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Systematic Review

Mapping the Endogenous GLP-1–Appetite Response to Acute Exercise in Humans: Temporal Dynamics, Modifiers and Dose–Response in a Multilevel Meta-Analysis

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Abstract

Background: Endogenous glucagon-like peptide-1 (GLP-1) may participate in exercise-related metabolic and appetite regulation, but its acute recovery profile and behavioural relevance are poorly quantified. We synthesised hormonal and appetite-related responses to single exercise bouts, including temporal patterns and potential modifiers. **Methods:** Six databases were searched through 8 September 2026. Reports were linked by independent study family; matched exercise–rest contrasts were synthesised using three-level random-effects models with family-clustered CR2 inference. **Results:** Fifty-nine reports representing 52 study families were included; 23 families contributed quantitatively. Direct post-exercise GLP-1 was higher after exercise (13 families, 52 effects; Hedges' g = 0.54, 95% CI 0.25–0.82). Pooled point estimates were positive at 0–15 min (g = 0.45, 0.19–0.70), >15–60 min (g = 0.58, 0.02–1.14) and >60–120 min (g = 0.71, −0.33–1.75). No temporal decline was demonstrated; later windows included fewer families and wider intervals. Exploratory comparisons suggested numerically greater direct responses after vigorous/high-intensity or shorter (<45 min) bouts, without conclusive between-subgroup differences. Post-prandial/challenge and delayed GLP-1 responses were less definitive. Subjective appetite ratings favoured suppression (g = 0.45, 0.06–0.84), but measured ad libitum energy intake was inconclusive (g = 0.16, −0.19–0.50). The appetite estimate was sensitive to dependence assumptions, and evidence certainty was low or very low. **Conclusions:** Acute exercise is associated with a moderate increase in circulating endogenous GLP-1, providing a quantitative foundation for investigating the exercise–gut–brain axis. Hormonal changes, perceived appetite and actual food intake are distinct endpoints; the duration of GLP-1 elevation and any downstream behavioural effects require targeted, time-resolved trials.

Keywords: acute exercise; glucagon-like peptide-1; appetite regulation; gut hormone; energy intake; meta-analysis

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1. Introduction

Obesity and physical inactivity are now converging global health problems. In 2022, almost 880 million adults were living with obesity, while 31.3% of adults worldwide—about 1.8 billion people—did not meet the recommended levels of physical activity [1,2]. The trajectory is moving in the wrong direction. The World Health Organization (WHO) has

set a target of a 15% relative reduction in insufficient physical activity by 2030, yet the current trends indicate that the world is off track [2,3]. Physical inactivity is also costly. If the current levels persist, WHO estimates suggest almost 500 million preventable cases of non-communicable disease and more than USD 300 billion in direct health-care costs between 2020 and 2030 [4]. These figures place scalable, low-cost strategies that improve metabolic health and appetite regulation high on the public-health agenda [3–5].

At the same time, GLP-1-based therapies have produced clinically important effects on body weight and cardiometabolic risk [6,7]. WHO guidance places these medicines within integrated care that includes behavioural support and physical exercise [6]. This creates a physiological question: does exercise also alter endogenous GLP-1, and do such changes accompany changes in appetite or eating behaviour? These questions concern endogenous hormone concentrations and cannot establish equivalence to sustained pharmacological GLP-1 receptor agonism.

Endogenous glucagon-like peptide-1 (GLP-1) is released mainly from enteroendocrine L cells in response to nutrient exposure [7,8]. It contributes to glucose-dependent insulin secretion, suppresses glucagon, slows gastric emptying and participates in the control of appetite and food intake [7–10]. Experimental GLP-1 administration can increase fullness and reduce ad libitum energy intake in humans [9,10]. Acute exercise can also transiently suppress appetite. This phenomenon has been linked to lower acylated ghrelin and, in some studies, higher GLP-1 and peptide YY (PYY) [11,12]. The response is not uniform. Exercise intensity, duration and mode may matter. So may feeding state, adiposity, sex, training status and the timing of blood sampling [11–14]. These sources of variation are not nuisance factors alone. They are potential determinants of when exercise most strongly engages the endogenous appetite-regulatory system.

The evidence base has outgrown the methods used in earlier syntheses. A 2014 meta-analysis included only five studies and eight trials for GLP-1 and restricted its GLP-1 synthesis to area-under-the-curve data [12]. A later meta-analysis focused on six studies in people with overweight or obesity [13]. A 2023 review addressed high-intensity interval or sprint exercise versus moderate-intensity continuous exercise in healthy adults [14]. These studies were important, but they do not resolve several questions that now matter. Direct post-exercise GLP-1 responses are biologically different from responses measured after a meal, an oral glucose tolerance test, or a mixed nutrient challenge. A next-day response is different again. Total GLP-1 and active or intact GLP-1 are also not interchangeable. Most acute studies collect repeated time points and often report several related outcomes from the same participants. Treating these estimates as independent can overstate precision [15,16].

We conducted a systematic review and multilevel meta-analysis to distinguish direct post-exercise, post-prandial/challenge, and delayed GLP-1 responses; to examine total and active or intact GLP-1 separately; and to synthesise PYY, ghrelin, subjective appetite, and energy intake as related but distinct outcomes. Three-level random-effects models and small-sample cluster-robust inference retained dependent effects without treating them as independent studies [15–18]. Exploratory subgroup analyses and linear and quadratic meta-regressions examined temporal and dose-related associations. The aim was to characterise the evidence and its uncertainty, not to establish GLP-1-mediated appetite suppression or an optimal exercise prescription.

2. Methods

2.1. Protocol, Registration and Reporting

The review was reported according to PRISMA 2020 and informed by the Cochrane Handbook [19,20]. It was registered in PROSPERO on 14 September 2026 (CRD420261506079). The registered plan specified the review question, eligibility framework, outcome hierarchy

and principal multilevel analysis. Supplementary Table S20 summarises its implementation, including the additional SPORTDiscus search, operational moderator definitions and qualitative synthesis of head-to-head exercise comparisons.

2.2. Information Sources and Search Strategy

We searched PubMed, Web of Science Core Collection, Embase (Embase.com), the Cochrane Library, SPORTDiscus (EBSCOhost) and the China National Knowledge Infrastructure (CNKI) from database inception. Final searches were run on 7 September 2026 for PubMed, Web of Science Core Collection, the Cochrane Library, and SPORTDiscus and on 8 September 2026 for Embase and CNKI. Search strategies combined controlled vocabulary, when available, with free-text terms for GLP-1, incretin biology, and exercise or physical activity. GLP-1 terms included "glucagon-like peptide-1", "GLP-1", "GLP1" and "incretin". Exercise terms included "exercise", "physical activity", "aerobic exercise", "resistance exercise", "interval exercise", "high-intensity interval training", "sprint interval training", "walking", "running" and "cycling". Database-specific syntax and Chinese-language equivalents were used where appropriate. We did not apply a study-design filter to the core search because acute crossover and non-randomised exercise studies are indexed inconsistently. The complete database-specific strategies and search dates are provided in Supplementary Table S1. The search and selection procedures followed PRISMA 2020 and Cochrane recommendations [19,20].

