NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

BEREK, Maria, KMIECIAK, Kacper, BORYCKA, Klaudia, STAŃSKA, Jagoda, BARANOWSKA, Aleksandra, DOBRZAŃSKA, Aleksandra, HAJDUK, Anna, SZYMAŃSKI, Piotr, SZYMAŃSKA, Aleksandra and KOSTANEK, Filip. Non-nutritive sweeteners and exercise in obesity: potential metabolic interactions and implications for exercise tolerance. Quality in Sport. 2026;74:75275. https://doi.org/10.12775/QS.2026.74.75275

ARTICLE TIMELINE
Received: 12.08.2026. Accepted: 11.09.2026. Published: 21.09.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Non-nutritive sweeteners and exercise in obesity: potential metabolic interactions and implications for exercise tolerance

Maria Berek

ORCID: https://orcid.org/0009-0006-2613-1123

mariaberek12@gmail.com

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz.

ul. Pomorska 251, 92-213 Łódź, Poland

Kacper Kmieciak

ORCID: https://orcid.org/0009-0007-2703-2538

kacper.kmieciak@o2.pl

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz,

ul. Pomorska 251, 92-213 Łódź, Poland

Klaudia Borycka

ORCID: https://orcid.org/0009-0006-0006-7813

k.borycka@interia.pl

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz,

ul. Pomorska 251, 92-213 Łódź, Poland

Jagoda Stańska

ORCID: https://orcid.org/0009-0000-4861-2856

lek.dent.jagoda.stanska@gmail.com

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz,

ul. Pomorska 251, 92-213 Łódź, Poland

Aleksandra Baranowska

ORCID: https://orcid.org/0009-0003-0373-7577

olabaranowska200@gmail.com

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz,

ul. Pomorska 251, 92-213 Łódź, Poland

Aleksandra Dobrzańska

ORCID: https://orcid.org/0009-0002-4694-793X

aleksandra.dobrzanska89@gmail.com

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz,

ul. Pomorska 251, 92-213 Łódź, Poland

Anna Hajduk

ORCID: https://orcid.org/0009-0003-1570-0608

hajduk.ania2001@gmail.com

Independent Researcher, Lodz, Poland

Piotr Szymański

ORCID: https://orcid.org/0009-0006-2891-640X

peterson2001@tlen.pl

Independent Researcher, Lodz, Poland

Aleksandra Szymańska

ORCID: https://orcid.org/0009-0002-2122-7522

ola.szymanska9000@tlen.pl

Independent Researcher, Lodz, Poland

Filip Kostanek

ORCID: https://orcid.org/0009-0003-0243-5219

filipkostanek@gmail.com

Independent Researcher, Lodz, Poland

Corresponding Author

Maria Berek: mariaberek12@gmail.com

Abstract

Introduction and purpose: Non-nutritive sweeteners (NNS) are used as alternatives to caloric sugars, but their metabolic effects vary according to the sweetener and exposure conditions. Physical exercise is a core component of obesity management, while evidence on interactions between NNS consumption and exercise remains insufficient. This narrative review aimed to evaluate NNS–exercise interactions in adults with obesity.

Material and methods: A literature search was conducted in PubMed and Scopus using terms related to NNS, exercise, obesity and metabolic responses. Articles published since 2012 were considered, including clinical and experimental studies, systematic reviews and meta-analyses.

Results: NNS-related metabolic responses vary across sweeteners and experimental conditions. Sucralose may produce different insulin responses according to metabolic phenotype. Exercise training improves glucose and insulin regulation, whereas few studies have directly examined NNS consumption during exercise. Most evidence derives from healthy or physically active populations, limiting its applicability to obesity.

Conclusions: Current evidence provides a biological rationale for NNS–exercise interactions but is insufficient to determine whether NNS consumption modifies exercise tolerance, performance or exercise-induced metabolic adaptations in adults with obesity. Well-controlled

studies examining NNS type, dose, timing, carbohydrate co-ingestion and metabolic phenotype are needed.

Keywords: “non-nutritive sweeteners”, “artificial sweeteners”, “exercise”, “physical activity”, “obesity”, “metabolic response”

1. Introduction

Obesity is a chronic, relapsing disease characterized by abnormal or excessive accumulation of body fat that may impair health (World Health Organization, 2025). In adults, obesity is commonly classified using body mass index (BMI), calculated as body weight in kilograms divided by height in metres squared (kg/m^2), with obesity defined as a BMI of ≥ 30 kg/m^2 and overweight as a BMI of 25-29.9 kg/m^2. Although BMI is a useful population-level measure, it is a surrogate marker of adiposity, and additional measures such as waist circumference may provide further information on obesity and associated health risks (World Health Organization, 2025). Obesity represents a major global public health challenge and is strongly associated with metabolic disorders, including impaired insulin-mediated glucose regulation, type 2 diabetes mellitus and cardiovascular disease (Choo et al., 2026). Excess adiposity is accompanied by metabolic disturbances, chronic low-grade inflammation and impaired insulin signaling, which may contribute to reduced metabolic flexibility and impaired glucose homeostasis (Choo et al., 2026). Regular physical activity and structured exercise training are therefore central components of obesity management and can improve metabolic health, insulin sensitivity and cardiorespiratory fitness, including in the absence of substantial weight loss (Oppert et al., 2021; Khalafi et al., 2023).

