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Post-competition recovery in natural physique athletes: body composition, metabolic adaptation, and refeeding responses

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ABSTRACT

Background: Post-competition refeeding in physique athletes is poorly understood. We aimed to characterize physiological and psychological changes in natural physique athletes across contest preparation and a 12-week recovery period, and to explore the influence of post-competition refeeding strategies.

Methods: Nineteen natural physique athletes (8 male, 11 female) were assessed at baseline (~21 weeks pre-competition; T0), 1–2 weeks pre-final competition (Tpre), and 2, 6, and 12 weeks post-competition (T2, T3, T4). Measures included body composition (DXA), resting metabolic rate (RMR), thyroid hormones (FSH, FT3, FT4), absolute strength (IMTP peak force), and psychometric questionnaires (POMS, ASSQ, EDE-QS).

Results: Body weight decreased from T0 to Tpre (−7.1 kg [−8.3, −5.9]), driven primarily by FM loss (−5.8 [−6.8, −4.8]), with modest FFM loss (−1.7 [−2.6, −0.9]). Both FM and FFM rebounded predominantly within the first 6 weeks post-competition (Tpre→T3: +3.4 [2.3, 4.4] and +2.7 [1.8, 3.6], respectively). By T4, FM was not clearly different from T0 (−0.8 [−1.8, 0.3], while FFM exceeded T0 (+1.6 [0.7, 2.5]). RMR·FFM^{-1} showed a small, uncertain reduction from T0 to Tpre (−0.9 kcal·kgFFM^{-1}·day^{-1} [−2.7, 0.9]), followed by increases from Tpre to T4 (+2.4 [0.7, 4.1]). Thyroid hormones decreased from T0 to Tpre (FT3: −1.4 [−1.8, −0.9], FT4: −1.4 [−2.6, −0.2]) and returned within reference ranges by T4. Strength was broadly maintained, while mood and sleep worsened from T0 to Tpre, and improved by T4. Eating-disorder symptom severity was highest during preparation and declined across the recovery period. In exploratory Bayesian modelling, larger post-competition increases in energy intake were associated with greater recovery of adjusted RMR.

Conclusions: Contest preparation was accompanied by fat loss, thyroid hormone suppression, and modest reductions in RMR, with recovery characterized by early increases in RMR and tissue restoration following competition. Larger post-competition increases in energy intake were associated with faster recovery of adjusted RMR, although FM regain occurred concurrently. Post-competition recovery should be treated as an active, structured phase, with refeeding individualized to athlete goals and psychological readiness and guided by multi-system monitoring rather than RMR alone. Athletes and coaches should plan ahead for this phase, with structured increases in food intake, realistic expectations around fat gain, and avoidance of unnecessarily prolonged restriction that may delay physiological recovery.

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Resting metabolic rate; bodybuilding; low energy availability; adaptive thermogenesis; resistance-trained athletes

1. Introduction

Physique athletes manipulate body composition to achieve extreme leanness, entering a state of low energy availability (LEA), where dietary energy is insufficient to support energetic costs of exercise and normal physiological function [1,2]. LEA drives Relative Energy Deficiency in Sport (RED-S), with consequences across metabolic, endocrine, and reproductive systems, and performance and psychological health [3]. Physique contest preparation is associated with reductions in fat mass (FM) and fat-free mass (FFM), alongside altered

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resting metabolic rate (RMR) and endocrine function [4–7]. Reductions in RMR may extend beyond that expected from changes in FM and FFM, reflecting mass-independent suppression of resting energy expenditure, or adaptive thermogenesis [8]. Such instances of metabolic suppression are relevant as they can reduce total daily energy expenditure (TDEE), increasing the energy deficit required for continued weight loss [5].

The transition out of contest preparation is physiologically and psychologically challenging; athletes attempt to restore energy availability while managing changes in appetite and body composition [9]. Following prolonged energy restriction, coordinated changes in appetite-regulating hormones (e.g. reduced leptin and insulin; elevated ghrelin) and behavioral pressures promote a heightened biological drive to eat, commonly termed rebound hyperphagia [10–12]. Concurrently, persistent metabolic adaptations, including suppressed TDEE, create a physiological environment that favors rapid fat storage [12]. This can result in "fat overshooting," whereby fat mass may be preferentially restored beyond pre-diet levels [9,12].

In physique athletes, these responses are further compounded by endocrine disturbances, including reductions in testosterone and triiodothyronine (T3), alongside elevations in cortisol, which reinforce susceptibility to rapid post-contest increases in body mass and FM [13–15]. While some FM regain may be necessary to reverse LEA-related disturbances, unwanted fat accumulation may have implications for athlete health, psychological well-being, and future competitive preparation [5]. Athletes may thus extend diet duration or increase energy deficit, compounding repeated exposure to LEA and its physiological and psychological disturbances. Qualitative accounts further suggest many athletes struggle to regulate post-competition intake, potentially eroding self-efficacy and reinforcing weight cycling [16].

Evidence informing post-competition refeeding is limited, but energy intake must increase post-competition to restore FM, FFM, and endocrine function [9]. However, the magnitude and timing of energy intake increase following physique competition remain poorly defined. Applied strategies include gradual, stepwise increases in intake ("reverse dieting"), an immediate return to estimated maintenance, or ad libitum approaches [9]. Reverse dieting is often used to limit rapid fat gain and fat overshooting; however, it remains controversial. By maintaining relatively low energy intake, it may prolong LEA and delay recovery of metabolic and endocrine function [17]. Thus, strategies intended to minimize fat gain may compromise physiological recovery. Consistent with this, case studies show heterogeneous recovery trajectories [13,18,19]. It remains unclear whether larger post-competition increases in energy are associated with better recovery of outcomes such as RMR and endocrine function, beyond that explained by changes in body composition, or whether increased intake primarily influences FM regain.

Therefore, this study aimed to characterize longitudinal changes in body composition and RMR relative to FFM ($\text{RMR}\cdot\text{FFM}^{-1}$) across contest preparation and a 12-week post-competition recovery period in natural physique athletes. Secondary aims were to (i) examine mass-independent RMR patterns and (ii) explore whether greater post-competition increases in energy (relative to the final pre-competition assessment) were associated with faster recovery of adjusted RMR, interpreted alongside changes in body composition and endocrine markers.

2. Methods

2.1. Participants and study design

This prospective, observational study is reported in accordance with STROBE guidelines [20] and approved by the University of Canberra ethics board (HREC-13593). Natural physique athletes were monitored across contest preparation and 12-week post-competition refeeding in Sydney ($n = 7$) or Canberra ($n = 12$), Australia.

Eligibility criteria included: (i) aged 18–45 years; (ii) self-declared natural athlete (compete in drug tested federations (ICN-ACT, NBA Australia) and affirmed compliance with anti-doping policies; (iii) previous experience preparing for≥ 1 natural bodybuilding show (any category) in the past 10 years, (iv) committed to competing in Season B 2024 (September to November), and (v) currently >10% above estimated stage weight. Athletes were excluded if they had prior clinically diagnosed eating disorders, used medications or had medical conditions possibly affecting body composition, appetite, heart rate, metabolic rate, and/or endocrine function. Recruitment concluded at the end of May 2024, after which additional participants were unlikely to be able to complete the baseline (T0) assessment > 10% above estimated stage weight.

Twenty-two athletes enrolled (9 male, 13 female); three withdrew prior to competition, leaving 19 athletes (8 male, 11 female) contributing ≥1 post-competition assessment. All participants provided written informed consent.

Athletes followed their own coach's guidance or received dietetic support from an Accredited Sports Dietitian during refeeding. Assessments occurred relative to each athlete's competition calendar: T0 (~15–25 weeks pre-competition), T1 (1–2 weeks pre-competition), T2 (2 weeks post), T3 (6 weeks post), and T4 (12 weeks post) (Figure 1). For athletes competing across ≥4 weeks, an additional pre-final competition visit (T1.5) was scheduled 1–2 weeks before the final show to capture their leanest state. Where carbohydrate loading was performed, T1/T1.5 visits were scheduled prior to loading to minimize acute effects on body composition [21,22]. Some missing data occurred due to scheduling conflicts or missed assessments (per-outcome sample sizes in Figure 2).

2.2. Food diaries

Seven-day weighed food diaries were collected at T0, T1, and T1.5. From the final competition through T4, athletes recorded daily intake using a food tracking app with optional photo and/or written diaries. Participants were encouraged to use Easy Diet Diary (EDD, Xyris Software Australia Pty Ltd); however, they could choose alternatives (Cronometer, MyFitnessPal) to support adherence. The lead researcher (Accredited Sports Dietitian) provided initial standardized instruction to support accurate weighing, portion estimation, and app entry. Accuracy was reviewed at face-to-face laboratory visits and post-competition, supplemented by weekly check-ins to review logs, cross-check app entries against photo/written records, and resolve discrepancies. Goldberg EI:BMR cut-offs were considered but not applied as intakes below RMR were expected consequent to time-bound dieting to achieve extreme leanness [23]. Dietary intake plausibility and accuracy were supported by the bodybuilder participants' prior dieting experience, structured familiarization, and ongoing dietitian-led reviews.