2.3. Eligibility Criteria

We included human studies that examined acute exercise exposure and measured endogenous circulating GLP-1. There were no restrictions on participant age, sex, bodyweight status, training status or clinical diagnosis. Eligible exercise exposures included a single structured exercise bout and a limited number of acute bouts performed within the same day. Aerobic, resistance, interval, sprint, walking, running, cycling, mixed-mode and other structured exercises were eligible. Studies performed in special metabolic states, including type 1 or type 2 diabetes, obesity and bariatric-surgery populations, were retained when the acute exercise contrast was otherwise interpretable.

The primary causal comparison was acute exercise versus a matched rest or no-exercise condition. For this comparison, exercise and control conditions had to be comparable apart from the exercise exposure. Meal timing, nutritional supplements, pharmacological treatment, environmental exposure and other co-interventions had to be matched or sufficiently balanced. Factorial studies were eligible when a contrast could be formed between exercise and non-exercise conditions under the same background co-intervention. Studies that used exogenous GLP-1 infusion or a GLP-1 receptor agonist as the experimental stimulus were not eligible for the endogenous GLP-1 synthesis.

The broader review also retained acute exercise studies without a matched non-exercise control when they contributed relevant mechanistic or comparative information. These included head-to-head comparisons such as high-intensity interval exercise versus moderate-intensity continuous exercise, modifier-only comparisons such as sex or menstrual phase, and exercise-by-nutrition, exercise-by-drug or exercise-by-environment designs. Such comparisons were not mixed with the primary matched exercise-versus-rest pool unless the background exposure was matched. Post-prandial, oral glucose tolerance test, oral fat tolerance test and mixed-meal responses were eligible but were analysed separately from direct post-exercise responses. Delayed responses, including evening exercise followed by next-day sampling or a next-day metabolic challenge, were also retained as a separate response category. Inclusion in the systematic review did not automatically imply inclusion

in a pooled meta-analysis. A report entered quantitative synthesis only when its data mapped to a prespecified, estimable contrast used in one of the final outcome models.

Studies were excluded if they were conducted on animals, cells or isolated tissues; examined chronic training without a separable acute response; did not measure endogenous GLP-1; or assessed only responses to exogenous GLP-1. Reviews, editorials, protocols and conference records without sufficient original information were excluded. A study could remain in the qualitative review when it contained interpretable acute-exercise findings but no recoverable effect estimate compatible with the pooled comparison. Duplicate reports were not counted as independent studies; non-duplicative information could be retained within the same family.

Quantitative synthesis required a matched exercise-versus-rest contrast, a compatible outcome and physiological context, sufficient numerical inputs, and a non-duplicated participant contribution after family linkage. Head-to-head exercise comparisons and unmatched nutritional, drug or environmental contrasts were not mixed with that estimand. The contribution register in Supplementary Table S18 distinguishes the 23 contributing families from the 29 non-contributing families and gives a specific reason for each. It also distinguishes reports that contributed no selected numerical rows from families that contributed in another report.

2.4. Study Selection and Study-Family Assignment

Two reviewers independently screened the records and assessed the full texts, resolving disagreements by discussion or third-reviewer adjudication. Reports were combined into families using trial registration, recruitment source, setting, participant characteristics, allocation, interventions and timing. Family identifiers were retained throughout the analysis [15,16,19]. For the overlapping Hazell/Hallworth reports, the combined-sex report was the canonical source for the shared moderate-continuous, sprint-interval and control trajectories; these trajectories were not additionally entered from the sex-specific reports [21]. Distinct cohorts and contrasts remained identified within each family.

2.5. Data Extraction

Two reviewers independently extracted data using a standardised structured form. Disagreements were resolved by discussion and, when necessary, by a third reviewer. We extracted first author, publication year, country, study design, randomisation and crossover features, washout period, sample size and the information needed to identify overlapping reports. Participant variables included age, sex, body mass, body mass index (BMI), body composition when available, health or disease status, diabetes and bariatric status, medication use, habitual physical activity or training status, and maximal or peak oxygen uptake (VO_2max or VO_2peak). Study-cohort and participant characteristics are summarised in Supplementary Table S2.

Exercise variables were extracted at the condition level. These included exercise mode, continuous or interval structure, bout duration, number of bouts, work and recovery intervals, intensity metric and value, speed, grade, power, resistance load, estimated or measured energy expenditure, fasting or fed state, time since the last caloric intake, pre-exercise meal timing and composition, post-exercise nutrition, oral glucose or fat tolerance tests, mixed-meal challenges, drug conditions, hypoxia, ambient temperature, blood-flow restriction and other relevant co-interventions. Comparator conditions were coded in parallel so that the matching of non-exercise exposures could be assessed directly. Acute exercise conditions and matched comparators are summarised in Supplementary Table S3.

For GLP-1, we extracted the biological matrix, assay information, molecular form, sampling schedule, reported unit, concentration values, change scores, total area under the

curve (tAUC), incremental area under the curve (iAUC), and the timing of each sample relative to exercise termination and, when applicable, meal or nutrient-challenge onset. Total GLP-1, active GLP-1, intact GLP-1 and GLP-1(7–36) were retained as distinct analyte forms at extraction. We also recorded the use of dipeptidyl peptidase-4 or protease inhibition when reported because pre-analytical handling is relevant to measurements of active GLP-1 [7,8]. Secondary outcomes included PYY, acylated and total ghrelin, subjective appetite ratings and subsequent energy intake. Exploratory metabolic and mechanistic outcomes, including glucose, insulin, glucagon, GIP, lactate, interleukin-6, GDF-15 and Lac-Phe, were extracted when reported.

When means or dispersion were not tabulated, values were recovered from figures where feasible and flagged as digitised. SEs were converted to SDs using $SD = SE \times \sqrt{n}$; reported change-score SEs were treated as the dispersion of the change, not as endpoint dispersion. Compatible endpoint or change inputs were selected once for each contrast and time. Relative percentage changes were not entered as additional independent outcomes when the corresponding raw concentration was already retained. Source selection, alternative representations and sampling contexts are described in Supplementary Table S19. Holliday's GLP-1 analysis denominator of seven was inferred from the reported four male and three female responses and examined in a sensitivity analysis that excluded those GLP-1 observations. Its other outcomes used the reported eight participants.

2.6. Outcome Classification and Temporal Framework

The primary outcome was endogenous circulating GLP-1. We separated three physiological contexts before pooling. Direct acute response referred to GLP-1 measured after exercise or matched rest and before the next caloric intake. The primary direct-recovery analysis focused on the first 120 min after exercise. Exercise-modified post-prandial or challenge response referred to GLP-1 that was measured after both conditions had been followed by the same meal, oral glucose tolerance test, oral fat tolerance test or other nutrient challenge. Delayed response refers to measurements obtained more than 6 h after exercise, including next-day fasting or challenge responses. Head-to-head exercise comparisons were treated separately from matched exercise-versus-rest contrasts. For same-day multiple-bout protocols, samples obtained after an intervening matched meal were classified with the nutrition-modified context rather than the isolated first-bout direct response.

The sampling windows were during exercise, 0–15 min, >15–60 min, >60–120 min, >120–360 min, >6–24 h, and >24 h, as specified in the registered framework. Time was anchored to exercise termination, and caloric intake determined whether an observation belonged to direct recovery or post-prandial/challenge synthesis. Pre-exercise baseline observations were not post-exercise outcomes. Total and active/intact forms were separated where possible. A reported change score was preferred over an endpoint representation of the same estimand when compatible dispersion was available; a relative percentage change and its raw concentration were not counted twice. Eligible repeated times were retained with family clustering [15,16]. Supplementary Table S4 defines each outcome domain.

