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# Toward standardized personalized nutrition: a methodological scoping review and meta-analysis of randomized controlled trials in adults with overweight and obesity

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## Abstract

**Background** Personalized nutrition (PN) interventions are increasingly used to address overweight and obesity, yet randomized controlled trials (RCTs) vary widely in how personalization is defined and delivered. This scoping review synthesized methodological characteristics of PN RCTs in adults with overweight or obesity and meta-analyzed PN effects on anthropometrics.

**Main text** We searched PubMed, Scopus, Web of Science, and ClinicalTrials.gov through November 20, 2025. Data were extracted using a standardized template capturing intervention characteristics. A random-effects meta-analysis was conducted, Bayesian meta-regression was used to explore the effect of intervention duration, and certainty of evidence was evaluated using GRADE. Among the 17 included RCTs, none provided a definition of PN, and substantial methodological heterogeneity was evident. Personalization inputs differed markedly in both number and type, and delivery modes ranged from digital platforms and group sessions to meal-box provision and routine dietitian consultations. PN interventions typically used the same delivery format as their control groups but layered on individualized components, with some providing higher contact frequency. Comparator structures also varied, from standardized guideline-based diets to minimal or no dietary counseling. Personalized diets were produced through algorithmic models, dietitian-guided tailoring, or continuous glucose monitoring-guided participant selection. PN produced a small but significant reduction in body weight, while effects on body mass index (BMI) were non-significant. Longer intervention duration was credibly associated with greater reductions in body weight and BMI. No publication bias was detected, and certainty of evidence was low for body weight and very low for BMI.

**Conclusions** PN interventions show modest effects on body weight but not BMI, within a highly heterogeneous methodological landscape. This review identifies design features to guide more effective PN interventions. Advancing toward more standardized approaches will require clearer operational definitions of personalization, better-aligned comparator diets, and greater transparency in how personalized strategies are generated and delivered.

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**Keywords** Personalized nutrition, Precision nutrition, Randomized controlled trials, Overweight, Obesity, Meta-analysis, Scoping review, Dietary personalization

## Introduction

The global obesity epidemic continues to pose an urgent challenge to public health, increasing the risk of type 2 diabetes, cardiovascular disease, and other metabolic disorders [1]. Although population-level dietary guidelines remain essential, they often overlook the considerable interindividual variability in metabolic responses to dietary intake, particularly among individuals with overweight or obesity [2, 3]. In response, personalized nutrition (PN) has emerged as a promising framework for tailoring dietary interventions based on individual-level features such as genotype, phenotype, gut microbiota, lifestyle, and environmental exposures [4].

Recent advances in molecular profiling, wearable technologies, and machine learning have enabled the development of PN strategies that move beyond generic dietary recommendations [5]. However, translating these innovations into clinically meaningful outcomes requires rigorous validation through randomized controlled trials (RCTs) [4, 5]. To date, the substantial methodological diversity across PN RCTs has constrained the reproducibility and generalizability of findings, owing in part to algorithmic bias, limited external validity, and data privacy concerns [5, 6]. Consequently, establishing coherent methodological standards that allow for valid and consistent comparisons across PN interventions remains a significant challenge [4]. Therefore, clarifying how personalization is operationalized is essential for identifying methodological patterns, exposing inconsistencies, and guiding efforts to improve standardization and real-world applicability. In the absence of this foundational insight, the field risks remaining fragmented, with interventions that are challenging to replicate, compare, or translate beyond controlled research environments [4, 7]. Despite growing interest in PN, there is a lack of reviews focusing specifically on methodological approaches used in current trials.

To advance the field, this scoping review summarizes several aspects of how RCTs have designed and implemented PN interventions for adults with overweight or obesity. To that end, we synthesize the spectrum of individual-level features used to guide personalization, the range of comparator diet strategies, and the delivery approaches and contact intensity that characterize current intervention models. In addition, we conducted a meta-analysis of body weight and body mass index (BMI) outcomes to assess the effectiveness of PN across heterogeneous trial designs. This dual focus enables a more integrated understanding of how current PN

interventions are structured and how their design may influence observed effectiveness.

## Methods

### Framework and protocol

This scoping review was guided by the methodological framework developed by Arksey and O'Malley, which comprises six sequential stages: (1) identifying the research question (2), identifying relevant studies (3), study selection (4), charting the data (5), collating, summarizing, and reporting the results, and (6) consulting stakeholders (optional) (8). The conduct and reporting of the review also adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews (PRISMA-ScR) guidelines. The protocol was registered prospectively in PROSPERO under the registration number CRD420251245137.

### Stage 1: Identification of research questions

In the first stage, we defined the overarching research questions that guided the scope and direction of the review, as well as the data-extraction framework and analytical strategy. Given the substantial heterogeneity in how PN interventions are conceptualized and operationalized, our primary question was: How have RCTs in adults with overweight and obesity designed and implemented PN interventions? To address this, we articulated four subsidiary questions:

- 1) What individual-level features have been used to generate personalized dietary recommendations?
- 2) What PN interventions and its control diets have been used in RCTs?
- 3) What delivery modes, levels of contact intensity, and types of personalized-diet generators are employed in RCTs?
- 4) What is the overall effectiveness of PN interventions on body weight and BMI across RCTs?