2.3. Training diaries

Seven-day training logs were collected at each time point consisting of session type, timing, and Borg's 1–10 rating of perceived exertion (RPE) [24]. Weekly resistance-training load was computed as sets*reps*RPE, and cardio load as RPE*time, with total weekly load computed as the sum. Participants also recorded daily step counts where available. Mode-specific training data (resistance, cardio, and steps) are presented in Supplementary Tables S2 and S3.

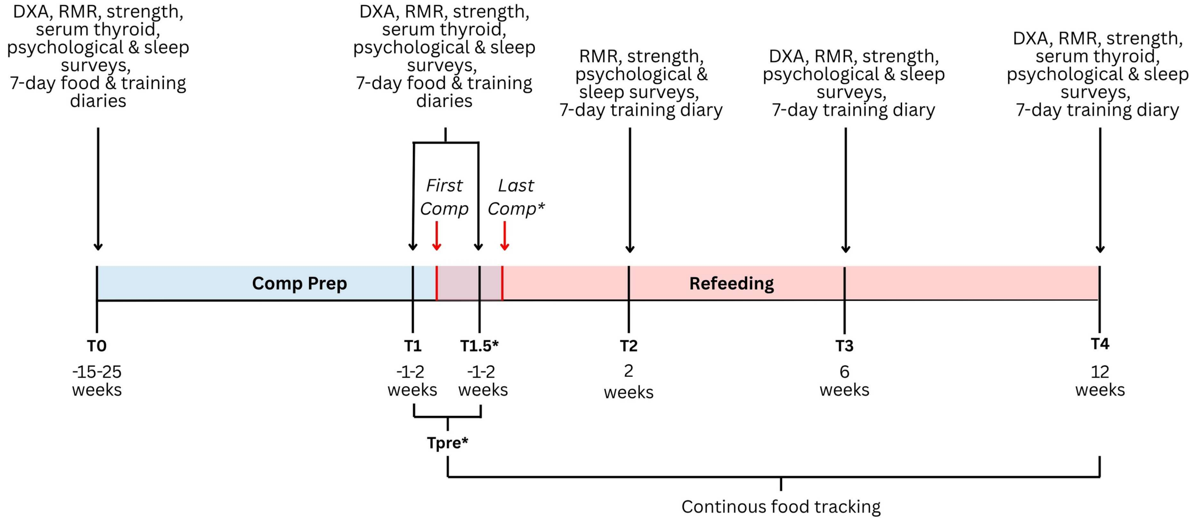

Figure 1. Study timeline. DXA = dual-energy X-ray absorptiometry; RMR = resting metabolic rate. *An additional time-point (T1.5) was added for athletes who competed in multiple shows over a period of ≥4 weeks. The final pre-competition assessment (Tpre) was defined as T1.5 where available, and otherwise T1.

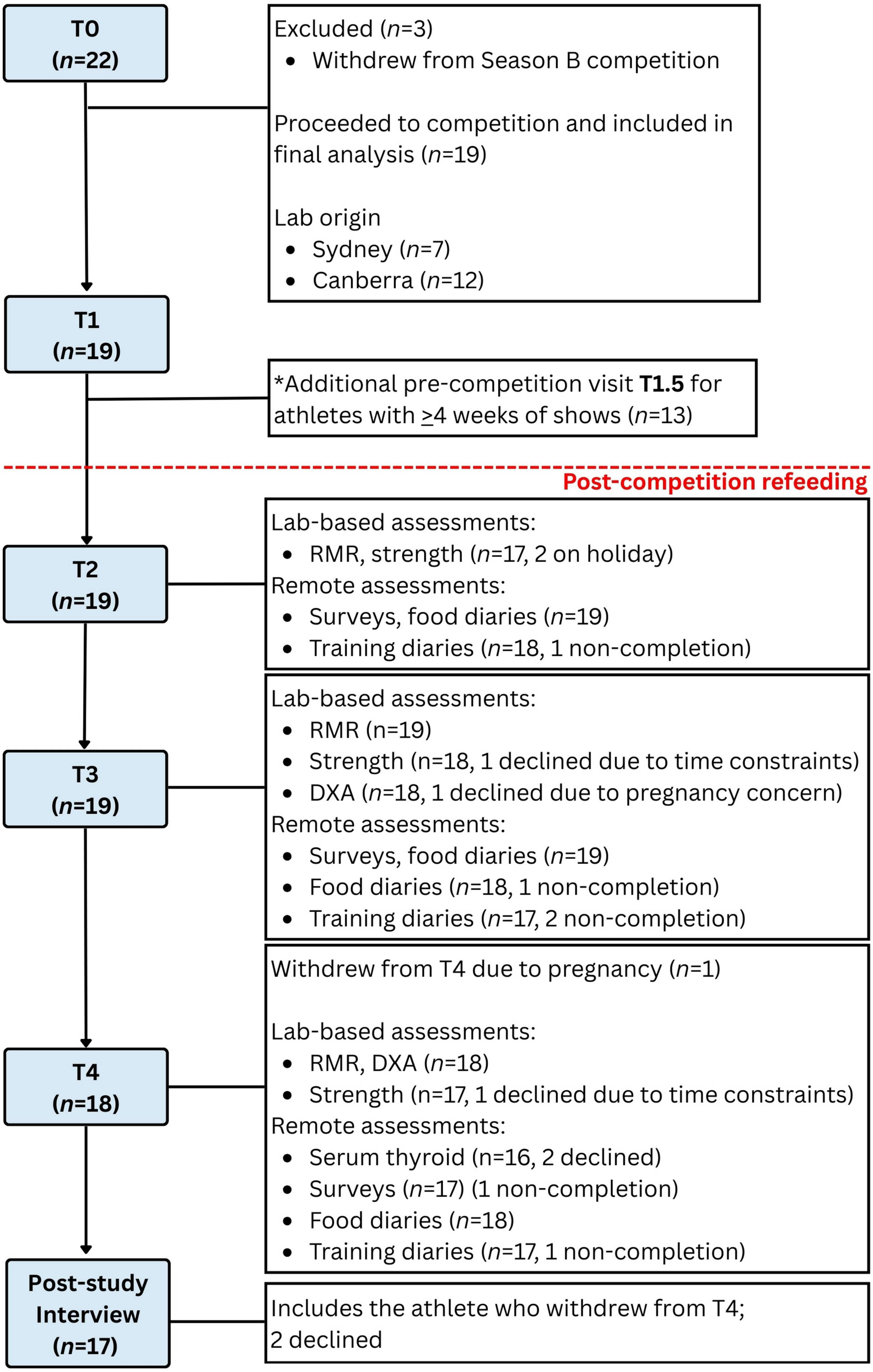

Figure 2. Flow of participants and data completeness across the study timeline. Per-outcome sample sizes are shown for lab-based and remote assessments. RMR = resting metabolic rate; DXA = dual-energy X-ray absorptiometry.

2.4. Body composition

Body composition was assessed using Dual-Energy X-ray Absorptiometry (DXA) at T0, T1, T1.5, T3, and T4 (GE Lunar Prodigy, GE Encore Software; V.17, GE Healthcare or Medix DR, Eazix Software; V5 0.3.2, depending on site) by a certified densitometrist in the morning after an 8-h fast following the Nana protocol [25]. DXA was not performed at T2 as glycogen and hydration shifts immediately post-competition can meaningfully increase DXA lean estimates by ~2-3% [21,22]. For continuous trajectory analysis, T2 FFM was estimated within-athlete via time-weighted interpolation between T1/1.5 and T3 DXA assessments using participant-specific assessment timing. FM and % body fat at T2 were derived from lab-measured body mass and DXA-derived bone mineral content. Interpolated T2 body-composition values were used only in analyses requiring alignment of body composition with longitudinal RMR measures (i.e. adjusted RMR and Bayesian models) and were not used in analyses restricted to measured DXA time points (FFM and FM models). Per prior testing, lean body mass CV was 0.7%, with least significant change (LSC) of 1.9% (95% CI). CV for FM was 1.3%, with a LSC of 3.7% (95% CI) [26].

2.5. Resting metabolic rate

RMR was measured at each time point (5–10 am) via indirect calorimetry (ParvoMedics TrueOne 2400 with mouthpiece/nose clip or Cosmed Quark CPET with face mask, by site). Testing occurred in a quiet, dimly lit, temperature-controlled environment (20 °C–24 °C). Devices were calibrated before tests using certified gas mixtures and a 3-L syringe. Participants fasted >10 h and avoided caffeine, alcohol, nicotine, and exercise 10 h prior in accordance with the Compher protocol [27]. After 10 min supine rest and 15 min habituation, gas exchange was recorded for 26 min with the first 6 min discarded [28]. Energy expenditure was calculated using the adapted nitrate-free Weir equation [29]. To support clinical interpretation of within-individual change, changes in $\text{RMR}\cdot\text{FFM}^{-1}$ were classified using our laboratory-derived LSC threshold of $2.9\ \text{kcal}\cdot\text{kgFFM}^{-1}\cdot\text{day}^{-1}$. DXA and RMR assessments were performed on the same morning under standardized conditions.

2.6. Strength

Strength was assessed via isometric midthigh pull (IMTP) at each time point using an adjustable rack and dual force plates (Vald ForceDecks, Vald Performance, Brisbane, Australia) following the Comfort protocol [30]. After a standardized warm-up and submaximal efforts (3 × 3 s trials at 50%, 75%, and 90% perceived maximal effort with 60 s rest), athletes performed three maximal trials using lifting straps with 2 min rest. If peak force continued increased across all 3 trials, additional trials occurred until values differed by <250 Newtons (*N*). The highest peak vertical force (N) was retained. Tests were scheduled last in each lab visit to avoid interference with DXA and RMR.