2.7. Direction of Effects

Direction coding was used to make each outcome interpretable, not to infer a common biological mechanism. Positive g indicates higher GLP-1, PYY, fullness or satiety; for acylated ghrelin, total ghrelin, hunger, prospective food consumption, composite appetite and energy intake, the means were multiplied by −1 so that positive g denotes a lower value. Dispersion was unchanged. Higher circulating GLP-1 is not itself evidence of reduced appetite, lower intake or clinical benefit. Desacylated/unspecified ghrelin and exploratory

metabolic markers were not assigned a generic beneficial direction [7,8,11,12]. The full rules are given in Supplementary Table S5.

2.8. Risk-of-Bias Assessment

Two reviewers independently assessed the risk of bias, resolving differences by discussion or third-reviewer adjudication. The assessment unit was the eligible report, not the independent study family. Randomised reports were assessed using RoB 2, including crossover period/carryover concerns where applicable [19,22]. Non-randomised reports were assessed using ROBINS-I [23]. Report-specific judgements were retained, while the analyses clustered related reports by family. Each of the 59 included reports received one assessment.

2.9. Certainty of Evidence

Two reviewers independently rated the certainty of evidence for the principal GLP-1 and appetite-related outcomes using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework [24,25]. Disagreements were resolved by discussion and, if unresolved, by a third reviewer. We considered risk of bias, inconsistency, indirectness, imprecision and publication bias. Randomised evidence began at high certainty. Contributions from non-randomised studies were considered in the design and risk-of-bias judgement for mixed bodies of evidence. Certainty was downgraded when prespecified GRADE domains indicated important limitations. No upgrading factor was applied unless supported by the evidence. Final ratings were reported as high, moderate, low or very low certainty [24,25]. Outcome-specific GRADE judgements are reported in Supplementary Table S6.

2.10. Effect-Size Calculation

The common metric was Hedges' g, a small-sample-corrected standardised effect [26,27]. All quantitatively synthesised contrasts were crossover comparisons. For completeness, the general extraction framework also specified a parallel-group estimator, although it was not used in the final pooled models:

$$d = (\bar{X}_E - \bar{X}_C)/\mathrm{SD}_{\mathrm{pooled}}$$

$$\mathrm{SD}_{\mathrm{pooled}} = \sqrt{\{[(n_E - 1)\mathrm{SD}_E^2 + (n_C - 1)\mathrm{SD}_C^2]/(n_E + n_C - 2)\}}$$

For a parallel-group contrast, the degrees of freedom would be m = nE + nC − 2. The exact Hedges correction is $J(m) = \Gamma(m/2)/\{\sqrt{(m/2)}\Gamma[(m-1)/2]\}$, giving $g = J(m)d$ [26,27].

For crossover or matched within-participant comparisons, effect sizes were calculated with the standardised mean-change/raw-score formulation implemented as SMCRP in the metafor package [27,28]. The standardising SD was the root mean square of the two condition-specific SDs:

$$\mathrm{SD}_{\mathrm{RM}} = \sqrt{\{(\mathrm{SD}_E^2 + \mathrm{SD}_C^2)/2\}}$$

$$d_{\mathrm{RM}} = (\bar{X}_E - \bar{X}_C)/\mathrm{SD}_{\mathrm{RM}}$$

A Hedges small-sample correction was applied. The main within-participant exercise–control correlation was $r = 0.50$ when it was unreported. Here, r links the two paired condition values (or paired condition changes), not a universal baseline–follow-up correlation. With n equal to the paired analysis sample size, the effective degrees-of-freedom term and sampling variance were:

$$m = 2(n-1)/(1+r^2)$$

$$v_g = 2(1-r)/n + g^2(1+r^2)/(4n)$$

These expressions follow the repeated-measure effect-size theory and the implementation in metafor [27,28]. Sensitivity analyses repeated the paired calculations with $r = 0.30$ and $r = 0.70$.

2.11. Primary Multilevel Meta-Analysis

Because many studies contributed several eligible estimates, we used a three-level random-effects meta-analysis rather than selecting one effect per study or treating all effects as independent [15,16]. Effect sizes were nested within study families. The model can be written as:

$$g_{ij} = \mu + u_{3j} + u_{2ij} + \varepsilon_{ij}$$

Here, g_{ij} is the effect i in family j, μ is the pooled effect, u_{3j} is between-family heterogeneity, u_{2ij} is residual within-family heterogeneity, and ε_{ij} is sampling error. REML estimated the variance components [15,16,27]. The primary sampling-variance matrix V was diagonal; dependence was represented by the nested random effects and family-clustered robust inference. Non-zero sampling covariances were examined separately in sensitivity analyses. Alternative optimiser starts were checked; models without convergence or valid robust inference were to be reported rather than silently replaced by normal-theory inference.

Pooled effects and moderator coefficients used family-clustered CR2 covariance estimates with Satterthwaite degrees of freedom and two-sided 95% confidence intervals [17,18]. Interpretation prioritised estimates, interval width and certainty. A nominal two-sided α of 0.05 was used; exploratory moderator tests were not interpreted as confirmatory evidence.

2.12. Heterogeneity

We partitioned heterogeneity into level 2, representing residual variation among dependent effects within a study family, and level 3, representing variation between study families [15,16]. I^2 was reported separately for both levels. A typical sampling variance was calculated from inverse-variance weights, $w_i = 1/v_i$, as follows:

$$\bar{v} = [(m-1)\Sigma w_i]/[(\Sigma w_i)^2 - \Sigma {w_i}^2]$$

where m is the number of effect estimates. The level-specific heterogeneity proportions were then calculated as:

$$I^2_{L2} = 100 \times \tau^2_{L2}/(\tau^2_{L2} + \tau^2_{L3} + \bar{v})$$

$$I^2_{L3} = 100 \times \tau^2_{L3}/(\tau^2_{L2} + \tau^2_{L3} + \bar{v})$$

These descriptive quantities distinguish within- and between-family heterogeneity [15,16,29]. Supplementary Table S15 also gives approximate 95% prediction intervals for a new true effect, calculated from the sum of the two heterogeneity variances and squared CR2 SE, with a t reference distribution using $K - p$ degrees of freedom (K families and p fitted coefficients). They are especially uncertain when few families contribute and should not be interpreted as precise bounds for an individual response.

2.13. Small-Study Effects and Reporting Bias

Small-study effects were evaluated only when an outcome contained at least five independent study families. We used contour-enhanced funnel plots to distinguish patterns compatible with selective publication from other causes of asymmetry [30]. Funnel plots were centred on the null for significance contours, while the pooled estimate was displayed separately. We also used an Egger-type meta-regression in which Hedges' g was regressed on its SE [31]. When an outcome contained dependent effects, the regression retained the multilevel random-effects structure and used CR2 inference clustered

by study family. This avoided treating multiple time points from the same participants as independent observations [17,18]. Funnel asymmetry was interpreted cautiously because low study counts limit power, and asymmetry can arise from heterogeneity as well as publication bias [19,30,31].