### Stage 2: Identification of relevant studies

A comprehensive search was conducted in PubMed, Scopus, Web of Science, and ClinicalTrials.gov from inception through November 20, 2025. The strategy integrated terms related to “personalized nutrition,” “precision nutrition,” “nutrigenomics,” and “randomized controlled trial,” employing Boolean operators and controlled vocabulary (e.g., MeSH). Additional records were identified through backward citation tracking of relevant reviews. No language or publication date restrictions

were applied. The full search strategy is provided in Supplementary Material S1.

Stage 3: Selection of studies

Eligibility criteria were established according to the Population, Intervention, Comparator, Outcome, and Study Design (PICOS) framework, which is presented in Table 1. Because no universally accepted definition of PN currently exists, we prespecified an operational definition to ensure transparent, reproducible, and consistent study selection. Previous literature describes PN aims to maintain or improve health through personalized dietary and lifestyle recommendations informed by genetic and phenotypic characteristics, medical history, dietary intake, lifestyle behaviors, nutritional status, and gut microbiome composition [4, 9, 10]. However, “personalization” based solely on lifestyle or behavioral information can be difficult to distinguish from conventional dietetic counseling and would substantially broaden eligibility to include many standard counseling interventions. Therefore, to improve conceptual clarity and reduce between-study heterogeneity, we required eligible interventions to incorporate at least one biological input (e.g., genetic, metabolic/clinical biomarker, physiological, or microbiome-related information). This minimum biological criterion reduced conceptual ambiguity and enabled a clearer distinction between biologically informed PN interventions and standard dietary advice.

All references were imported into Zotero Desktop for deduplication. Two independent reviewers initially screened titles and abstracts, followed by full-text articles, according to the pre-specified inclusion criteria.

Discrepancies were resolved through discussion or by a third reviewer.

Stage 4: data charting

Data from all included studies were charted using a standardized extraction template developed in Microsoft Excel for this review. Two reviewers independently extracted and cross-verified all entries, resolving discrepancies through discussion and consensus. Extracted variables captured both methodological and intervention-specific characteristics, including:

- **Study characteristics** such as authors, publication year, country, specific RCT designs, sample size, and intervention duration;
- **Participant characteristics**, including age, body weight, and BMI;
- **Personalization features**, documenting all individual-level features used to generate personalized dietary recommendations and categorizing them into the following eight groups: (1) anthropometric measures (e.g., BMI, body weight, waist circumference, body fat percentage); (2) blood biomarkers and clinical laboratory measures (e.g., fasting glucose, HbA1c, triglycerides, cholesterol, liver enzymes, insulin, cytokines); (3) genetic data and single-nucleotide polymorphisms (e.g., individual genotypes, nutrigenetic panels); (4) microbiome features (e.g., stool or oral microbiome profiles, taxonomic or functional microbial markers); (5) nutrient or dietary composition measures (e.g., macronutrient intake, food metadata, glycemic load,

**Table 1** Inclusion and exclusion criteria applied to randomized controlled trials of personalized nutrition in adults with overweight or obesity

|               | Inclusion criteria                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              | Exclusion criteria                                                                                                                                                                           |
|---------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Population    | Free-living adults (aged ≥ 18 years) classified as overweight or obese based on BMI or other adiposity measures as defined in the original studies.                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             | Studies involving non-human models, individuals younger than 18 years, pregnant women, or those who were breastfeeding.                                                                      |
| Intervention  | Dietary interventions were considered personalized or individualized if the authors reported tailoring recommendations based on individual participant characteristics. Personalization had to include at least one biological input, such as genetic markers (e.g., FTO), metabolic biomarkers (e.g., blood glucose or lipid profiles), or gut microbiome composition. Interventions could also incorporate additional individual-level characteristics, including anthropometric measures, dietary intake, medical history, or physical activity, alongside these biological inputs. Interventions may have been tailored through clinical judgment, algorithmic prediction models, or a combination of both. | • Dietary counseling provided solely on the basis of lifestyle or behavioral factors.<br>• Insufficient detail regarding how the personalized dietary approach was developed or implemented. |
| Comparator    | Any control condition, such as standard care, population-based dietary guidance, or alternative non-personalized dietary interventions.                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         | No control group.                                                                                                                                                                            |
| Outcome       | Changes in anthropometric measures body weight [kg] and (BMI [kg/m <sup>2</sup> ]) were assessed by comparing values before and after the PN intervention.                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      | Reports outcomes solely in original units that cannot be converted to a unified or standardized metric.                                                                                      |
| Study Designs | RCTs with at least two arms.                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    | Non-RCTs.                                                                                                                                                                                    |

Abbreviations: BMI, body mass index; PN, personalized nutrition; RCT, randomized controlled trial; FTO, fat mass and obesity–associated gene; kg, kilogram; kg/m<sup>2</sup>, kilograms per square meter

nutrient profiles); (6) glycemic response features derived from continuous glucose monitoring or postprandial glucose tests; (4) lifestyle and behavioral questionnaire data (e.g., self-reported lifestyle, physical activity, sleep, behavioral factors); and (7) sensor-derived physiological data (e.g., steps, heart rate, energy expenditure, sleep, and other wearable-based metrics not related to glucose).

- **Comparator group structure and intervention group**, including the type of control condition (e.g., standard dietary guidance, usual care, or no intervention), and detailed information on the PN intervention, such as the specific personalization approach used and the algorithms or clinical personnel involved in generating personalized dietary recommendations;
- **Intervention delivery**, encompassing the delivery format (e.g., in-person, app-based, or remote), and frequency and duration of contact (intervention intensity);
- **Outcomes**, measuring baseline and endline values or changes in body weight (kg) and BMI (kg/m<sup>2</sup>), when reported. All data fields were reviewed iteratively to ensure completeness and consistency across the included trials.

