and compensatory behaviour.[29,30,33,25]

DOI: 10.5281/zenodo.22673106

The EPK framework was therefore developed partly in response to these limitations.

Rather than viewing energy intake as a universal numerical target, EPK attempts to contextualise intake relative to:

body mass,

physiological demand,

behavioural stability,

and functional capacity.

Human Function Versus Weight Reduction Alone

A further implication of the present framework is the potential need to shift emphasis away from purely weight-centric models toward function-centric physiology.

Many dietary interventions prioritise:

scale weight reduction,

short-term deficits,

and rapid weight loss.

However, sports physiology literature consistently demonstrates that low-energy availability impairs:[20,21,22,23]

recovery,

endocrine function,

mood,

immune activity,

and performance.

The present paper argues that these physiological principles may extend beyond athletic populations.

Humans do not appear to function optimally in chronically energy-depleted states.

Indeed, many symptoms commonly normalised within modern society:

fatigue,

poor sleep,

low motivation,

reduced movement,

irritability,

food obsession,

and impaired recovery

may partially reflect chronic insufficient energy availability in susceptible individuals.[20,21,22,23]

DOI: 10.5281/zenodo.22673106

From this perspective, successful long-term health management may depend less upon maximising restriction severity and more upon maintaining sufficient physiological energy availability to support stable human function.[20,21,22,23]

Behavioural Stability as a Physiological Outcome

A key proposition of the EPK framework is that behavioural stability may itself be physiologically mediated.

Traditional approaches frequently assume that:

stable adherence produces physiological improvement.

However, the present framework proposes that:

improving physiological sufficiency may itself improve adherence capacity.

This distinction is important.

If compensatory overeating emerges partly from biological attempts to restore energy availability, then repeated escalation of restriction may paradoxically worsen adherence by intensifying compensatory drive.[20,21,22,23]

The author's MSc findings appeared consistent with this interpretation, as larger calorie deficits predicted greater intake instability.

Consequently, gradual restoration of physiological stability may:

reduce compensatory behaviour,

improve behavioural consistency,

and enhance long-term sustainability.

Integration With Constrained Energy Theory

The EPK framework also aligns conceptually with constrained energy expenditure research.[10,11,24,18,19,25,26]

Pontzer's constrained energy model proposes that total daily energy expenditure may be biologically regulated within constrained ranges rather than increasing linearly indefinitely with activity.[10,11,24,18,19,25,26]

If correct, this has major implications for traditional "eat less, move more" approaches.

Attempts to create progressively larger deficits may trigger:

metabolic adaptation,

reductions in spontaneous movement,[27,28]

endocrine compensation,

or increased appetite drive.

Under such conditions, escalating restriction may produce diminishing returns whilst increasing physiological stress and behavioural instability.

The present framework therefore proposes that:

DOI: 10.5281/zenodo.22673106

movement capacity,

recovery,

spontaneous activity,

and physiological resilience

may represent important long-term markers of sustainable health.

Potential Applications of the EPK Framework

Although further validation research is required, the EPK framework may have practical applications within:

weight management,

health coaching,

sports performance,

recovery assessment,

and long-term behavioural adherence.

Potential applications may include:

identifying compensatory risk,

monitoring recovery capacity,

improving physiological awareness,

assessing behavioural stability,

and reducing reliance upon excessively restrictive interventions.

The framework may also help shift educational focus away from:

moral interpretations of eating behaviour,

toward:

physiological interpretation of compensatory responses.

This perspective may reduce stigma surrounding dieting failure whilst encouraging more individualised and sustainable approaches.

Future Research Directions

The present paper represents a conceptual and observational framework rather than definitive proof of causality.

Future research should aim to investigate:

relationships between EPK and compensatory behaviour,

associations between EPK and metabolic health markers,

behavioural stability across different energy availability states,[20,21,22,23]

longitudinal adherence outcomes,

and physiological responses across different populations.

Controlled intervention studies incorporating:

indirect calorimetry,[29,30,33,25]

doubly labelled water,

hormonal assessment,

movement monitoring,

and psychological measures

would substantially strengthen understanding of these relationships.

In particular, future work should determine whether maintaining higher physiological energy availability improves:[20,21,22,23]

long-term adherence,

spontaneous movement,[27,28]

recovery,

mood,

and sustainable body composition outcomes.

8. Limitations

Although the present paper integrates observational findings, physiological literature, and conceptual modelling into the proposed EPK framework, several important limitations must be acknowledged.

First, the original MSc research utilised a relatively small sample size, with only 21 participants included within the final analysis following exclusions for incomplete dietary recording and protocol adherence. This limited statistical power and may have reduced the ability to detect significant relationships between psychological variables, compensatory health beliefs, motivation, and adherence outcomes.

Additionally, the study relied heavily upon self-reported dietary intake through MyFitnessPal logging. Self-reported dietary data are well known to contain inaccuracies, including:[31,32]

under-reporting,[31,32]

omitted foods,

inaccurate portion estimation,

and inconsistent recording behaviours.

Consequently, true energy intake and adherence may have differed from recorded values.

The use of predictive equations to estimate energy requirements also represents an important limitation. As discussed throughout both the MSc research and present paper, standardised predictive equations may inadequately account for:[29,30,33,25]

DOI: 10.5281/zenodo.22673106

metabolic individuality,

adaptive thermogenesis,[10,11,24,25,26]

constrained energy expenditure,[10,11,24,18,19,25,26]

recovery demands,

and inter-individual physiological variability.