2.14. Sensitivity Analyses for Dependence Assumptions

Two different correlations were varied. The paired-condition correlation r determines crossover g and its sampling variance; $r = 0.30$ and 0.70 were compared with $r = 0.50$. The working within-family sampling correlation ρ determines off-diagonal covariances between dependent estimates. A correlated-V bridge model used $r = 0.50$ and $\rho = 0.50$. The r sensitivity models held ρ at 0.50; the ρ sensitivity models held r at 0.50 and used $\rho = 0.30$ or 0.70. Thus, the bridge is not the diagonal-V primary model. The off-diagonal elements were:

$$\mathrm{Cov}(e_i, e_k) = \rho\sqrt{(v_i\, v_k)}, \qquad i \neq k$$

The diagonal remained v_i. Relative to the primary diagonal-V model, correlated-V models change weights and can change the pooled estimate even with identical included studies and observations. Changing r can additionally change the small-sample correction and sampling variance of each effect. All scenarios and intervals are reported in Supplementary Table S16. A separate sensitivity analysis excluded Holliday GLP-1 observations with an inferred denominator; no other outcome from that report was excluded for this reason.

2.15. Subgroup Analyses

Exploratory subgroup analysis required at least five families with complete moderator information and at least two families in each retained binary category. Operational categories were cohort mean age (<30/≥30 years), cohort female proportion (<50%/≥50%), exercise duration (<45/≥45 min), BMI (<25/≥25 kg/m^2), resistance content, clinical status, feeding state, intensity, training status, mode, exercise pattern and GLP-1 form. Intensity followed the source protocol label together with its reported workload or physiological metric: low and moderate were combined; vigorous, high-intensity intervals and all-out sprints were combined. Different %VO_2max/peak, ventilatory threshold, heart rate and resistance metrics were not treated as interchangeable or converted to one universal cut-off. Unclassifiable protocols were omitted only from that moderator analysis. The MICT protocol in Islam 2017 was classified in the moderate category. Protocol-specific values are retained in Supplementary Table S3 and Supplementary_Data_and_Code.xlsx.

Time of day was coded operationally as early morning (05:00–08:59), morning (09:00–11:59), afternoon (12:00–17:59) or evening/night (18:00–04:59) when recoverable. Overweight/obesity alone was distinguished from diagnosed clinical metabolic disease or bariatric surgery. Duration described the exercise bout, including interval recovery where applicable, rather than equalised laboratory observation time; the Hazell MICT and SIT bouts were 30 and 23 min, respectively. Subgroup effects used REML and CR2; between-category differences were evaluated with robust moderator-coefficient tests where available [17,18,27]. A significant effect in one category and a non-significant effect in another was not treated as evidence of a subgroup difference. The full results appear in Supplementary Tables S7–S14.

2.16. Continuous and Dose-Response Meta-Regression

Continuous meta-regression required at least seven families and three distinct moderator values, as seen in the registered plan. Separate models considered exercise duration, cohort mean age, cohort mean BMI, cohort female percentage, baseline VO_2max/peak and

post-exercise sampling time. Age, BMI, sex composition and fitness were aggregate cohort characteristics: their associations cannot be interpreted as participant-level causal effects or individual treatment-selection rules. Sampling time was a time-response variable, not an exercise dose.

For each eligible moderator, we fitted both linear and quadratic three-level REML meta-regression models. The linear model was:

$$g_{ij} = \beta_0 + \beta_1(x_{ij} - \bar{x}) + u_{3j} + u_{2ij} + \varepsilon_{ij}$$

The exploratory quadratic model added a squared centred term:

$$g_{ij} = \beta_0 + \beta_1(x_{ij} - \bar{x}) + \beta_2(x_{ij} - \bar{x})^2 + u_{3j} + u_{2ij} + \varepsilon_{ij}$$

Centring reduced the correlation between linear and squared terms. Coefficients, confidence intervals and pointwise bands used family-clustered CR2 inference [17,18,27]. Linear models were primary continuous analyses; quadratic models were exploratory. Subgroup and moderator–outcome p values were interpreted as exploratory and hypothesis-generating, not confirmatory evidence of effect modification. An apparent curve or turning point was not used to define an optimal dose or duration. Coefficients and degrees of freedom are provided in Supplementary Table S17.

2.17. Statistical Software

Effect sizes, nested REML models and CR2/Satterthwaite inference were computed using Python 3.12, with calculations checked against the corresponding original R 4.5.1 implementation using metafor and cluster-robust inference [17,18,27]. Supplementary_Data_and_Code.xlsx contains the processed inputs, the exact model memberships, the Python implementation used for the reported analyses and an equivalent R script for independent reproduction.

3. Results

3.1. Study Selection and Characteristics

The searches identified 9951 records. After the removal of 2146 duplicates, 7805 records underwent title/abstract screening. All 143 sought full-text reports were retrieved. Eighty-four were excluded: 18 for ineligible population/intervention, 54 for an ineligible or unmatched comparator, eight for unavailable usable GLP-1 outcome information and four for conference, registry, protocol or review record type. The review included 59 reports from 52 independent families. Twenty-three reports from 23 families contributed to quantitative synthesis (Figure 1).

The evidence included randomised and non-randomised crossover designs and other acute-exercise designs, with healthy or trained participants, people with overweight/obesity or diabetes, and post-bariatric-surgery populations. Cycling, locomotor, interval/sprint, resistance and multi-bout protocols were studied in differing feeding and metabolic contexts (Supplementary Tables S2 and S3). Twenty-three reports contributed selected numerical observations [21,32–53]. The other 36 reports included 30 reports from the 29 non-contributing families and six secondary reports in families represented by another report. Family-specific contributions and analytical source decisions are described in Supplementary Tables S18 and S19.



Figure 1. PRISMA 2020 flow diagram. The six-database search identified 9951 records; 7805 were screened after deduplication. Fifty-nine reports representing 52 independent families were included, of which 23 families contributed to at least one pooled outcome model.

3.2. Risk of Bias and Certainty of Evidence

There were 59 report-level assessments for 52 independent families: 48 RoB 2 assessments (47 with some concerns and one high risk) and 11 ROBINS-I assessments (six serious, four moderate and one critical). Thus, reports and families have different denominators, but the final number of assessments equals the number of included reports. Concerns were centred on randomisation reporting, period/carryover effects, missing information and selective reporting; confounding and selection were important for non-randomised reports. The plots are available in Supplementary Figures S21 and S22.

Certainty was low or very low across the principal outcomes. Risk of bias, unexplained heterogeneity, small numbers of independent families, imprecision and possible small-study effects limited confidence in the estimates. Outcome-specific ratings and reasons are reported (Supplementary Table S6).

3.3. Direct Acute GLP-1 Response

Thirteen independent families contributed 52 eligible direct-recovery effects [21,32,33,35,37,39–43,45,46,48]. Circulating GLP-1 was higher after exercise than the matched control ($g = 0.54$ (95% CI 0.25 to 0.82); $p = 0.002$). Heterogeneity was substantial: level 2 $I^2 = 35.2\%$ and level 3 $I^2 = 38.0\%$. The approximate prediction interval was −0.61 to 1.69 (Figure 2; Supplementary Figure S1; Supplementary Table S15).