#### Stage 5: data collation, summary, and reporting

All extracted information was summarized descriptively and organized using Microsoft Excel 2025. When two or more studies reported sufficiently comparable quantitative data for an outcome, a meta-analysis was performed. Because substantial heterogeneity was anticipated across trials, reflecting differences in participant characteristics, combinations of personalization inputs, and modes of delivering personalized diets, we used an inverse-variance random-effects model. The Sidik–Jonkman estimator with Hartung–Knapp adjustment was used to account for both within-study and between-study variance. Mean differences (MDs) from baseline, together with their corresponding 95% confidence intervals (CIs), were pooled. The effects of PN interventions on body weight and BMI were quantified using the MD between the PN and control groups. When studies did not report absolute change values, mean changes and their corresponding standard deviations (SDs) were derived in accordance with the Cochrane Handbook for Systematic Reviews of Interventions [11, 12]. Mean change was calculated as the within-group difference between baseline and post-intervention measurements, and the SD of the change was estimated using the following formula [12]:

$$SD_{change} = \sqrt{SD_{baseline}^2 + SD_{final}^2 - (2 \times Corr \times SD_{baseline} \times SD_{final})}$$

In instances where SDs were not provided, we estimated them from the reported confidence intervals according to the following formula [11]:

$$SD = \sqrt{N} \times (upper\ limit - lower\ limit) / 3.92$$

Heterogeneity was quantified using the  $I^2$  statistic and interpreted using conventional thresholds: 0–40% (low), 30–60% (moderate), 50–90% (substantial), and 75–100% (considerable heterogeneity). In line with Cochrane recommendations, potential publication bias was evaluated through visual inspection of funnel plots and Egger's regression test when an outcome included at least ten studies [13].

Additionally, a Bayesian meta-regression was performed using intervention duration (weeks) as a moderator to evaluate its influence on effect sizes. The model employed weakly informative priors and was fitted using Markov Chain Monte Carlo sampling. Posterior estimates were summarized using means and 95% credible intervals to quantify uncertainty around the effect of duration.

Statistical significance was defined as a two-sided  $p < 0.05$ . All analyses and visualizations were conducted in RStudio (version 2025.05.0 Build 496; RStudio, Boston, MA, USA) using the *meta*, *metafor*, *brms* and *ggplot2* packages.

#### Stage 6: Relevant stakeholder consultation

This stage was not undertaken because the review was designed as an evidence-based methodological synthesis using published data only, and stakeholder involvement was outside the planned scope and resources of this review.