2.7. Thyroid and menstrual function

Thyroid-stimulating hormone (TSH), triiodothyronine (FT3), and thyroxine (FT4), were collected from serum at T0, T1, T1.5, and T4, via a commercial pathology provider (Intelligent Screening (Pty) Ltd, Australia). Blood sampling was completed within ±7 days of laboratory assessments. Participants attended in the morning, fasted (8 h), ≥12 h post vigorous exercise [31]. Reference ranges were: TSH $0.5\text{–}4.0\ \text{mIU}\cdot\text{L}^{-1}$, FT4 $10\text{–}20\ \text{pmol}\cdot\text{L}^{-1}$, and FT3 $3.5\text{–}6.5\ \text{pmol}\cdot\text{L}^{-1}$. Menstrual regularity, frequency, symptoms, and hormonal contraceptive use were reported via online questionnaire at each time point (Qualtrics Core XM) [32].

2.8. Psychometrics

Mood and sleep were assessed at each time point using the Abbreviated Profile of Mood States questionnaire (POMS) [33] and the Athlete Sleep Screening Questionnaire (ASSQ) [34]. The POMS (40 items) rates mood states on a 5-point Likert scale, providing a Total Mood Disturbance (TMD) score, calculated as an index of mood disturbance (range 68 to 228; higher TMD indicates greater disturbance). The ASSQ provides a Sleep Difficulty Score (SDS) by summing 5 items related to sleep timing and quality (Likert-type ordinal response scales; range 0 to 17; higher scores indicate greater sleep difficulties). Eating-related cognition and behavior disturbances were evaluated at T0, T2, T3, and T4 using the Eating Disorder Examination-Questionnaire Short (EDE-QS) consisting of 12, 4-point Likert scale items providing a global score (range 0–36; higher scores indicating greater eating-disorder symptom severity) [35].

2.9. Athlete interviews

Semi-structured Zoom interviews (~60 min) were conducted after the study to record participant experiences. Interviews were guided by a semi-structured interview guide but remained participant-led, and flexible [36]. Questions (Supplementary 2) explored how athletes structured their diet, tools for adherence, challenges, perceived health and mental function changes, and post-competition recovery refeeding practices. Audio was transcribed using Zoom transcription software and checked for accuracy by CB. Data were analyzed using Braun & Clarke's reflexive thematic analysis [37]. CB and MM independently read, and coded transcripts before consolidating codes and themes through discussion [38]. Themes were critically reviewed by all authors to ensure data reflected the participants' voices [39].

2.10. Data analysis

Raw data were exported to Microsoft Excel (v16.98) and analyzed in R (v4.4.1, R Core Team). Descriptive statistics (mean ± SD) summarized participant characteristics and outcomes by time point. Analyses included athletes who completed ≥1 post-competition assessment ($n = 19$). To standardize athletes with and without an additional pre-competition assessment, the final pre-competition assessment (Tpre) was defined as T1.5 where available, and otherwise T1. For descriptive figures, dietary intake was summarized as 7-day mean intake at pre-competition time points and continuous weekly means across 12-weeks post-competition. For inferential analyses, post-competition intake was summarized as the mean daily intake across recovery intervals (Tpre → T2, T2→T3, T3→T4) and expressed as change from Tpre (Δkcal). Energy intake was expressed as kcal·day^{-1} and kcal·kgFFM^{-1}·day^{-1}. Macronutrient intake was expressed as g·kg·day^{-1} and is shown in Supplementary Tables S2 and S3.

Longitudinal changes in RMR·FFM^{-1}, absolute RMR, and body weight were examined using linear mixed-effects models with participant-specific random intercepts and fixed effects for sex and laboratory site. Time was specified a priori using a piecewise structure (linear term for contest preparation; natural cubic spline for post-competition), allowing non-linear recovery trajectories while limiting overfitting. FFM and FM were analyzed with time specified as a categorical factor (T0, Tpre, T3, T4) to align with available DXA assessments. Model-estimated means were summarized at T0, Tpre, and 2, 6, and 12 weeks post-competition, with planned contrasts for T0 → Tpre, Tpre → post-competition time points, and T0 → T4. Models were fitted using REML and checked using residual and Q–Q plots. Results are reported with 95% confidence intervals. Sex-by-time interaction terms were explored in sensitivity analyses but were not included in the primary model due to the modest sample size and lack of evidence for sex-by-time interaction. Behavioral variables (energy intake and training load), and secondary physiological and psychological measures (thyroid function, strength, and psychometric outcomes), were summarized descriptively using within-participant contrasts (mean change ± 95% CI).

To evaluate mass-independent RMR patterns, a residual-based approach was applied [7,40]. A regression model was fitted at baseline (T0) predicting RMR from FFM and FM, with laboratory site included to account for systematic between-site differences (sex did not improve model fit). The final equation explained baseline RMR well ($R^2 = 0.76$; adjusted $R^2 = 0.71$, $p < 0.001$): pRMR (kcal·day^{-1}) $= 350.18 + 22.39 \cdot \text{FFM} + 15.49 \cdot \text{FM} - 290.13 \cdot \text{Site}$ (Canberra) (Supplementary Figure S1). Adjusted RMR was summarized using a linear mixed-effects model with time as a categorical fixed effect and participant-specific random intercepts. As a sensitivity analysis, residuals were also derived using the Ten Haaf equation to assess observed patterns' consistency (Figure 5) [35].

Associations between post-competition increases in energy intake and recovery of adjusted RMR were examined with Bayesian linear mixed-effects models [41]. The dependent variable was change in adjusted RMR from Tpre (ΔRMR$_{\text{Adj}}$; kcal·day^{-1}). Energy intake change (Δkcal) was decomposed into within-athlete and between-athlete components and scaled per 100 kcal·day^{-1}. Models included weeks since competition (centered at 6 weeks), adjusted RMR at Tpre (scaled per 100 kcal·day^{-1}), sex, and laboratory site as fixed effects, with participant-specific random intercepts. Sensitivity analyses were conducted to assess robustness to alternative model specifications. These included: (i) expressing energy intake relative to FFM (kcal·kgFFM^{-1}·day^{-1}), (ii) adjusting for protein intake, (iii) inclusion of sex-by-intake and time-by-intake interaction terms, (iv) modelling time as a categorical factor (T2–T4), and (v) exclusion of T2 observations to account for interpolated body-composition values. Weakly informative priors were specified to stabilize estimation in this modest sample and reflect typical indirect calorimetry variability (Normal (0,50) for slopes; Normal (0,200) for intercept; Exponential (0.005) for random-intercept and residual SDs) [27,42]. Convergence was assessed using Rhat, effective sample sizes, leave-one-out information criterion, and posterior predictive checks. Results are reported as posterior means with 90% credible intervals (CrI) to balance precision and interpretability in small samples [43].

3. Results

3.1. Participant characteristics

Nineteen athletes (8 male, 11 female) completed at least one post-competition assessment and were included in the analysis (Table 1). At baseline (T0), participants were −20.9 ± 3.4 weeks from their final competition. Thirteen athletes (68%) competed more than once across a 4–10-week season and for these athletes, T1 represented the assessment 1–2 weeks before the first show, and T1.5 as 1–2 weeks before the last show.

3.2. Energy intake and training loads

Figure 3 summarizes FFM standardized energy intake and training load. Energy intake declined from T0 to Tpre (−4.4 $\text{kcal}\cdot\text{kgFFM}^{-1}$ [−7.7, −1.0]), increased from Tpre to T4 (14.3 [10.3, 18.2]), and remained above T0 at T4 (+10.3 [6.3, 14.3]). Training load increased from T0 to Tpre (+740.5 AU [−681.2, 2162.2]), decreased from Tpre to T4 (−3433.2 [−7160.5, 294.0]), and remained below T0 at T4 (−2709.2 [−6129.2, 710.8]) (Table 2).

3.3. Body composition

Body weight decreased from T0 to Tpre (−7.1 kg, 95% CI [−8.3, −5.9], $p < 0.001$), followed by an initial increase from Tpre to T2 (+2.7 [2.0, 3.4], $p < 0.001$), and continued increases from Tpre to T3 (+6.6 [5.3, 7.8], $p < 0.001$) and Tpre to T4 (+8.3 [7.1, 9.4], $p < 0.001$) (Figure 4). At T4, body weight was not clearly different from T0 (+1.1 [−0.1, 2.4], $p = 0.075$). FM decreased from T0 to Tpre (−5.8 [−6.8, −4.8], $p < 0.001$) and increased from Tpre to T3 (+3.4 [2.3, 4.4], $p < 0.001$) and Tpre to T4 (+5.1 [4.0, 6.1], $p < 0.001$). At T4, FM was not clearly different from T0 (−0.8 [−1.8, 0.3], $p = 0.141$). FFM declined from T0 to Tpre (−1.7 [−2.6, −0.9], $p < 0.001$) and increased from Tpre to T3 (+2.7 [1.8, 3.6], $p < 0.001$) and Tpre to T4 (+3.3 [2.4, 4.2], $p < 0.001$). At T4, FFM was higher than at T0 (+1.6 [0.7, 2.5], $p < 0.001$). There was no consistent evidence of sex-by-time interaction for body composition outcomes, with interaction estimates small and imprecise, sex-stratified descriptive data are presented in Supplementary Tables S2 and S3.