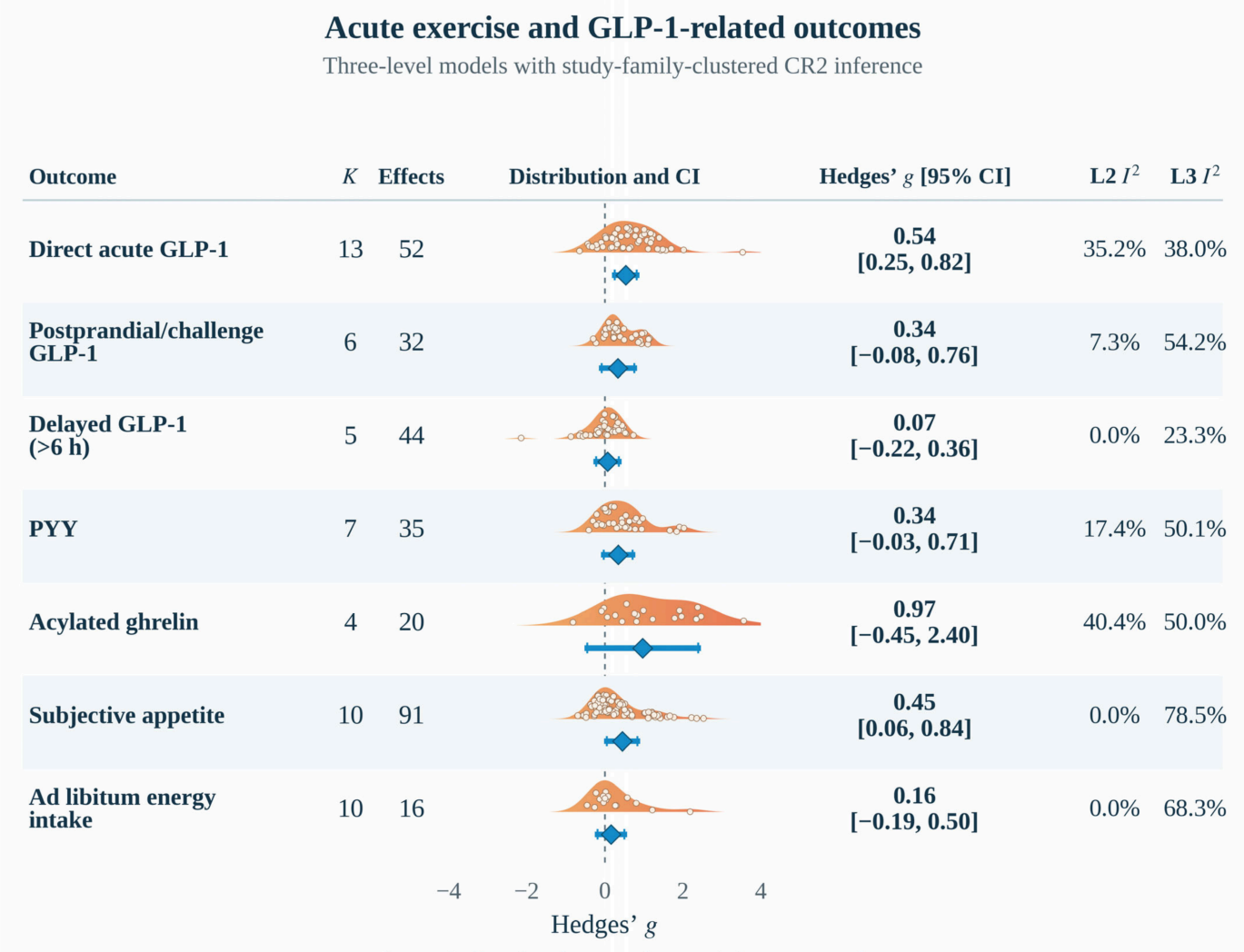


Figure 2. Main pooled outcomes. Diamonds and bars show Hedges' g and 95% confidence intervals; the distribution display is descriptive and does not add independent studies. Positive values denote higher GLP-1/PYY or lower ghrelin, appetite ratings and intake after outcome-specific direction coding, not demonstrated GLP-1-mediated appetite suppression. K denotes independent families and E denotes effect estimates. Level 2 and level 3 I^2 describe within- and between-family heterogeneity.

Total GLP-1 contributed 7 families and 27 effects (g = 0.50 (95% CI 0.16 to 0.84)); active/intact GLP-1 contributed 3 families and 16 effects (g = 0.75 (95% CI −0.45 to 1.95)). The active/intact interval was much wider. These separate estimates do not establish a difference between molecular forms (Supplementary Figure S2).

3.4. Temporal Profile of GLP-1

At 0–15 min, 13 families contributed 22 effects (g = 0.45 (95% CI 0.19 to 0.70)); at >15–60 min, 8 families contributed 18 effects (g = 0.58 (95% CI 0.02 to 1.14)); and at >60–120 min, 4 families contributed 12 effects (g = 0.71 (95% CI −0.33 to 1.75)) [21,32,33,35,37,39–43,45,46,48]. The later interval was wider and included the null (Figure 3; Supplementary Figure S3).