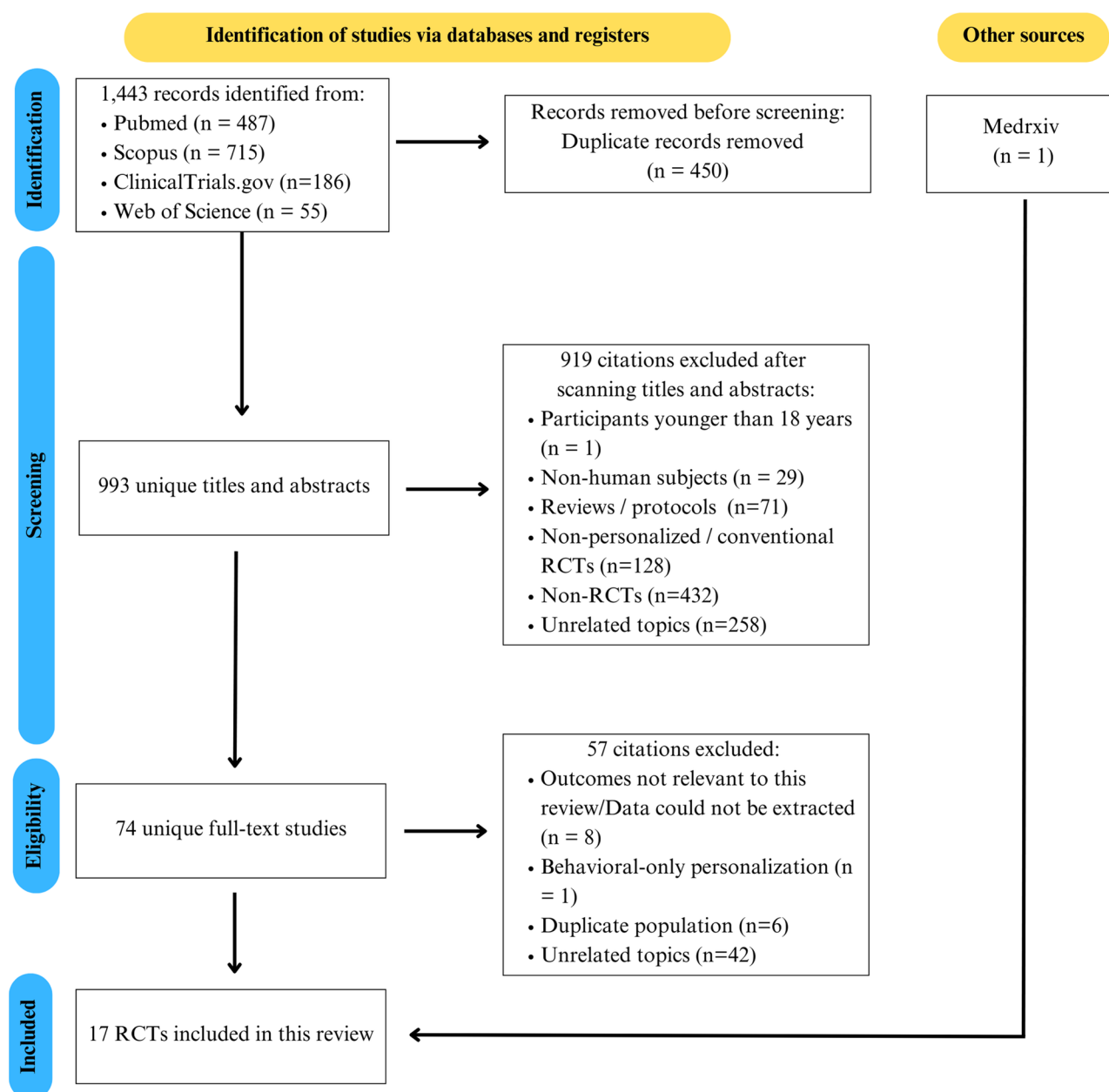
#### Quality of evidence assessment

We assessed the overall certainty of evidence using the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) approach [14]. All outcomes were independently evaluated by two researchers.

## Results

### Selected papers

As detailed in Fig. 1, our initial search identified 1,444 records. After duplicate removal, 919 articles were excluded based on title and abstract screening, leaving 74 articles for full-text assessment. The reasons for exclusion at this stage were unrelated topics, outcomes not relevant to this review or insufficient data for extraction, duplicate populations, and behavioral-only personalization without



**Fig. 1** Flowchart of the study selection

biological inputs. Ultimately, 17 articles met all eligibility criteria and were included in the review [15–31].

#### Characteristics of the Included Interventions

As described in Supplementary S2, across the 17 included RCTs, PN groups ranged from 4 to 233 participants and control groups from 5 to 177 participants, with mean ages of  $48.2 \pm 9.5$  and  $49.1 \pm 9.3$  years respectively. Intervention durations spanned 8 [21] to 52 [26] weeks, with most trials running between 12 and 24 weeks. The included interventions were predominantly RCTs with parallel-group designs, ranging from open-label pilot

or exploratory studies [18, 27] to larger multicenter or double-blind RCTs [25, 26]. Study populations consisted of adults with overweight or obesity, older adults, or individuals with metabolic risk conditions such as pre-diabetes or early type 2 diabetes [28, 29], non-diabetic hyperglycaemia [27], and NAFLD [24]. Trials were conducted across geographically diverse settings, including the USA, Europe, Canada, South Korea, China, Mexico, India, Israel, and the Philippines.

### Objective 1: Individual-level features used for personalization

Overall, although none of the seventeen studies provided their own definition of PN, all identified individual-level features used to guide personalized dietary or lifestyle recommendations. Supplementary S2 shows that these features were categorized into eight distinct domains, as outlined below.

#### Biological and clinical features

Across the 17 RCTs, a wide range of biological and clinical features were used to generate PN recommendations. Blood lipid biomarkers, including triglycerides, LDL cholesterol, HDL cholesterol, and total cholesterol, were the most frequently used and were reported in seven trials [17, 19–22, 28, 30]. Fasting glycemic markers, such as fasting glucose, insulin, and HbA1c, appeared in 6 trials [19, 20, 22, 28–30], where they served as indicators of metabolic status or guided dietary recommendations. Inflammatory and hepatic markers, including CRP, IL-6, TNF- $\alpha$ , and liver enzymes, were used in three trials [21, 24, 25], particularly those targeting lipid metabolism or NAFLD-related risk. Lastly, blood pressure was included in two trials [17, 26] as a marker of metabolic dysfunction.

#### Genetic features

Genotype-based personalization was a major component of several trials. In six studies, dietary recommendations were based exclusively on genetic variants [16, 21, 23, 24, 27, 31]. These studies used single nucleotide polymorphisms (SNPs) related to obesity risk (e.g., FTO, UCP1), lipid metabolism (e.g., LIPC, APOA5, APOC3), inflammation (e.g., TNF- $\alpha$ ), and metabolic regulation (e.g., PPARG, ADIPOQ). An additional five studies implemented multi-gene personalization approaches that combined genetic profiles with anthropometric or biochemical characteristics [17, 19, 20, 22, 25]. In all genotype-based interventions, SNPs informed tailored macronutrient distribution, nutrient targets, or categorization into nutrigenetic diet profiles.

#### Microbiome features

Four studies incorporated gut microbiome data, either stool-derived microbial abundance measures or estimates of microbial functional modules, into their personalization frameworks [25, 28–30]. In these studies, microbial profiles were integrated with additional data layers, including blood biomarkers, dietary intake, and CGM-derived glycemic responses, to refine dietary recommendations based on microbial associations with macronutrients, food types, or predicted metabolic responses.

#### Metabolomic features

Two trials incorporated targeted metabolomic profiling as personalization inputs. Study [25] centered its personalization framework on plasma and urine metabolomics panels, including amino acids, acylcarnitines, phospholipids, oxidative stress markers, and inflammatory metabolites. Study [30] included selected metabolic markers, such as postprandial triglycerides and related cardiometabolic indicators, to support dietary decision-making.

#### Postprandial response measures

Postprandial glycemic responses, assessed via continuous glucose monitoring (CGM), were applied in four trials [15, 18, 26, 28]. In one of these, CGM data were used exclusively to generate personalized dietary recommendations [15]. The other three combined postprandial glucose patterns with anthropometric, physical activity, microbiome, or dietary features to produce individualized dietary scores or meal rankings [18, 26, 28].