3.4. Resting metabolic rate

$\text{RMR}\cdot\text{FFM}^{-1}$ showed a small, uncertain decrease from T0 to Tpre (−0.9 $\text{kcal}\cdot\text{kgFFM}^{-1}\cdot\text{day}^{-1}$, 95% CI [−2.7, 0.9], $p = 0.320$) and increased from Tpre to T2 (+1.5 [0.5, 2.6], $p = 0.005$), Tpre to T3 (+3.0 [1.1, 4.9], $p = 0.002$), and

Table 1. Preparation baseline (T0) participant characteristics.

	Group (n = 19)	Female (n = 11)	Male (n = 8)
≥4 weeks competing n (%)	13 (68%)	7 (64%)	6 (75%)
Weeks to final competition	−20.9 ± 3.4	−20.8 ± 2.6	−21.0 ± 4.3
Age (years)	28.3 ± 6.2	28.5 ± 5.0	27.9 ± 7.9
Height (cm)	168.7 ± 8.9	162.1 ± 2.2	177.8 ± 5.7
Body weight (kg)	70.3 ± 15.1	59.0 ± 6.4	85.9 ± 6.9
FFM (kg)	56.2 ± 13.4	45.9 ± 4.4	70.5 ± 5.9
FM (kg)	14.4 ± 3.2	13.3 ± 3.0	15.8 ± 3.0
BF (%)	21.5 ± 3.9	23.3 ± 3.4	19.0 ± 3.1
RMR (kcals)	1648.5 ± 426.7	1404.1 ± 347.2	1984.5 ± 270.1
$\text{RMR}\cdot\text{FFM}^{-1}$	29.5 ± 4.5	30.4 ± 4.8	28.3 ± 4.1

Values are presented as mean ± standard deviation (SD), unless otherwise stated. FFM = fat free mass; FM = fat mass; BF = body fat percentage; RMR = resting metabolic rate ($\text{kcal}\cdot\text{day}^{-1}$); $\text{RMR}\cdot\text{FFM}^{-1}$ = RMR normalized to FFM ($\text{kcal}\cdot\text{day}^{-1}\cdot\text{kgFFM}^{-1}$). Lab origin: Sydney ($n = 7$), Canberra ($n = 12$). Individual participant characteristics are provided in Supplementary Table S1.

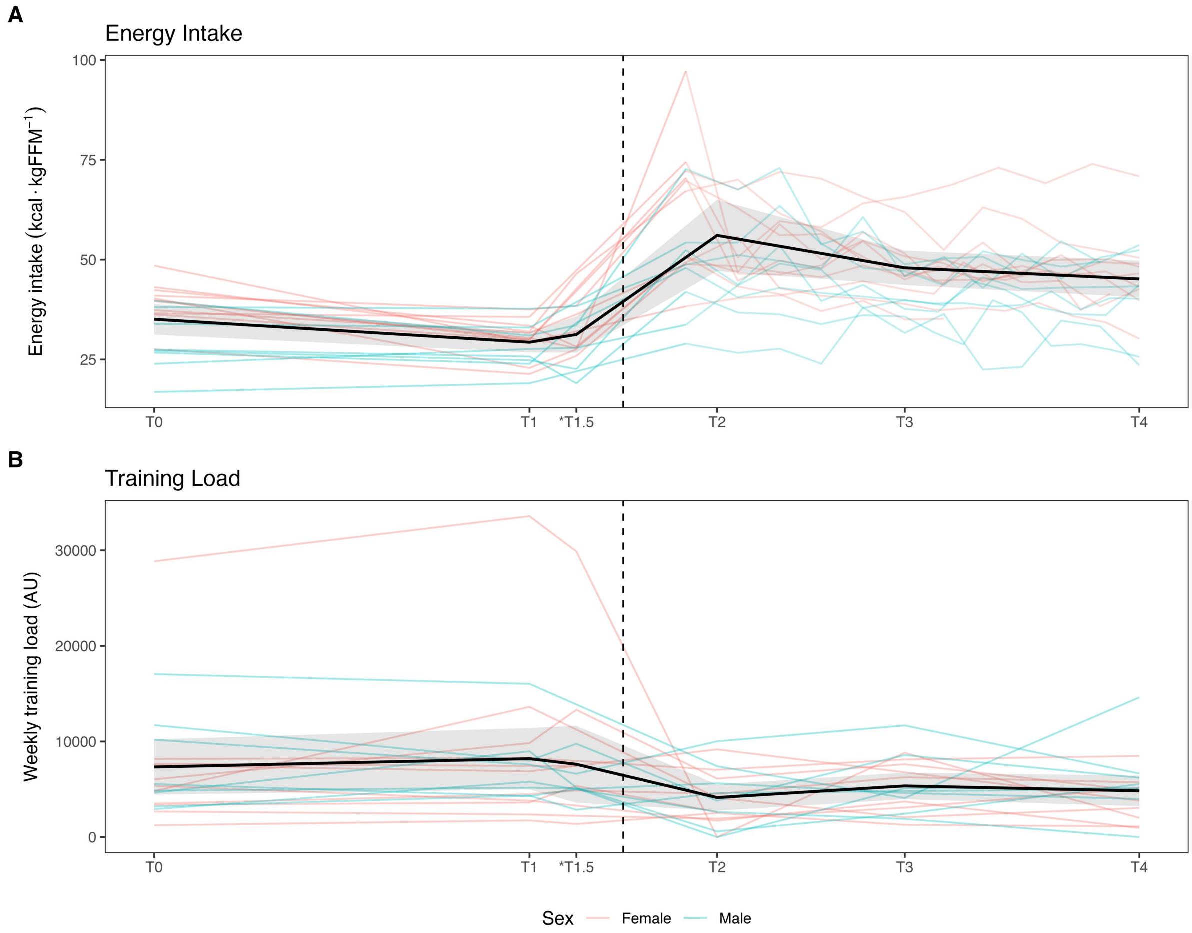

Figure 3. Energy intake and training load across competition preparation and 12-weeks post-competition. (A) Pre-competition energy intake (T0–T1.5) reflects average daily intake normalized to fat-free mass at each time point (kcal·kgFFM^{-1}·day^{-1}). Post-competition intake (T2–T4) is presented as individual weekly averages with the thick black line indicating group mean at each time point. (B) Total weekly training load (AU) is presented as weekly totals at each time point with the thick black line indicating the group mean at each time point. Colored lines represent individual athletes. Shaded areas indicate 95% confidence intervals. The vertical dashed line represents the transition from contest preparation to the post-competition period. *T1.5 denotes an additional pre-final competition assessment for athletes competing ≥4 weeks ($n = 13$).

Tpre to T4 (+2.4 [0.7, 4.1], $p = 0.007$). The overall change from T0 to T4 was uncertain (+1.5 [−0.4, 3.4], $p = 0.125$). Absolute RMR showed a similar pattern, with a reduction from T0 to Tpre (−94.7 kcal·day^{-1}, 95% CI [−211.4, 22.0], $p = 0.110$), followed by increases from Tpre to T2 (+109.5 [42.4, 176.6], $p = 0.002$), Tpre to T3 (+231.9 [113.6, 350.3], $p < 0.001$), and Tpre to T4 (+237.2 [125.5, 349.0], $p < 0.001$). Absolute RMR was higher at T4 relative to T0 (+142.6 [19.2, 265.9], $p = 0.024$). There was no consistent evidence of sex-by-time interactions across RMR models, with interaction estimates small and imprecise.

RMR·FFM^{-1} changes were classified relative to the laboratory-derived LSC. From T0 to Tpre, 9 athletes (47%) declined below, 2 (11%) increased above, and 8 (42%) remained within the LSC. From Tpre to T4, 8 athletes (44%) increased above, 1 (6%) decreased below, and 9 (50%) remained within the LSC. From T0 to T4, 11 athletes (61%) were within the LSC, 5 (28%) were above, and 2 (11%) were below.

Adjusted RMR showed minimal change from T0 to Tpre (−6.7 kcal·day^{-1}, 95% CI [−117.9, 104.6], $p = 0.905$), and increased from Tpre to T2 (+186.4 [68.9, 303.9], $p = 0.002$), Tpre to T3 (+119.7 [6.4, 233.0], $p = 0.039$), and Tpre to T4 (+120.3 [7.1, 233.6], $p = 0.038$). Adjusted RMR was higher at T4 relative to T0 (+113.7 [0.4, 226.9], $p = 0.049$). Ten Haaf-derived residuals showed a similar overall pattern (Figure 5).

In an exploratory Bayesian mixed-effects model, greater increases in daily energy intake (relative to Tpre) were associated with larger increases in adjusted RMR (ΔRMR_{Adj}; Supplementary Table S6). Within athletes,

Table 2. Descriptive outcomes across contest preparation and 12-weeks post-competition.