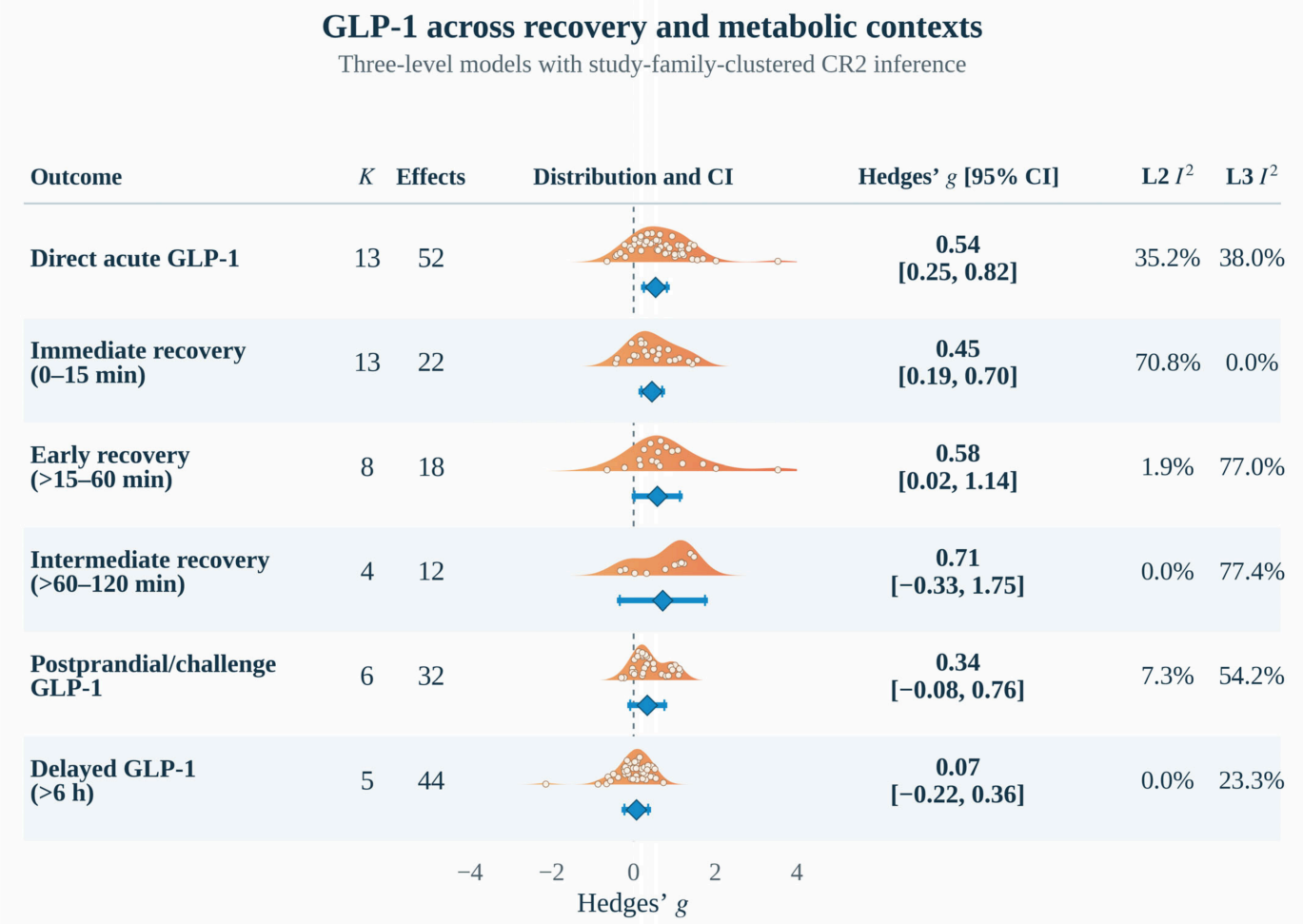


Figure 3. GLP-1 by recovery window and metabolic context. Diamonds and bars show pooled Hedges' g and 95% confidence intervals; full numerical limits are printed. The direct-recovery windows contain partly different study families. Post-prandial/challenge and delayed outcomes are different physiological contexts, not consecutive points on the same direct-recovery trajectory. Wider intervals are uncertainty, not evidence of declining hormone responses.

Point estimates did not decline across these windows. The direct time-response slope over 0–120 min was $\beta = 0.00247$ (95% CI −0.00317 to 0.00810) per minute ($p = 0.315$). In the quadratic model centred at 35.96 min, $\beta_1 = 0.00347$ (95% CI −0.00403 to 0.01097) and $\beta_2 = -0.0000570$ (95% CI −0.0002559 to 0.0001419) per minute^2 ($p = 0.478$ for the squared term). Neither model demonstrated a temporal decline. The windows contain partly different families and are not a single longitudinal decay curve; neither persistence for a defined duration nor return to baseline can be established.

3.5. Post-Prandial, Challenge and Delayed GLP-1 Responses

Post-prandial/challenge GLP-1 comprised six families and 32 effects ($g = 0.34$ (95% CI −0.08 to 0.76)) [32,35,41,48,52,53]. The total-GLP-1 subset included four families/24 effects ($g = 0.26$ (95% CI −0.51 to 1.02)), and the AUC subset included three families/six effects ($g = 0.04$ (95% CI −0.49 to 0.58)). These contexts should not be combined with pre-meal direct recovery (Supplementary Figure S4).

Delayed GLP-1 (>6 h) comprised five families and 44 effects ($g = 0.07$ (95% CI −0.22 to 0.36)) [34–36,38,47]. All observations met the delayed-response definition. The result

concerns delayed measurements, including next-day contexts, and does not establish when a direct response ended (Supplementary Figure S5; Supplementary Table S19).

3.6. PYY, Ghrelin, Subjective Appetite and Energy Intake

PYY comprised seven families/35 effects ($g = 0.34$ (95% CI −0.03 to 0.71)); acylated-ghrelin suppression comprised four families/20 effects ($g = 0.97$ (95% CI −0.45 to 2.40)). Molecular-form-specific PYY and ghrelin results are reported separately; these secondary hormone outcomes should not be treated as proof of a common behavioural mechanism (Supplementary Figures S6 and S7).

The combined subjective-appetite model included 10 families and 91 effects ($g = 0.45$ (95% CI 0.06 to 0.84)) [21,37,39–46]. Its interval excluded zero and favoured suppression on the harmonised scale, but certainty was very low. The signal was sensitive to covariance assumptions: at $r = 0.50$ and $\rho = 0.70$, $g = 0.29$ (95% CI −0.02 to 0.60). This aggregate includes distinct ratings, not measured food consumption. Hunger, fullness, satiety, prospective food consumption and composite appetite were also analysed separately (Supplementary Figures S8 and S9).

Ad libitum energy intake included 10 families and 16 effects ($g = 0.16$ (95% CI −0.19 to 0.50)) [37,40,41,43–46,50,51,53]. The interval included lower and higher intakes; a reduction in actual food consumption was not established. Absolute intake, rather than intake after subtracting exercise energy expenditure, was used (Supplementary Figure S10).

3.7. Qualitative Synthesis of Additional Included Evidence

Twenty-nine families did not contribute to the selected pooled models. Their primary reasons were no compatible matched exercise–rest contrast (19), unresolved dispersion or time assignment (five), unresolved outcome-specific denominators or condition matching (two), non-harmonised numerical summaries (two), and an intermittent-walking/iAUC estimand outside the selected model (one). These studies were retained in the qualitative review. Supplementary Table S18 identifies each family and the six additional non-selected reports within contributing families, distinguishing non-contribution to a particular pooled model from exclusion from the review.

Additional included reports broadened the evidence base across dietary and pharmacological co-interventions, glycaemic and clinical contexts, hypoxia or environmental exposures, sex-specific responses, and other mechanistic or protocol comparisons [54–72].

Head-to-head exercise studies did not support a single intensity or mode rule for GLP-1. Comparisons of moderate continuous exercise with high-intensity continuous, interval or sprint exercise often showed post-exercise changes in GLP-1, but between-protocol differences were inconsistent [73–79]. In several experiments, intensity or mode appeared to separate PYY, acylated ghrelin or perceived appetite more clearly than GLP-1 itself [74,75,78]. This qualitative evidence supports the use of intensity and mode as hypothesis-generating modifiers rather than fixed determinants of the GLP-1 response.

The response was also sensitive to the metabolic context surrounding exercise. Studies of metformin, post-exercise fasting or feeding, exogenous ketones, pre-exercise meal composition and recovery nutrition showed that GLP-1 and related appetite signals can be altered by what occurs before, during or after the exercise bout [80–84]. These hormonal changes did not consistently translate into lower subsequent food intake. The findings support our decision to keep factorial and nutrition-modified experiments separate from the simple exercise-versus-rest pooled estimand.

Participant and clinical modifiers produced similarly mixed findings. Direct comparisons between lean and overweight or obese groups before and after bariatric surgery and between reproductive or sex-related strata suggested that GLP-1, PYY and ghrelin responses

can differ in selected settings, but no modifier produced a consistent clinical rule [53,85–87]. These studies strengthen the rationale for testing age, adiposity and sex-related factors in future trials, while also showing why study-level subgroup signals should not be interpreted as individual-level treatment effects.

Environmental and mechanistic experiments further widened the range of plausible pathways. Ambient temperature and the experimental manipulation of lactate changed appetite-related responses in some studies [88,89]. Other included reports examined hypoxia, water-based exercise, intermittent walking and related mechanistic contexts. Taken together, the qualitative evidence shows that the endogenous GLP-1–appetite response is context dependent. It complements the pooled estimate rather than providing a larger pool of exchangeable effects.

3.8. Small-Study Effects and Sensitivity Analyses

For direct GLP-1, the Egger-type slope test gave $p = 0.006$; for post-prandial/challenge GLP-1, $p = 0.009$. All eligible diagnostics are shown in Supplementary Figures S11–S13. Asymmetry may reflect selective reporting, heterogeneity or dependence-related features. Non-significant tests, particularly with five to nine families, do not exclude reporting bias.