It is therefore possible that some participants were prescribed deficits substantially larger or smaller than intended.

Furthermore, the correlational nature of the original study prevents causal conclusions from being established. Although larger calorie deficits were associated with greater fluctuations in intake behaviour, causality cannot be definitively inferred. Additional experimental research is required to determine whether increasing restriction directly intensifies compensatory physiology and behavioural instability.

The EPK framework itself should presently be viewed as a conceptual and practical model rather than a clinically validated diagnostic system.

At present:

definitive EPK thresholds have not been experimentally established,

optimal ranges remain provisional,

and physiological responses likely vary considerably between individuals.

Variables such as:

body composition,

sex,

age,

hormonal status,

physical activity,

recovery demands,

psychological stress,

and health status

may all substantially alter energy requirements and compensatory responses.

Additionally, whilst the framework draws conceptual support from sports energy availability literature, important distinctions exist between athletic and non-athletic populations. Athletes often exhibit substantially greater movement demands and performance sensitivity, meaning direct translation of thresholds between populations should be approached cautiously.[20,21,22,23]

Another limitation concerns the complexity of human eating behaviour itself. Dietary adherence and compensatory behaviour are influenced not only by physiology, but also by:[6,7]

social environment,

food accessibility,

emotional state,

behavioural conditioning,

cultural norms,

sleep quality,

stress exposure,

and psychological factors.

The present framework does not suggest that physiology is the sole determinant of eating behaviour. Rather, it proposes that physiological state may play a larger role in behavioural regulation than is often acknowledged within conventional dieting models.

Finally, the EPK framework may risk oversimplification if applied rigidly or dogmatically. The intention of the model is not to replace established nutritional science, nor to imply that energy balance principles are invalid. Instead, the framework attempts to integrate:

energy availability,[20,21,22,23]

adaptive physiology,

constrained energy expenditure,[10,11,24,18,19,25,26]

metabolic individuality,

and behavioural compensation

into a more physiology-aware interpretation of long-term dietary adherence.[6,7]

Future research utilising:

indirect calorimetry,[29,30,33,25]

doubly labelled water,

hormonal profiling,

movement monitoring,

and long-term intervention studies

will be necessary to determine the scientific validity, predictive utility, and clinical applications of the EPK model.

9. Conclusion

The present paper explored compensatory responses to energy restriction through the integration of original MSc research findings, modern physiological literature, sports energy availability research, and constrained energy expenditure theory.[10,11,24,18,19,25,26]

Although the original MSc study failed to demonstrate statistically significant relationships between compensatory health beliefs, motivational orientation, and dieting outcomes, important behavioural patterns emerged. Participants consistently failed to sustain intended calorie deficits,

and larger intended deficits were associated with greater fluctuations in intake behaviour. These findings suggested that aggressive dietary restriction may destabilise eating behaviour rather than improve adherence.

Importantly, the observed oscillatory behaviours appeared consistent with compensatory physiological responses to insufficient energy availability.[8,9,20,21,22,23]

The broader physiological literature reviewed throughout this paper further supports the concept that the human organism actively defends energy availability through:[20,21,22,23]

adaptive thermogenesis,[10,11,24,25,26]

appetite regulation,[4,12,13,14,15,16,17]

endocrine adaptation,

constrained energy expenditure,[10,11,24,18,19,25,26]

behavioural compensation,

and reductions in energetically expensive physiological processes.

Collectively, these findings challenge simplistic interpretations of dieting failure as merely the consequence of weak willpower or poor discipline.

Instead, the evidence suggests that many behaviours commonly associated with “poor adherence” may represent biologically mediated attempts to restore sufficient energy availability and maintain physiological equilibrium.[20,21,22,23]

The EPK (Energy Per Kilogram) framework emerged conceptually from these observations.

Rather than focusing exclusively upon static calorie deficits and body weight reduction, the framework proposes that energy intake should be interpreted relative to:

body mass,

physiological demand,

recovery capacity,

behavioural stability,

and functional output.

Drawing parallels from sports physiology and energy availability research, the paper argued that all humans-not solely athletes-require sufficient energy availability to support:[20,21,22,23]

neurological function,

recovery,

endocrine health,

spontaneous movement,[27,28]

psychological stability,

and sustainable behavioural regulation.

DOI: 10.5281/zenodo.22673106

From this perspective, many symptoms frequently normalised within modern dieting culture and aging populations may partially reflect chronic low-energy physiological states.

The framework therefore proposes that sustainable health management may depend less upon maximising restriction severity and more upon maintaining sufficient physiological energy availability to support stable human function.[20,21,22,23]

Importantly, the EPK model does not reject energy balance principles or deny that body composition changes occur through alterations in energy balance. Rather, it proposes that human energy regulation is adaptive, biologically defended, and heavily influenced by compensatory physiology.

Consequently, interventions that ignore these compensatory mechanisms may inadvertently increase:

behavioural instability,

metabolic adaptation,

food preoccupation,

and long-term regain risk.

The present paper represents an initial conceptual and observational model rather than definitive proof of causality. Considerable further research is required to:

validate EPK thresholds,

examine relationships between EPK and compensatory behaviour,

assess long-term adherence outcomes,

investigate endocrine and metabolic responses,

and determine clinical applications across different populations.

Nevertheless, the findings presented throughout this paper suggest that physiology-aware approaches to energy management may offer a more sustainable framework for understanding long-term dietary adherence and human function than traditional restriction-centric models alone.[6,7]

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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.