	T0 (n = 19)	T1 (n = 19)	T1.5 (n = 13)	T2 (n = 19)	T3 (n = 19)	T4 (n = 18)
Body weight (kg)	70.3 ± 15.1	63.7 ± 14.1	64.6 ± 14.0	65.1 ± 14.2 (n = 17)	68.8 ± 15.0	71.6 ± 16.0
FFM (kg)	56.2 ± 13.4	55.0 ± 13.2	56.2 ± 13.7	55.4 ± 13.4* (n = 16)	57.7 ± 14.1 (n = 18)	58.4 ± 14.4
FM (kg)	14.4 ± 3.2	9.2 ± 2.8	8.6 ± 2.4	10.5 ± 2.7* (n = 16)	12.0 ± 2.4 (n = 18)	13.6 ± 2.7
BF (%)	21.5 ± 3.9	15.2 ± 4.1	14.3 ± 4.4	16.9 ± 4.2* (n = 16)	18.3 ± 3.3 (n = 18)	20.0 ± 3.0
RMR (kcal)	1648.5 ± 426.7	1519.6 ± 422.0	1515.6 ± 428.0	1748.6 ± 518.7 (n = 17)	1733.4 ± 475.5	1799.8 ± 539.9
RMR·FFM^{-1}	29.5 ± 4.5	27.8 ± 5.3	27.2 ± 5.7	32.1 ± 5.0 (n = 16)	30.6 ± 4.4 (n = 18)	30.9 ± 5.2
Energy intake (kcal)	1901.1 ± 373.8	1584.0 ± 390.4	1722.7 ± 484.4	2954.7 ± 769.3	2655.7 ± 600.6	2564.7 ± 541.9
Energy intake (kcal·kgFFM^{-1})	35.1 ± 8.0	29.3 ± 5.3	31.3 ± 7.5	56.0 ± 16.6 (n = 16)	47.9 ± 8.7 (n = 18)	45.2 ± 9.2
Training load (AU)	7336.0 ± 6410.1	8204.1 ± 7132.7	7617.9 ± 7397.1	4139.3 ± 2974.5 (n = 18)	5354.8 ± 2915.5 (n = 17)	4840.5 ± 3361.5 (n = 17)
Strength (N·kgFFM^{-1})	48.0 ± 5.9	48.4 ± 7.4	49.5 ± 6.9	48.3 ± 5.5 (n = 17)	46.9 ± 6.2 (n = 17)	47.0 ± 6.1 (n = 17)
TSH (mIU/L)	1.7 ± 0.8	1.5 ± 0.9	1.3 ± 0.7	–	–	1.6 ± 0.6 (n = 16)
FT3 (pmol/L)	5.1 ± 0.6	3.7 ± 0.9	3.8 ± 0.9	–	–	5.5 ± 0.7 (n = 16)
FT4 (pmol/L)	15.2 ± 2.2	13.8 ± 2.3	14.2 ± 2.1	–	–	15.8 ± 2.6 (n = 16)
A-POMS	90.8 ± 19.3	105.7 ± 21.5	103.4 ± 25.2	89.5 ± 16.1	88.5 ± 19.3	87.2 ± 17.0
ASSQ	6.1 ± 2.3	7.2 ± 3.1	8.8 ± 2.4	7.7 ± 2.7	7.2 ± 3.3	6.2 ± 2.5
EDE-QS	7.9 ± 5.8	–	–	7.9 ± 7.6	5.6 ± 5.2	3.6 ± 4.5

Values are presented as mean ± standard deviation (SD). T0 = baseline (~15–25 weeks pre-competition), T1 = 1–2 weeks pre-competition, T1.5 = 1–2 weeks before the final competition for athletes competing across ≥4 weeks, T2 = 2 weeks post-competition, T3 = 6 weeks post-competition, and T4 = 12 weeks post-competition. FFM = fat free mass; FM = fat mass; BF = body fat percentage; RMR = resting metabolic rate (kcal·day^{-1}); RMR·FFM^{-1} = RMR normalized to FFM (kcal·day^{-1}·kgFFM^{-1}). Strength = isometric mid-thigh pull peak vertical force expressed relative to FFM (N·kgFFM^{-1}); TSH = thyroid-stimulating hormone; FT3 = free triiodothyronine; FT4 = free thyroxine. A-POMS = abbreviated profile of mood states (higher scores indicate greater total mood disturbance); ASSQ = athlete sleep screening questionnaire (higher scores indicate greater sleep disturbance); EDE-QS = eating disorder examination questionnaire-short (higher scores indicate greater eating-disorder symptom severity). Lab origin: Sydney (n = 7), Canberra (n = 12). *T2 DXA values were interpolated. Sex-stratified descriptive outcomes are provided in Supplementary Tables S2 and S3.

each +100 kcal·day^{-1} increase beyond their typical Δkcal across recovery was associated with a +16.1 kcal·day^{-1} greater ΔRMR$_{\text{Adj}}$ (90% CrI: 4.8, 27.2). Between athletes, those who increased daily energy intake more from Tpre across the recovery period demonstrated greater ΔRMR$_{\text{Adj}}$ (+18.0 kcal·day^{-1} per +100 kcal·day^{-1}; 90% CrI: 1.6, 33.8). Adjusted RMR at Tpre was negatively associated with subsequent change (−81.8 kcal·day^{-1} per 100 kcal·day^{-1}; 90% CrI: −112.8, −48.3), suggesting greater recovery among athletes with lower adjusted RMR at the start of refeeding. Typical post-competition increases of ~500–1000 kcal·day^{-1} correspond to an estimated ~80–160 kcal·day^{-1} greater ΔRMR$_{\text{Adj}}$.

Sensitivity analyses showed consistent associations between energy intake and ΔRMRAdj, although magnitude and certainty varied. When intake was expressed relative to FFM (kcal·kgFFM^{-1}·day^{-1}), the within-athlete effect remained positive (6.7 [1.1, 12.4]), while the between-athlete effect was attenuated (4.6 [−5.9, 14.4]), suggesting between-athlete differences may be partially explained by body size. Adjusting for protein intake did not materially change the association, and protein was not clearly associated with ΔRMRAdj (all 95% CrIs crossing zero). Excluding T2 observations to account for interpolated body composition did not change the between-athlete estimate (18.3 [−0.9, 37.3]) but reduced the within-athlete estimate (11.0 [−19.8, 42.3]). Models including sex-by-intake and time-by-intake interaction terms did not provide clear evidence of effect modification, with all interaction estimates showing uncertainty and 95% credible intervals crossing zero.

3.5. Thyroid and menstrual function

TSH decreased from T0 to Tpre (−0.3 mIU/L, 95% CI [−0.5, −0.1]) and showed minimal change from Tpre to T4 (+0.2 [−0.1, 0.5]), with minimal change from T0 to T4 (−0.1 [−0.5, 0.3]). FT3 decreased from T0 to Tpre (−1.4 [−1.8, −0.9]) and increased from Tpre to T4 (+1.8 [1.3, 2.3]), with no clear difference from T0 at T4 (+0.3 [0.0, 0.6]). FT4 decreased from T0 to Tpre (−1.4 [−2.6, −0.2]) and increasing from Tpre to T4 (+2.4 [0.9, 3.9]), with no clear difference from T0 at T4 (+0.8 [−0.2, 1.7]). At T1, 8/19 participants had FT3 below the laboratory reference range, and among those assessed at T1.5, 4/12 were below. One participant had FT4 below the reference range at T1. All values were within reference ranges by T4.