The diagonal-V primary direct GLP-1 result was $g = 0.54$ (95% CI 0.25 to 0.82). With the same observations, the $r = 0.50$ and $\rho = 0.50$ correlated-V bridge gave 0.45 (95% CI 0.19 to 0.70). At $\rho = 0.50$, $r = 0.30$ and 0.70 gave 0.45 (95% CI 0.19 to 0.72) and 0.44 (95% CI 0.20 to 0.68), respectively; at $r = 0.50$, $\rho = 0.30$ and 0.70 gave 0.49 (95% CI 0.22 to 0.75) and 0.41 (95% CI 0.16 to 0.67). These differences arise from covariance assumptions and weighting, not from different inclusion sets. Excluding the Holliday GLP-1 observations with an inferred n gave 0.49 (95% CI 0.20 to 0.78) (12 families/47 effects). The full scenario results are provided in Supplementary Table S16 and Supplementary Figures S14 and S15.

3.9. Subgroup Analyses

Subgroup analyses were exploratory and frequently based on few or unbalanced families. Table 1 summarises the selected direct GLP-1 comparisons with their between-subgroup tests; Supplementary Tables S7–S14 provide all eligible outcome-specific results. Differences in point estimates were not treated as individual-level response predictors.

Table 1. Selected exploratory subgroup comparisons for direct acute GLP-1.

Characteristic	Direct GLP-1: g (95% CI)	p for Difference	Interpretation
Cohort mean age	<30 y: 0.56 (95% CI 0.25 to 0.87) ≥30 y: 0.49 (95% CI −0.16 to 1.14)	0.673	Exploratory cohort-level association; not an individual response rule.
Exercise intensity	Low/moderate: 0.37 (95% CI 0.09 to 0.65) Vigorous/high: 0.77 (95% CI 0.41 to 1.12)	0.055	Exploratory protocol-level association; categories are not prescribing thresholds.
Exercise duration	<45 min: 0.74 (95% CI 0.36 to 1.11) ≥45 min: 0.26 (95% CI −0.17 to 0.68)	0.059	Exploratory protocol-level association; categories are not prescribing thresholds.
Cohort mean BMI	BMI < 25: 0.60 (95% CI 0.24 to 0.96) BMI ≥ 25: 0.47 (95% CI −0.09 to 1.02)	0.629	Exploratory cohort-level association; not an individual response rule.
Cohort female proportion	Female < 50%: 0.52 (95% CI 0.24 to 0.80) Female ≥ 50%: 0.67 (95% CI −0.21 to 1.55)	0.391	Exploratory cohort-level association; not an individual response rule.
Exercise mode	Cycling: 0.68 (95% CI 0.22 to 1.14) Weight-bearing locomotor: 0.48 (95% CI 0.04 to 0.91)	0.387	Exploratory protocol-level association; categories are not prescribing thresholds.

Values are subgroup-specific estimates; the separate moderator test evaluates a between-subgroup association. Cohort means and proportions are ecological characteristics. The categories are not clinical thresholds.

For direct GLP-1, <30 y: $g = 0.56$ (95% CI 0.25 to 0.87); ≥30 y: $g = 0.49$ (95% CI −0.16 to 1.14). For exercise intensity, low/moderate: $g = 0.37$ (95% CI 0.09 to 0.65); vigorous/high: $g = 0.77$ (95% CI 0.41 to 1.12). These are study/cohort-level associations with estimated effects, not evidence that age or intensity independently determines an individual's GLP-1 response.

The duration, BMI, sex-composition and mode estimates in Table 1 are imprecise cohort/protocol associations. Their study-level nature precludes individual response prediction, sex-specific prescriptions or an optimal bout duration.

3.10. Continuous and Dose-Response Meta-Regression

The direct sampling-time coefficients are reported in Section 3.4 and Supplementary Table S17. Neither the linear slope nor the exploratory quadratic term demonstrated a decline. Sampling windows from different protocols cannot substitute for a controlled within-person recovery trajectory.

For direct GLP-1, the linear duration coefficient was $\beta = -0.00534$ (95% CI −0.01315 to 0.00248) per minute. Other eligible cohort and protocol moderators are reported with coefficients, confidence intervals, degrees of freedom and observed ranges in Supplementary Table S17.

Table 2 separates the continuous associations from their inferential limits. Quadratic fits were not used to identify an optimum or a causal recovery-time mechanism. Limited independent family counts, correlated protocol characteristics and ecological confounding restrict interpretation (Supplementary Figures S16–S20).

Table 2. Continuous exploratory moderators of direct acute GLP-1.

Moderator	Observed Range/Families	Linear Coefficient (95% CI)	Interpretation
Post-exercise time	0.0–120.0; $K = 13$	0.00247 (95% CI −0.00317 to 0.00810); $p = 0.315$	No demonstrated decay; separate studies/times are not a single trajectory.
Exercise duration	14.0–120.0; $K = 13$	−0.00534 (95% CI −0.01315 to 0.00248); $p = 0.129$	Univariable and exploratory; confounding and ecological interpretation apply.
Cohort mean age	20.6–68.5; $K = 13$	−0.01005 (95% CI −0.03847 to 0.01836); $p = 0.228$	Univariable and exploratory; confounding and ecological interpretation apply.
Cohort female percentage	0.0–100.0; $K = 13$	0.00535 (95% CI −0.01251 to 0.02322); $p = 0.272$	Univariable and exploratory; confounding and ecological interpretation apply.
Cohort mean BMI	21.0–35.5; $K = 12$	−0.01278 (95% CI −0.06792 to 0.04236); $p = 0.541$	Univariable and exploratory; confounding and ecological interpretation apply.
Cohort VO_2max/peak	24.2–62.8; $K = 9$	−0.00003 (95% CI −0.03567 to 0.03561); $p = 0.998$	Univariable and exploratory; confounding and ecological interpretation apply.

Coefficients refer to study/cohort-level associations in separate univariable models. Linear models are primary continuous analyses; squared terms are exploratory. Confidence intervals use family-clustered CR2/Satterthwaite inference.

4. Discussion

4.1. Principal Findings

The principal finding is an acute exercise-associated increase in circulating GLP-1 during direct recovery, with low or very low certainty and substantial variation across studies. The direct time-window point estimates do not decline, while later windows contain fewer families and wider confidence intervals. This is increasing uncertainty, not demonstrated decay. Post-prandial and delayed measurements answer different physiological questions and cannot establish the duration of the direct response.

The aggregate subjective appetite estimate favours suppression, whereas the measured energy intake remains inconclusive. These are separate outcomes, often drawn from different subsets of studies. Their coexistence does not establish that GLP-1 mediated either response. Endogenous GLP-1 biology also differs from sustained pharmacological receptor agonism, so the present effect size is not a pharmacological equivalence or weight-loss estimate [6,7,90–93].

4.2. Physiological and Measurement Considerations

GLP-1 secretion and action involve nutrient sensing, metabolic signals and neural pathways [90–93]. Exercise changes sympathetic activity, blood flow, substrate availability, and lactate and myokine release; these are plausible contributors, not mechanisms identified by this meta-analysis. Individual experiments reported associations of lactate and interleukin-6 with appetite hormones [42], and blood-flow-restricted exercise altered Lac-Phe and related signals [45]. Experimental Lac-Phe work provides a separate biological hypothesis [94]. None of these observations establish that a specific metabolite causes the pooled GLP-1 effect or mediates food intake.