#### Anthropometric features

Seven studies [17–20, 22, 28, 29] used anthropometric measures, including BMI, body weight, waist circumference, and body fat percentage, as inputs for personalization. These features were commonly integrated with dietary, genetic, or biochemical data to assign individualized diet types or energy targets.

#### Demographic feature

Age was incorporated into the personalization model in one study [19], whereas in the remaining trials, demographic features such as age, gender, and educational level were recorded as baseline characteristics but not used within the personalization frameworks.

#### Lifestyle and behavioral features

Dietary variables, including dietary intake patterns, food frequency estimates, and 24-hour diet records, were used to guide or refine personalization in seven studies [18–20, 22, 28–31]. Physical activity was incorporated as a personalization input in three of these studies, with accelerometry or step-count data contributing to individualized recommendations [18, 26, 29]. One study uniquely employed self-monitoring of diet and physical activity to support participant-led behavioral adjustments targeting postprandial glucose control [18].

### Objective 2: Variation in control diets versus personalized intervention interventions

As described in Supplementary S3, the control conditions did not include any personalized dietary components. However, there was variation in the specific diets used across the 17 trials, including low-fat diets [16, 21, 24, 29], balanced diets [22, 23, 25, 27], Mediterranean diets

[28], generic national healthy-eating advice [30, 31], and standard weight management advice [18–20, 26] or no received dietary counseling [15, 17]. Collectively, six out of the 17 control diets followed specific dietary patterns.

In contrast, the intervention groups employed personalization methods that relied on individual-level biological, metabolic, behavioral, or sensor-derived data. These included postprandial glycemic response (PPGR)-based personalized diets [28, 29], multi-omics approaches incorporating microbiome and/or metabolomic profiles [25, 30], strictly genotype-based nutrigenetic diets [16, 21, 23, 24, 27, 31], genotype combined with biomarker information [17, 20, 22], CGM-guided dietary adjustments [15, 18], genetics plus additional biological-factor personalization [19], and AI-driven sensor-based dietary optimization [26].

### **Objective 3: Heterogeneity in delivery modes, contact intensity, and generators of personalized diets**

As shown in Table S4, the 17 trials exhibited substantial heterogeneity in both delivery modes and the intensity of participant contact across control and intervention arms. Control conditions were delivered through multiple formats, including digital platforms such as online counseling [20, 29], email or web-based communication [30, 31], mobile apps [17, 18], and messaging tools like WeChat [22]. Printed or static materials were used in the form of leaflets, standard diet menus, or nutrition education handouts [15, 19, 21, 24]. In-person formats included group classes, face-to-face counseling, and verbal consultations with dietitians [15, 16, 23, 27, 28]. A few trials employed hybrid or enhanced approaches, combining digital tools (e.g., smart scales, Fitbits, app-based recipes) with individualized coaching, weekly messages, or meal delivery services [18, 25, 26].

Intervention delivery showed similar diversity and typically mirrored the control format while adding personalized-nutrition components. Formats included online or app-based platforms [17, 19–22, 26, 27, 29–31], face-to-face or hybrid counseling [15–18, 23, 28], and the integration of personalized tools such as genetic feedback [16], CGM devices [15, 18, 28], algorithm-based recommendations [28], and app-linked meal kits or hard-copy materials [25, 27].

The frequency of participant contact varied substantially across studies for both control and PN groups. In several trials, contact intensity was identical between arms, such as in studies [16, 20–25, 28, 29], where intervention and control groups followed matched schedules (e.g., weekly, biweekly, or predefined sessions). In contrast, studies [15, 17–19, 26, 31] reported increased frequency or personalization in the intervention arms, often involving weekly counseling, phone sessions, feedback reports, or technology-enabled follow-up. In a few

studies [17, 19, 27, 30], contact frequency in control group was not clearly detailed, limiting comparability. Notably, studies [15] and [26] demonstrated the most intensive intervention contact, with multimodal weekly interactions that combined phone, face-to-face, or digital coaching elements.

Across the trials, the generators of personalized dietary prescriptions also varied considerably. Some interventions relied on algorithm-generated personalized diets, such as machine-learning PPGR models, multi-omics or metabolomics algorithms, or artificial intelligence-based digital-twin systems [25, 26, 28–31]. Others used dietitian-generated personalized diets, derived from genotype reports or from individual clinical, biological, or glycemic-response profiles [15–17, 19–24, 27]. A single study implemented participant-selected personalized diets, in which participants used CGM feedback alongside coaching to choose their own individualized dietary patterns [18].