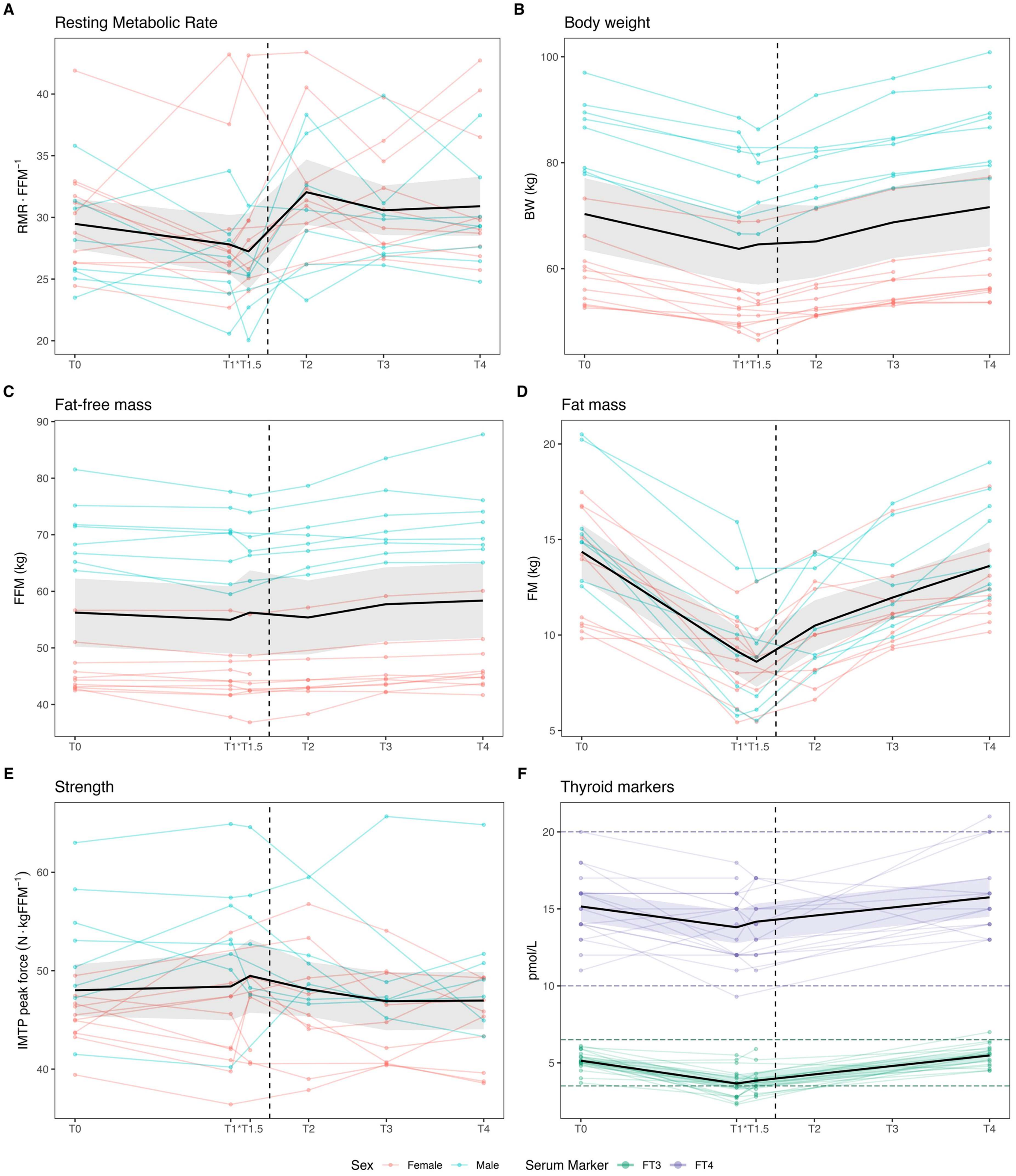

Figure 4. Group mean and individual trajectories for key outcomes across contest preparation and 12-weeks post-competition. (A) RMR normalized to FFM (kcal·kgFFM^{-1}·day^{-1}), (B) Body weight (kg), (C) Fat-free mass (kg), (D) Fat mass (kg), (E) Isometric mid-thigh pull (IMTP) normalized to FFM (N·kgFFM^{-1}), (F) Serum FT3 and FT4 thyroid markers (pmol·L^{-1}) are shown with laboratory reference ranges represented by horizontal dashed lines. Thick black lines represent observed group means with shaded areas indicating 95% confidence intervals. The vertical dashed line represents the transition from contest preparation to the post-competition period. *T1.5 denotes an additional pre-final competition assessment for athletes competing ≥4 weeks ($n = 13$). DXA-derived body composition at T2 (panels C and D) show interpolated estimates.

All female participants reported regular cycles at T0 (24–34 days); four used hormonal contraception ($n = 3$ levonorgestrel IUD; $n = 1$ progestin-only mini-pill). Among naturally cycling athletes ($n = 7$), menstrual disruption was common from T0 to T2 (amenorrhea in 4/7 at T1 and 6/7 at T2). By T4, four resumed menses and two remained amenorrhoeic. One IUD user removed her device at T3 and became pregnant, withdrawing before T4.

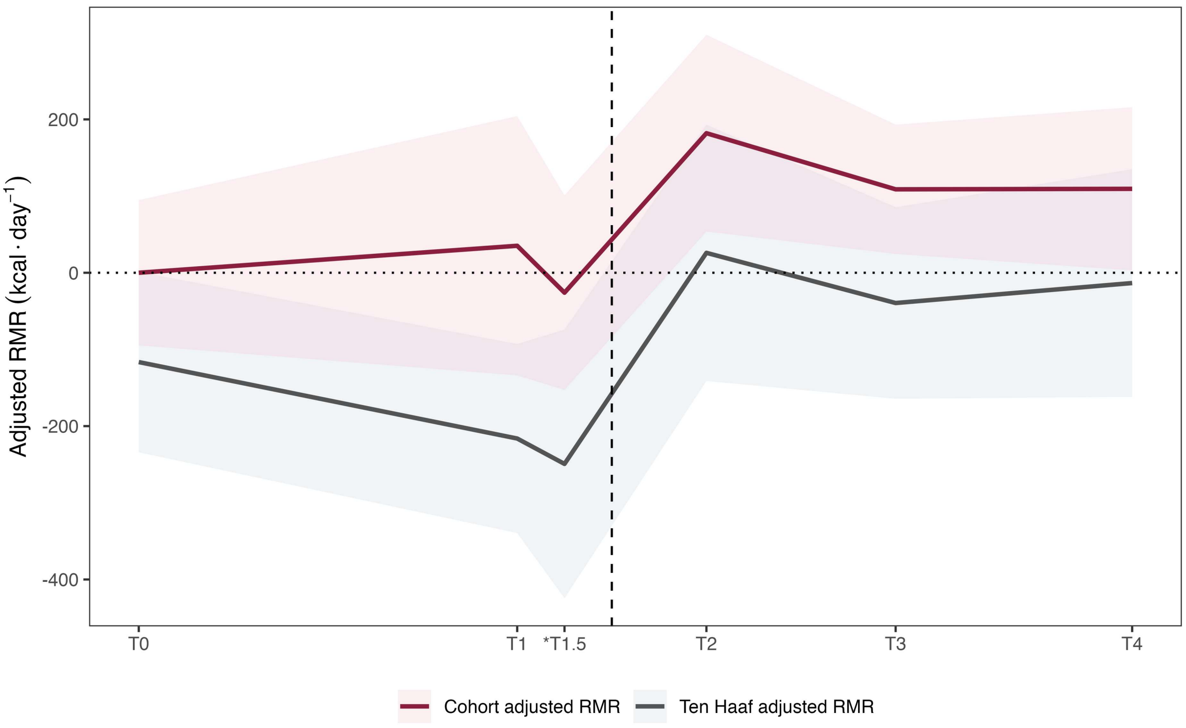

Figure 5. Group mean (±95% confidence interval) adjusted RMR across contest preparation and 12-weeks post-competition for cohort-derived and Ten Haaf-derived residuals. The vertical dashed line represents the transition from contest preparation to the post-competition period. *T1.5 denotes an additional pre-final competition assessment for athletes competing >4 weeks ($n = 13$).

3.6. Strength

IMTP peak force relative to FFM was maintained from T0 to Tpre (+0.6 $N\cdot kgFFM^{-1}$ [−1.6, 2.7]), with small reductions from Tpre to T4 (−2.0 [−5.4, 1.5]), coinciding with reduced weekly training load (Table 2). Relative to T0, strength remained slightly lower at T4, (−1.1 [−4.2, 2.1]).

3.7. Mood, sleep, and eating-disorder symptoms

TMD increased from T0 to Tpre (+13.4 [−1.3, 28.1]) and improved by T4 (Tpre → T4: −16.6 [−30.0, −3.1]), returning near T0 by T4 (T0→T4: −0.3 [−7.8, 7.2]). Sleep disturbance increased from T0 to Tpre (+1.6 [0.3, 2.9]) and improved by T4 (Tpre → T4: −1.4 [−2.5, −0.3]), returning near T0 by T4 (T0→T4: +0.1 [−1.5, 1.6]). Eating-disorder symptom severity decreased from T2 to T3 (−2.3 [−5.0, 0.4]) and from T3 to T4 (−2.1 [−4.0, −0.1]), with an overall change from T0 to T4 of −4.3 [−6.3, −2.4]).

3.8. Athlete interviews

Reflexive thematic analysis identified three themes that characterized athletes' post-competition recovery experiences (Table 3).

Theme 1: The depth of prep shapes the recovery starting point. Athletes described recovery experiences as influenced by preparation severity; more aggressive or prolonged preps were linked to harsher symptoms (e.g. fatigue, sleep disruption, hunger/food focus, libido/menstrual disruption), whereas better-managed preps were perceived to facilitate a smoother transition into recovery. Show day was commonly framed as a deliberate transition into a planned recovery phase rather than an endpoint. Athletes described three broad post-show nutrition approaches: (1) short-term (1–7 days) ad libitum eating followed by a return to structure, (2) “recovery dieting” with an initial jump in dietary intake followed by gradual adjustments

Table 3. Themes, example codes, and example quotes from athlete interviews.