Assay and pre-analytical differences are particularly important. Total assays and active/intact assays quantify different aspects of the GLP-1 system. The rapid degradation of active GLP-1, use of DPP-4/protease inhibitors, processing delay, storage, matrix and immunoassay specificity can affect measured concentrations [7,8,92]. The active/intact synthesis is also based on fewer families. Standardising outcomes as g does not remove these biological or laboratory differences. An analyte's short circulating half-life is not evidence that the exercise-associated secretion or concentration response must be short-lived.

4.3. Hormonal Changes and Eating Behaviour Are Not the Same Endpoint

Subjective appetite, circulating hormone concentrations and eating behaviour are not interchangeable. Food intake integrates gastric distension, other gut peptides, nutrient availability, reward, expectations and food access [91,92]. The subjective-rating result should therefore be described as a pooled rating difference, not a demonstrated GLP-1-driven functional benefit. The present review did not perform a mediation analysis, and temporal covariation within the same participants was not consistently available.

Individual studies illustrate this distinction. Walking in women with overweight/obesity did not provoke greater absolute intake despite the exercise energy cost [52]; other trials showed hormone or appetite differences without a consistent intake difference [37,40,42–44,46,51]. Preserved exercise-related energy expenditure without compensatory eating is plausible, but our absolute-intake synthesis neither proves equivalence between conditions nor establishes a clinically meaningful reduction in food consumption.

Head-to-head protocols, nutritional manipulations and reproductive or clinical contexts also produced heterogeneous hormone and appetite patterns [53,73–87]. These qualitative observations help define mechanistic questions but were not pooled with unmatched exercise–rest contrasts. Future experiments need simultaneous hormone measures, ratings and objectively measured intake to distinguish parallel responses from a causal sequence.

4.4. Intensity, Duration and the Timing of Recovery

Intensity and duration could contribute to heterogeneity through metabolic demand, work, energy expenditure and feeding context. However, intensity categories retain study-specific labels rather than a common calibrated scale, and dose variables are correlated with participant and protocol characteristics. A subgroup difference or a univariable duration slope cannot isolate an intensity- or duration-specific mechanism.

The operational 45 min split is not a therapeutic threshold. Work- and energy-matched comparisons have produced heterogeneous hormone and behavioural findings [42,46,50,73–79]. More informative experiments would manipulate intensity and duration within participants, match relevant co-interventions, and measure recovery at the same times. This would separate protocol effects from differences in who was studied and when blood was sampled.

4.5. Age, Sex and Adiposity as Candidate Modifiers

Mean age, mean BMI and female proportion are cohort descriptors. They are subject to the same ecological limitation: a between-cohort association is not an individual-level moderator effect. Their estimates can reflect differences in fitness, medication, feeding, exercise mode, laboratory methods or sampling times. The age results do not establish a loss of GLP-1 responsiveness with ageing; the age-stratified trial also did not show a simple age-related GLP-1 rule [39].

Sex-composition results likewise should not be translated into claims about how a given woman or man responds. The estimates do not consistently favour male-dominant cohorts. Sex-balanced designs with menstrual-phase, contraceptive and menopausal information, followed by participant-level interaction analyses, are needed. The reproductive-phase evidence is mechanistically relevant but does not establish a universal binary sex effect [86,87].

Positive numerical effects across BMI strata do not prove equivalence or preserved responsiveness for every person with obesity. Clinical and bariatric contexts may change both physiology and measurement, while small cohorts yield unstable subgroup estimates [43,46,51,53,85]. Current aggregate data support targeted research across weight and metabolic states, not individual prediction from BMI.

4.6. Clinical and Public-Health Relevance in the GLP-1 Era

These findings are relevant to metabolic research but do not establish a clinical exercise prescription. Exercise has health benefits that extend beyond appetite or GLP-1, and guideline recommendations do not depend on demonstrating this particular hormonal pathway [3–6]. The synthesis adds evidence about circulating GLP-1 after acute exercise while leaving the response duration and behavioural mechanism unresolved.

Exercise and GLP-1-based medicines may have complementary effects, as trials of combined treatment have examined weight-loss maintenance, cardiometabolic outcomes and bone health [95,96]. Those intervention trials address different exposures and timescales. Our acute endogenous-hormone findings do not support medication replacement, a change in drug dose, or prescribing exercise around an injection. Exercise–meal and exercise–medicine timing remain questions for direct experimental testing.

A useful translational design would standardise meals and measurement timing while assessing both hormone exposure and actual eating. The current results cannot identify a duration of appetite protection or an optimal meal-planning window. Clinical relevance depends on reproducible behavioural and metabolic outcomes, not on the direction of a single biomarker.

4.7. Strengths and Limitations

Strengths include a six-database search, explicit family linkage, the separation of physiological contexts and GLP-1 forms, and the retention of repeated measurements with multilevel models and family-clustered inference. The supplements provide a complete contribution register, documented source selections and directly accessible processed data and code. Direction harmonisation aids reading across outcomes without treating them as a single causal appetite pathway.

Limitations include the small number of independent families contributing to several models: 23 of 52 included families supplied selected quantitative observations, and 13 contributed to the principal direct model. Incomplete primary reporting and figure digitisation introduce uncertainty. Risk-of-bias concerns, assay heterogeneity and possible small-study effects limit certainty. Later recovery windows contain partially different families from early windows. Cohort-level moderators are ecological and exploratory, and nonlinear fits are especially uncertain. The inferred Holliday GLP-1 denominator was examined in a separate sensitivity analysis. These constraints limit clinical interpretation, duration claims and individual response prediction.

4.8. Priorities for Future Research

Future trials should randomise exercise and matched rest, standardise recent meals and hydration, and obtain measurements before and at prespecified times after exercise, extending sufficiently beyond 120 min to assess persistence and return towards the baseline. Both total and active GLP-1 should be measured with documented inhibition, processing and storage. Concurrent PYY, ghrelin, metabolic signals, subjective ratings and a standardised ad libitum meal would support explicit temporal and mediation analyses rather than an assumed hormone-to-appetite chain.

Larger and more diverse studies should report outcome-specific denominators, paired correlations, numerical time-course tables and all planned outcomes. Experiments should separate intensity from energy expenditure and bout duration from total work and retain matched feeding. Participant-level analyses are needed to assess age, sex, BMI and fitness without ecological inference. Data sharing would also reduce the reliance on figure digitisation and enable direct comparisons of recovery trajectories.

5. Conclusions

Acute exercise is associated with higher circulating GLP-1 during direct recovery, but the current evidence does not establish the duration of this response or a temporal decline. Pooled subjective appetite ratings favour suppression in the primary model, but are sensitive to dependence assumptions, whereas ad libitum energy intake remains inconclusive; neither result demonstrates that GLP-1 mediates eating behaviour. Heterogeneity and low or very low certainty limit clinical interpretation, and cohort-level subgroup or dose-response associations remain exploratory. Standardised, densely sampled trials with matched feeding and concurrent hormone, appetite and intake measurements are needed before duration claims or individual exercise prescriptions can be made.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/metabo16120945/s1, Supplementary Figures S1–S22 and Supplementary Tables S1–S20 are supplied as two PDF documents. Supplementary_Data_and_Code.xlsx contains the processed numerical inputs, source identifiers, model memberships, moderator coding, Python analysis code and equivalent R reproduction code.

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X.L., Y.D., D.L., M.Z. and X.M.; visualization, M.Z. and X.M.; writing—original draft preparation, X.L. and Y.D.; writing—review and editing, X.L., Y.D. and B.L.; supervision, B.L.; project administration, B.L.; funding acquisition, X.L. and B.L. All authors have read and agreed to the published version of the manuscript.

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