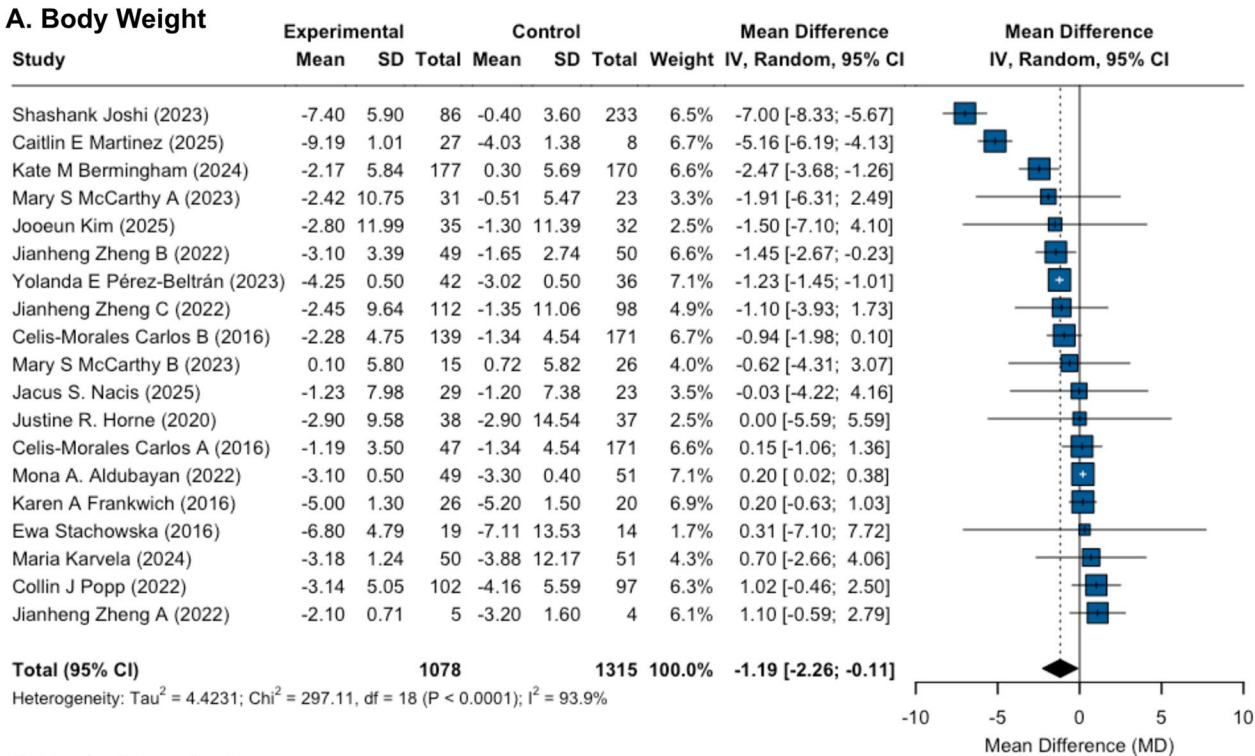
### **Objective 4: Effectiveness of personalized nutrition on body weight and BMI**

Across the included trials, 19 effect sizes contributed to the meta-analysis of body weight. PN produced a small but statistically significant reduction in body weight compared with control diets (MD = − 1.19 kg, 95% CI − 2.26 to − 0.11), with high heterogeneity ( $I^2 = 93.9\%$ ; Fig. 2A). Leave-one-out sensitivity analysis showed that the pooled effect was generally stable; however, omitting three studies [18, 26, 30] shifted the confidence interval to non-significance (Supplementary S5.A). Bayesian meta-regression indicated that longer intervention duration was credibly associated with greater weight loss (−0.08 kg/week; 95% CrI − 0.10 to − 0.06; Supplementary S6.A). For BMI, 14 effect sizes were analyzed, yielding a non-significant pooled effect (MD = − 0.32 kg/m<sup>2</sup>, 95% CI − 0.85 to 0.21;  $I^2 = 89.7\%$ ; Fig. 2B), and all leave-one-out iterations remained non-significant (Supplementary S5.B). However, Bayesian meta-regression showed a credible association between longer duration and greater BMI reduction (−0.04 kg/m<sup>2</sup> per week; 95% CrI − 0.05 to − 0.03; Supplementary S6.B). Egger's regression tests found no evidence of funnel-plot asymmetry for either body weight ( $p = 0.276$ ) or BMI ( $p = 0.426$ ), indicating no detectable small-study publication bias (Supplementary S7).