Theme	Example codes	Example quotes
The depth of the prep shapes the recovery starting point	1. Prep severity determines symptom load 2. Show day as transition into recovery 3. Structured versus unstructured refeeding	"There's a certain amount of stress that goes into prep, not just physically, but also emotionally and mentally. The deeper the hole that you dig, the harder your recovery is." (M12) "It comes down to how you view your reverse and accepting that prep doesn't just end on show day, you've still got another 8 to 12 weeks post show that you've got to manage as well." (F1) "When I came off stage, he (coach) gave me 2 days straight completely free (ad libitum). Then we were going back to 1700 calories with 2 untracked meals per week." (F11) "We went straight up to 1750 kcal post-show (previously 1200), but you can't just put it back to like your peak offseason calories because you are going to regain weight very rapidly... You can get into disorder territory as well so it's important to be flexible." (F1)
Refeeding is negotiated between restoring function and managing body composition changes	1. Food noise and overeating 2. Fear of weight gain 3. Body acceptance 4. Performance-focused reframing 5. Normalizing recovery symptoms	"I'm struggling the most with food noise. Regardless of if I'm hungry or not, some days I can't stop thinking about food." (F6) "I didn't want to put on too much weight too quickly… I just felt like any weight gain was too much weight gain even though I knew I had too… I just try to focus on the reasons that the weight is important. Sometimes I feel chunky at the gym, but then I remind myself that it's ok and this is what I'm meant to look like." (F7) "I think the biggest thing for athletes in recovery is it's important to have a tangible, close goal… Not how you look or how you weigh or anything like that… I had to focus on how strong I felt and learning new movements." (M12) "Normalize feelings that you're going to go through… The days when you're eating a lot of food, know that this is what your body wants… just don't beat yourself up after one massive day of eating." (F13)
Support systems for recovery	1. Post-show support influences recovery capacity 2. Coaches as a risk or a protection 3. Peer and partner support 4. Data and education reassurance	"It's something about what coach did to me that just kind of ruined my mentality and my body as well… it's going to take the right kind of coach to get me back on stage." (F9) "A lot of people can go astray and binge, and as soon as they don't have that guidance and accountability from a coach, especially coming out of a severe dieting period." (M15) "Having a person who's in the trenches with you makes all the difference… who knows how low you can feel or how hard it can be." (F12) "It can be disheartening to see the scales go up every day... the DXA's really put it into perspective that I'm not putting on fat mass, its actually more muscle mass which helps and I'm very grateful for." (F6)

based on recovery responses, and (3) more flexible structure using energy ranges/templates with weekly planned untracked meals and progression towards more intuitive eating.

Theme 2: Refeeding is negotiated between restoring function and managing body composition change. Athletes described refeeding as balancing energy restoration, mood, training performance, and reproductive function with rapid weight/fat gain apprehension. Increased hunger, persistent food focus, and occasional binge-eating episodes were common early in recovery and were interpreted either as distressing or as an expected/temporary response to prolonged restriction. Fear of weight gain often reinforced monitoring/structure reliance, while others emphasized body acceptance and reframing weight gain as necessary, with performance-focused goals helping shift attention from aesthetics.

Theme 3: Support systems shape recovery. Recovery experiences were influenced by support quality. Coaches were described as providing helpful structure and normalizing expectations or contributing harm via extreme practices and limited recovery support. Peer/partner support was frequently described as valuable, particularly when shared with others who had undergone similar preparation. Study assessments (e.g. DXA feedback) were perceived as reassuring, aiding body composition change interpretation beyond scale weight, reducing anxiety.

4. Discussion

The present study examined physiological and psychological changes across contest preparation and 12-week post-competition recovery in natural physique athletes and explored whether post-competition increases in energy intake were associated with recovery of adjusted RMR independent of body composition change. Qualitative interviews provided context to these findings by highlighting how athletes experienced preparation severity, refeeding decisions, and support systems during recovery.

4.1. Resting metabolic rate

Contest preparation was associated with small, uncertain RMR·FFM^{-1} reductions, followed by rapid post-competition increases and stabilization through 12 weeks. Adjusted RMR also increased post-competition, indicating recovery was not explained solely by body composition changes. Although group-level changes were modest, LSC threshold analysis highlighted substantial inter-individual variability. Nearly half of athletes demonstrated meaningful RMR·FFM^{-1} declines during preparation, whereas others remained within expected measurement variability, indicating reliance on group means may obscure clinically relevant individual responses. This variability likely reflects day-to-day indirect calorimetry technical error and biological heterogeneity expected in a multi-site design [27,44]. Individual differences persisted through study end, with some athlete RMR's meaningfully above and others below baseline at 12 weeks. Similar bidirectional patterns were reported by Longstrom et al. (2020), who observed post-competition RMR changes exceeding measurement variability in both directions [13], reinforcing the value of athlete-specific RMR interpretation to inform recovery planning.

While metabolic rate is strongly explained by changes in body composition [45], some athletes may exhibit mass-independent RMR suppression during energy restriction, reflective of adaptive thermogenesis [7]. Our findings broadly align with prior studies in physique athletes reporting reductions in RMR alongside coordinated endocrine adaptations, including reductions in leptin and thyroid hormones, which collectively act to reduce energy expenditure and conserve energy during weight loss [5,7,13,15]. Isola et al. (2023) reported decreases in adjusted RMR during a ~23-week competition preparation, alongside reductions in heart rate, leptin, FT3, and ghrelin increases, suggesting coordinated responses to lower TDEE and drive appetite [7]. While the decline in absolute RMR for our cohort was modest, concurrent reductions in FT3 and FT4 and the high prevalence of menstrual disruption among naturally cycling athletes suggest that preparation induced a physiologically meaningful energy-conserving state. Ten Haaf equation sensitivity analyses further highlighted that RMR was below predicted at baseline and rebounded post-competition. This suggests that some athletes entered testing partially energy-restricted, such that cohort-based residuals could underestimate absolute magnitudes of suppression relative to fully weight-stable conditions. Indeed, short-term LEA (e.g. ~10 days at ~25 kcal·kgFFM^{-1}·day^{-1}) can reduce RMR ~75 kcal·day^{-1} in trained females [46], indicating adaptation may have preceded T0. Notably, RMR reflects one TDEE component; further adaptation may involve non-resting components (e.g. spontaneous activity/NEAT and diet-induced thermogenesis) that were not directly measured [47].

Post-competition, RMR increased rapidly by 2 weeks and remained above Tpre, with minimal change between 6–12 weeks, plateauing by study end. This pattern mirrors prior physique literature describing early RMR increases within the first 4–6 weeks and heterogeneous trajectories thereafter [13,14,18,48]. The early RMR rise is expected partly due to rapid body mass regain, particularly FM, and modest FFM increases, which both increase resting energy requirements [27,45]. However, it also reflects acute metabolic responses to refeeding, including elevations in diet-induced thermogenesis and the energetic cost of rapid substrate storage, which contribute to non-linear increases in energy expenditure and may be amplified by post-restriction hyperphagia [14,49]. Interpretation of metabolic recovery therefore depends on whether changes reflect short-term responses to acute refeeding or more sustained resolution of mass-independent suppression [8]. In this context, increases in adjusted RMR are consistent with partial restoration beyond that explained by concurrent body composition changes, although early post-competition values may be influenced by temporary metabolic overshoot. Importantly, RMR restoration should not be interpreted as complete TDEE recovery, as non-resting components may recover on different timescales or remain suppressed despite RMR normalization [50].

4.2. Energy intake and RMR recovery

Greater post-competition increases in daily energy intake were associated with larger increases in adjusted RMR during recovery. Our exploratory model indicated typical early post-competition increases (~500–1000 kcal·day^{-1}) corresponded to estimated ~80–160 kcal·day^{-1} recovery in adjusted RMR. These magnitudes are modest but suggest larger refeeding may restore RMR faster than what occurs due to body composition changes alone. Notably, lower adjusted RMR at Tpre predicted greater increases in adjusted RMR, indicating athletes with more negative residual values at preparation end typically showed better recovery during refeeding. Similarly, Trexler et al. [14] reported RMR relative to predicted values (Cunningham equation) increased from ~92% one week pre-competition to ~105% within one week post-competition, before returning toward predicted at 4–6 weeks [14]. They also reported positive associations between RMR changes, protein intake, and body fat percentage, suggesting dietary factors and body composition changes may jointly influence the trajectory of metabolic recovery. However, in our cohort, protein intake did not materially alter the association between energy intake and adjusted RMR, and protein intake was not clearly associated with RMR recovery. This may suggest that total energy intake rather than macronutrient composition may be the primary driver of early metabolic increases in this context. Complementary evidence from controlled overfeeding (+1000 kcal·day^{-1}) in non-athletes also demonstrates metabolic rate can increase during energy surplus in a non-linear fashion, even when measurable FFM change is minimal, supporting the plausibility of intake-related RMR shifts [51]. However, given our observational design, effect directionality should not be assumed; endocrine recovery, mood, or appetite may have influenced energy intake and RMR.

Practically, larger post-competition energy intake increases may modestly accelerate recovery of mass-independent RMR. However, we urge cautious interpretation given the FM regain pattern. Interviews highlighted the challenge of selecting refeeding approaches that support physiological recovery while managing weight regain, body image concerns, eating behaviors, and future competitive goals. Athletes generally aimed to increase diet flexibility while retaining some structure to maintain dietary control and manage weight regain rates. Across approaches, they described tension between needing sufficient energy for health/performance restoration and body composition concerns relating to reduced dietary control. Decisions were influenced by physiological and psychological factors and were commonly framed relative to the preparation phase severity and support availability during recovery.

4.3. Body composition

Across preparation, body weight decreased ~7.1 kg, consisting mostly of FM with minimal FFM reductions. This aligns with recommendations for slower weight loss rates to preserve FFM [17] and systematic reviews reporting better FFM preservation when resistance training is combined with adequate dietary protein [4]. Post-competition, body composition rebounded rapidly, with most FM and FFM restoration occurring within the first 6 weeks. This early increase in FFM is likely driven in part by glycogen repletion and associated intracellular water retention, whereby each gram of glycogen binds ~3–4 g of water, which can inflate DXA-derived lean mass estimates [21]. Acute carbohydrate loading has been shown to increase lean body mass by ~2% within 48 h [22], indicating that early FFM increases largely reflect "wet mass" rather than true hypertrophy. These early body composition changes occurred alongside RMR·FFM^{-1} and adjusted RMR increases, suggesting that rapid restoration of substrate availability and metabolically active tissue contributes to early metabolic increases. While FM accounted for most initial weight regain, increases in FFM, despite reflecting "wet mass" in the short term, are still associated with higher resting energy requirements and may contribute to increases in absolute RMR [45]. Accordingly, a more rapid post-competition increase in body weight and FM may reflect a practical trade-off between restoring physiological function and limiting body composition changes that athletes may find difficult to accept. Athlete accounts suggested they were often aware that some weight regain was necessary for recovery, yet still feared excessive fat accumulation, particularly where this was perceived to threaten athlete identity, body image, or future performance and competitive goals.

Mean FFM exceeded baseline despite reduced training load and minimal strength change, suggesting the increase may not reflect hypertrophy alone. Physiologically, the observed rise in FFM may reflect a combination of factors, including glycogen-associated water repletion, restoration of organ tissue and other metabolically active components reduced during LEA, and, in some cases, accrual of contractile tissue during return to higher energy availability and sustained resistance training exposure [22,50]. Controlled refeeding studies support the plausibility of lean mass restoration during energy surplus even without structured exercise [49,52], particularly with sufficient dietary protein [53]. Athlete accounts were consistent with these findings. Several described the DXA feedback as reassuring and were positively surprised by greater-than-expected FFM restoration and less FM gain than anticipated, which they viewed as favorable body recomposition. This helped contextualize post-competition weight regain and reduce anxiety about increasing body mass. Together, these findings suggest that early post-competition body composition changes likely reflect physiological recovery, while athletes' interpretations of these changes may influence how acceptable and manageable recovery feels. Athlete accounts of heightened food focus and apprehension about rapid weight gain demonstrate that refeeding is both physiologically driven and psychologically demanding. Participants emphasized that recovery should be viewed as a planned extension of preparation, with clear goals and continued structure to navigate the transition.

4.4. Thyroid and menstrual function

Thyroid function declined during contest preparation, primarily FT3, returning within reference ranges by 12 weeks post-competition. This pattern aligns with adaptive responses to LEA, where peripheral thyroid hormones downregulate to conserve energy expenditure without overt clinical hypothyroidism [54]. The directional changes in FT3/FT4 aligned with the RMR trajectory, suggesting largely reversible energy-conserving responses to preparation. Hulmi et al. (2017) reported similar reductions in FT3 in 27 female physique competitors, with 13 remaining below reference ranges 3–4 months post-competition [55]. FM in their cohort remained slightly below baseline (−1.9 kg), suggesting full thyroid restoration may require more complete FM recovery and/or longer exposure to adequate energy availability, as peripheral thyroid hormones appear sensitive to energetic status and adipose-related signaling [56]. Notably, leptin and estradiol returned to baseline despite persistent FT3 suppression in a subset, indicating endocrine recovery may be dissociated across axes. Post-competition increases in FM and leptin have been associated with RMR recovery [13], indicating adipose-derived signaling may contribute to metabolic normalization. Although we did not measure leptin, it plays a central role as a primary adipostat regulating the hypothalamic-pituitary-thyroid axis, whereby reductions in energy availability and fat mass suppress leptin and downregulate T3, while restoration of energy and fat mass restores leptin signaling and supports normalization of thyroid function and RMR [57].

Among naturally cycling athletes, amenorrhea was common during preparation, and although most resumed menses by 12 weeks, two remained amenorrhoeic. Similar delayed reproductive recovery has been documented in physique athletes [7,13,15], indicating endocrine restoration may lag behind weight and RMR recovery in some individuals. This disruption is driven by suppression of hypothalamic luteinizing hormone pulsatility under conditions of LEA, which reduces estradiol and halts menstrual function [54]. Recovery timelines are highly variable; endocrine function may take substantially longer to normalize, with reports of menstrual and hormonal recovery requiring >70 weeks post-competition despite restoration of body weight and energy availability [58]. Thus, contest preparation induces coordinated but reversible endocrine suppression, with specific metabolic and reproductive markers recovering over dissociated timelines. Notably, one athlete who discontinued intrauterine contraception during recovery subsequently conceived, highlighting the potential for rapid reproductive restoration once energetic and hormonal conditions permit.

4.5. Psychometrics and qualitative accounts

Mood disturbance and sleep disruption worsened during contest preparation and improved across recovery, while eating-disorder symptom severity peaked from baseline through the immediate post-competition period (T2) before declining by 12 weeks. Thus, psychological strain was greatest during preparation and early refeeding, even as physiological markers began to recover. Comparable trajectories are reported in other physique studies, with mood and sleep disruption approaching competition and gradual improvement thereafter [13,18,59]. Specifically, Tinsley et al. (2018) reported sustained dietary restraint alongside increased uncontrolled eating during recovery, highlighting this period as psychologically vulnerable despite structured intentions [15]. Our athletes further support this tension, describing heightened food focus, discomfort with rapid body composition change, and tension between maintaining structure and responding to strong appetite signals. At the same time, athletes identified protective coping strategies, such as reframing weight restoration as functional for performance, normalizing transient post-show mood changes, and maintaining flexibly structured dietary approaches. The availability and quality of support systems were described as influential, with coaching guidance and peer reassurance helping athletes reframe weight regain as part of recovery versus personal failure.

4.6. Limitations

This observational study cannot establish causal relationships between energy intake and RMR recovery. Dietary intake and training load were self-reported; despite participants being experienced with weighed food tracking and logs being regularly reviewed, systematic misreporting and database-related variability may persist. Additionally, inconsistent reporting of absolute training load (e.g. kg lifted) required reliance on RPE-based metrics, limiting the ability to distinguish dietary versus mechanical drivers of FFM change. RMR and body composition were assessed across two labs using different equipment. While site was accounted for statistically, between-system differences and inherent variability may contribute to imprecision. RMR may also have been influenced by residual excess post-exercise oxygen consumption, as heavy resistance training can elevate energy expenditure for up to 24–48 h [60], which may not be fully mitigated by the ≥10-h rest period. However, the use of our lab-derived LSC, established under similar ecologically valid conditions in strength-trained populations, aids interpretation of whether observed changes exceeded expected variability. DXA was not performed at T2 due to known acute glycogen and fluid shifts during early refeeding; body composition was therefore interpolated using adjacent measures. While this reduces misclassification of transient changes, it introduces modelling uncertainty. Sensitivity analyses indicate findings were robust, although some uncertainty remains. The Bayesian model estimated an average association between energy intake and RMR recovery across the post-competition period. Although time- and sex-by-intake interactions were explored in sensitivity analyses, these estimates were highly uncertain, with wide credible intervals overlapping zero. Accordingly, potential time- or sex-specific differences in the intake-RMR relationship cannot be excluded, limiting certainty in the magnitude and generalizability of the observed associations. Finally, the sample was modest ($n = 19$) and heterogeneous, and natural status was self-declared. Future studies should incorporate TDEE assessment via doubly labelled water to quantify real-world changes and/or accelerometry to differentiate TDEE component-specific recovery. More frequent early post-competition hormonal sampling (e.g. leptin, insulin, cortisol) would improve interpretation of the time course and mechanisms of RMR change. Longer follow-ups and experimentally comparing refeeding strategies could determine whether the apparent stabilization by ~12 weeks reflects full recovery or ongoing adaptation.

4.7. Practical applications

Coaches and athletes should plan post-competition periods as active recovery phases, recognizing the largest body composition changes typically occur within ~6 weeks. Energy intake should increase post-competition to support metabolic and endocrine restoration. In this cohort, larger post-competition energy increases were associated with greater RMR recovery. However, reductions in training load may have contributed to FM regain, and the relative contribution of increased intake versus reduced expenditure remains uncertain. Given the rapid

FM regain and heterogeneous recovery trajectories, post-competition strategies should be individualized rather than uniformly conservative or aggressive. RMR should not be used as a sole indicator of recovery. Practitioners should monitor multiple markers, including menstrual function, thyroid, mood, sleep, appetite, eating-related distress, and training readiness. Given measurement variability, interpreting individual RMR changes via LSC/MDC is preferable. Qualitative findings highlight the importance of structured support, realistic expectations, and accountability after competition to help navigate early recovery and align physiological restoration with longer-term performance and body composition goals.

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Author contributions

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Disclosure statement

No potential conflict interest was reported by the author(s). In the interest of transparency, while no direct financial or indirect non-financial conflict of interest exists in relation to this manuscript, ERH declares he is a business owner and career writer and content creator in the fitness and health industry. Further, he has provided educational content creation and consultancy services to Optimum Nutrition, a sports nutrition supplement company subsidiary of Glanbia plc, in exchange for personal payment and research funding, personal nutrition supplements, apparel, and travel expenses reimbursement.

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Data availability statement

The analysis code used in this study has been archived on the Open Science Framework under doi:10.17605/OSF.IO/HB5Y9. Raw anonymized data are available upon request from the corresponding author.

Ethics approval & consent to participate

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Human Research Ethics Committee at the University of Canberra (HREC-13593) and the Australian Catholic University (2024-3645RC). Informed consent was obtained from all individual participants included in the study.

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