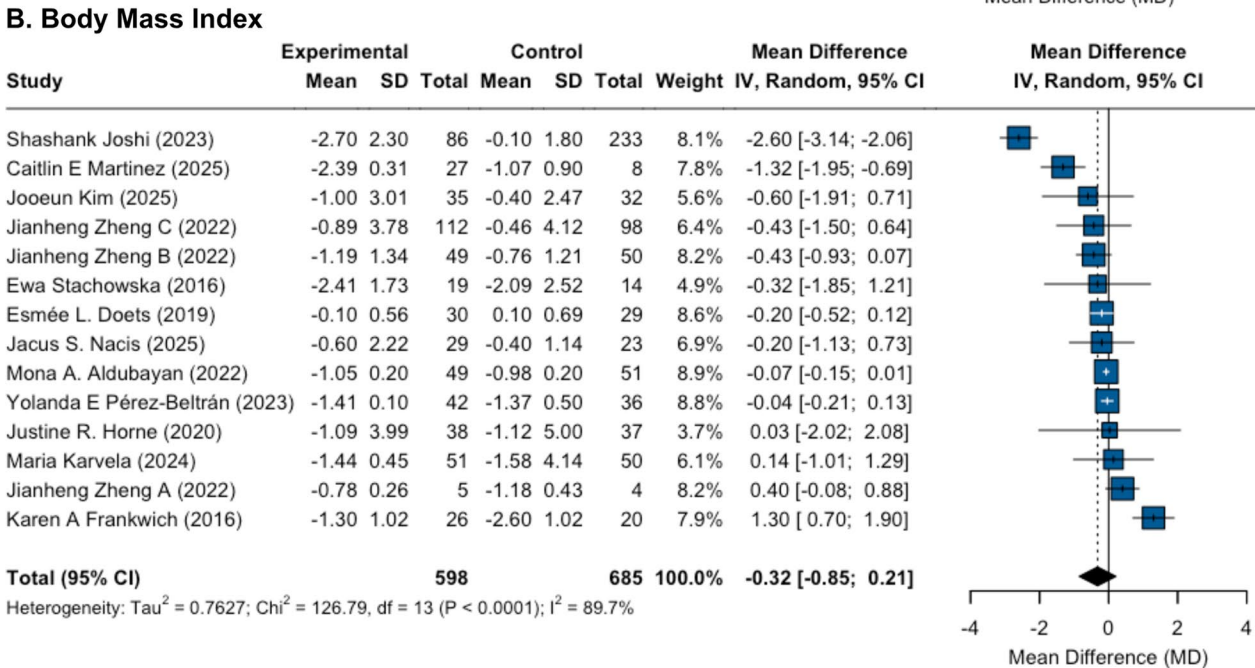
### **GRADE assessment**

Supplementary S8 shows that evidence certainty for both body weight and BMI outcomes was downgraded from high due to considerable heterogeneity and imprecision. For body weight, the final certainty was low, as sensitivity analysis revealed loss of significance when three studies were removed. For BMI, the evidence was rated very low

A. Body Weight



B. Body Mass Index



**Fig. 2** Forest plots displaying the effect of personalized nutrition in overweight and obesity adults on (A) body weight (kg), and (B) body mass index (kg/m<sup>2</sup>). Abbreviations: CI, confidence interval; MD, mean difference; SD, standard deviation; Total, total number of participants who completed the study

due to non-significant effects and high between-study variability.

## Dicussion

Across 17 RCTs in adults with overweight and obesity, we found that PN is currently implemented using highly heterogeneous features, comparators, and delivery strategies, yet yields relatively modest and uncertain effects on weight-related outcomes. At the level of personalization features (Objective 1), interventions drew on diverse biological, genetic, microbiome, metabolomic, anthropometric, demographic, and behavioral data, ranging from single-domain genotype or CGM-guided approaches to complex multi-omics platforms. Across trials (Objective 2), control diets were non-personalized but still adhered to established healthy-eating guidelines, such as low-fat diets, balanced or Mediterranean-style diets, or standard behavioral weight-loss advice. Thus, PN interventions were evaluated against active, guideline-based comparators rather than against minimal or no dietary counseling. Implementation characteristics (Objective 3) also varied widely: interventions used face-to-face counseling, web platforms, smartphone apps, and connected devices, with contact intensity spanning frequent weekly or biweekly interactions to sparse or poorly specified follow-up. Personalized prescriptions were generated by different “engines,” including machine-learning and multi-omics algorithms, dietitians applying genotype or biomarker reports, and, in one case, participant self-selection using CGM feedback. In meta-analysis (Objective 4), the diverse methodologies applied in PN interventions produced a modest reduction in body weight (−1.19 kg vs. control), whereas the effect on BMI was not statistically significant. Both outcomes showed high between-study heterogeneity, and the weight effect was sensitive to exclusion of several trials. Overall, these findings suggest that while PN is feasible to implement and can integrate a wide range of personalized features, its incremental benefit for weight loss beyond well-structured standard dietary interventions appears modest.

The personalized features employed across trials were selected based on their presumed relevance for capturing person-specific variation in metabolic or lifestyle patterns. First, several interventions used postprandial glycemic measures or CGM-derived glucose profiles based on evidence that glycemic excursions contribute to insulin resistance and ectopic fat accumulation, and that individuals exhibit large, partly microbiome- and phenotype-dependent differences in postprandial glucose responses to identical foods [15, 18, 26, 28, 30]. Second, genotype-based interventions targeted variants in genes related to obesity risk, insulin secretion, or lipid metabolism (e.g., *FTO*, *TCF7L2*, *PPARG*, *APOE*, *APOA5*, *APOC3*, *TNF-α*). These targets reflect evidence

that such loci modulate susceptibility to weight gain, insulin resistance or dyslipidemia and may interact with dietary fat or carbohydrate intake [16, 17, 20–24, 27, 31]. In NAFLD-focused and lipid-focused trials, multi-gene panels such as the nutrient-induced insulin output ratio or weighted genetic risk scores were explicitly designed to capture “fat-sensitive” or “glucose-sensitive” phenotypes and guide macronutrient distribution (e.g., lower-fat or lower-carbohydrate patterns) to improve hepatic steatosis and dyslipidemia [21, 24]. Third, microbiome- and metabolomics-guided personalization drew on specific biological signals reported in the included trials. Specifically, stool-derived microbial abundances were associated with postprandial glycemia and macronutrient handling [22, 25, 28–30]. Building on these signals, targeted metabolomic biomarkers, including amino acids, acylcarnitines, phospholipids, sphingolipids, oxidative stress-related metabolites, and postprandial triglyceride responses, were used to classify individuals into metabolic clusters, or “metabotypes,” reflecting lipid- and glucose-handling phenotypes [25, 30]. Finally, several trials incorporated behavioral or technology-enabled personalization using mHealth tools, self-experimentation, or digital twins to align dietary prescriptions with individual behavioral patterns, daily routines, and predicted postprandial responses. These approaches aimed to improve adherence while simultaneously addressing multiple lifestyle domains, including diet, physical activity, and sleep [17, 18, 26, 27].

Several factors may help explain why PN interventions have generally produced modest reductions in body weight and BMI. First, PN trials delivered multiple layers of advice (dietary quality, physical activity, behavior change, and in some cases genetic information), meaning that the influence of any single personalization features (e.g., genotype-based tailoring) was likely diluted within broader lifestyle guidance [31]. Second, many biological inputs used for personalization, such as SNPs, microbial taxa, or metabolomic signatures, explain only a small proportion of variation in body weight. To date, identified hereditary factors explain only about 5% of BMI variability [32]; polygenic risk scores increase the explained proportion to roughly 17.6% [33], but substantial variability remains unaccounted for. Additionally, the gut microbiome’s contribution to obesity remains unclear [34], while body weight is defended by physiological mechanisms such as compensatory metabolic adaptation [33] and strongly shaped by environmental drivers of energy balance [35]. As a result, tailoring diets based on limited biological features may be insufficient to overcome these dominant regulatory forces. Third, several trials reported limited or inconsistent adherence, which constrained the ability of personalized interventions to translate dietary prescriptions into sustained energy deficits. For example,

in the MyGeneMyDiet study [20], modest differences in dietary intake were insufficient to produce meaningful differences in anthropometric outcomes, and low adherence, partly due to COVID-19–related disruptions, was explicitly cited as a contributor to minimal weight change [29]. Fourth, several PN algorithms were developed with the primary aim of optimizing postprandial glycemic and lipid responses, rather than directly targeting energy balance. Consequently, investigators have suggested that achieving meaningful weight loss may require more comprehensive models that incorporate additional physiological domains, such as appetite-regulating hormones, energy expenditure, and fat mass regulation, which are not addressed by current PN algorithms [29]. Finally, in several trials, the control arms were not minimal interventions but included structured dietary guidance, regular contact, or digital support [25, 28, 31]. As a result, improvements in both groups may have attenuated the incremental effect of PN on anthropometric outcomes. Collectively, these findings reflect the complex barriers to achieving effective weight outcomes through biological personalization, emphasizing the need for more precise algorithms, enhanced adherence support, and better-controlled comparators.

A strength of this review is the inclusion of only RCTs of PN in adults with overweight or obesity, which enhances the robustness of causal interpretation. The systematic and detailed characterization of intervention methodologies provided clear insights into how PN approaches have been designed and implemented across trials. In addition, the use of a clear, a priori definition of PN ensured consistency in study selection and enabled meaningful comparisons across intervention designs.

This review has several limitations. First, the meta-analysis findings should be interpreted cautiously because between-study heterogeneity was high and the pooled effect on body weight was sensitive to the exclusion of some trials. Second, although we applied a clear definition of PN to guide study selection, variability in how personalization is described in the literature may have led to the exclusion of some relevant interventions. Third, our characterization of intervention methods depended on the level of detail reported in each trial; inconsistent reporting may have produced incomplete descriptions of intervention components and possible misclassification of personalization features. Fourth, substantial heterogeneity in study designs and PN approaches meant we could describe patterns across trials but could not quantitatively isolate which specific personalization features drove anthropometric outcomes, limiting mechanistic interpretation. Finally, several studies did not fully report the algorithms, decision rules, or weighting schemes used to generate personalized prescriptions, which

constrained our ability to classify personalization mechanisms precisely.

## Conclusion

To our knowledge, this is the first review to systematically characterize how PN has been operationalized in RCTs involving adults with overweight or obesity. We found that current PN interventions draw on diverse biological, behavioral, and technological inputs; however, their effects on body weight and BMI remain modest compared to diverse dietary control groups. Variation in personalization methods, delivery modes, and intervention intensity likely contributes to the heterogeneity observed in outcomes. This review highlights key design features that can inform the development of more effective PN interventions moving forward. Continued progress will require clearer definitions of PN, identification of personalization features that meaningfully influence metabolic regulation, and greater transparency and reproducibility in the algorithms used to generate personalized recommendations. Moreover, to more accurately estimate the incremental value of personalization, future trials should use control conditions matched not only for delivery mode and contact intensity but also for core dietary features such as macronutrient composition, energy prescription, and behavioral support.

## Abbreviations

|       |                                                                      |
|-------|----------------------------------------------------------------------|
| BMI   | Body Mass Index                                                      |
| CI    | Confidence Interval                                                  |
| CVD   | Cardiovascular disease                                               |
| CGM   | Continuous Glucose Monitoring                                        |
| FTO   | Fat Mass And Obesity-Associated                                      |
| GRADE | Grading of Recommendations, Assessment, Development, and Evaluations |
| MD    | Mean Difference                                                      |
| ML    | Machine Learning                                                     |
| PICOS | Participants, Interventions, Control, Outcomes, Study Design         |
| NAFLD | Non-alcoholic fatty liver disease                                    |
| PPGR  | Postprandial Glycemic Response                                       |
| PN    | Personalized Nutrition                                               |
| SNPs  | Single Nucleotide Polymorphisms                                      |
| RCTs  | Randomized Controlled Trials                                         |
| ROS   | Reactive Oxygen Species                                              |
| SD    | Standard Deviation                                                   |

## Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12263-026-00800-4>.

Supplementary Material 1

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## Author contributions

TQD: Conceptualization, Methodology.-TQD and NDK: Data curation, Writing-Original draft preparation.-TQD and VTQC: Visualization, Investigation.-TQD: Supervision.-TQD, DKD and NTH: Software, Validation.-All authors reviewed the results and approved the final version of the manuscript.

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## Data availability

All data generated or analysed during this study are included in this published article and its Supplementary materials.

## Declarations

### Ethics approval and consent to participate

Not applicable.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

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