Non-nutritive sweeteners (NNS) have been widely introduced as alternatives to caloric sugars with the aim of reducing energy and free sugar intake. Their potential role in weight management remains controversial. Randomized controlled trials have generally shown neutral or modestly beneficial effects of NNS on body weight and BMI, particularly when NNS replace caloric sweeteners, whereas observational studies have reported associations between NNS consumption and adverse cardiometabolic outcomes (Laviada-Molina et al., 2020; Buszek et al., 2025, Li et al., 2026, Azad et al., 2017; Toews et al., 2019). These apparently divergent findings may partly reflect differences in study design, exposure patterns, dietary context and the characteristics of NNS consumers. Consequently, associations observed in observational studies should not necessarily be interpreted as evidence of a causal adverse effect of NNS.

The metabolic effects of NNS may vary according to the specific sweetener and the context of exposure, including the metabolic characteristics of the individual. Experimental and clinical evidence suggests that NNS are associated with changes in glucose homeostasis, endocrine signaling and other metabolic processes, although findings have varied across individual studies (Pang et al., 2021; Moriconi et al., 2020; Nobs and Elinav, 2023). A systematic review of human studies has additionally suggested that some non- and low-calorie sweeteners may influence substrate oxidation, although the magnitude and consistency of these effects vary across experimental conditions (Andersen et al., 2023). Metabolic effects may differ according to adiposity. In a randomized crossover study, sucralose ingestion increased postprandial glucose excursions in both normal-weight individuals and individuals with obesity, while the insulin response differed between the two groups (Nichol et al., 2020). Such findings suggest that obesity-associated metabolic dysfunction may modify the physiological response to NNS exposure.

NNS have also been investigated in the context of physical exercise, although relatively few studies have directly examined their effects during exercise. Studies of NNS-containing mouth rinses have primarily examined sensory and ergogenic responses rather than systemic metabolic effects. In a randomized running trial, sucrose mouth rinsing improved endurance performance compared with water, whereas sucralose mouth rinsing did not produce a comparable improvement; lower-intensity sucralose rinsing nevertheless reduced the maximum rating of perceived exertion (Hawkins et al., 2017). More directly, a small randomized crossover study found that co-ingestion of aspartame with carbohydrate during endurance exercise altered the insulin response compared with carbohydrate alone (Siegler et al., 2012). Both studies involved healthy, physically active participants and therefore did not specifically represent populations with obesity.

Despite increasing interest in the metabolic effects of NNS and the established role of exercise in obesity management, the interaction between these two factors remains insufficiently characterized. Current evidence indicates that NNS can affect metabolic pathways associated with exercise physiology, including glucose and insulin regulation and substrate utilization, although the magnitude and direction of these effects vary across studies. Obesity itself is associated with impaired insulin action, altered substrate metabolism and reduced exercise capacity (Choo et al., 2026; Khalafi et al., 2023; Andersen et al., 2023). Because direct studies combining NNS exposure, physical exercise and obesity are scarce, evidence from studies examining NNS-related metabolic effects or exercise responses separately is also considered to provide the physiological context for evaluating this potential interaction. This represents an important research gap, as the metabolic effects of NNS may depend on the timing of exposure, carbohydrate availability, the type of sweetener and the baseline metabolic phenotype of the individual.

Therefore, this narrative review aims to critically evaluate the current evidence regarding potential metabolic interactions between non-nutritive sweetener (NNS) consumption and physical exercise in the context of obesity. Particular attention is given to glucose and insulin regulation, substrate utilization, inflammatory and metabolic pathways, exercise performance and tolerance. The review also considers whether the available evidence is sufficient to determine a direct relationship between NNS consumption and exercise tolerance in individuals with obesity.

2. Materials and methods

A literature search was conducted in electronic databases, including PubMed and Scopus, using the following keywords: “non-nutritive sweeteners”, “artificial sweeteners”, “exercise”, “physical activity”, “obesity”, “exercise tolerance”, “metabolic response”, “glucose”, “insulin”, and “substrate oxidation”. Articles published since 2012 were considered, including randomized controlled trials, systematic reviews, meta-analyses, narrative reviews, and other relevant human and experimental studies. Additional publications were identified by screening the reference lists of selected articles.

3. Non-nutritive sweeteners: classification and biological mechanisms

3.1. Classification and metabolic fate

Non-nutritive sweeteners (NNS), also referred to as non-sugar sweeteners or low- and no-calorie sweeteners, are a diverse group of compounds that provide a sweet taste with little or no energy contribution. They include aspartame, sucralose, saccharin, acesulfame potassium, cyclamate, neotame, advantame and steviol glycosides (Sylvetsky and Rother, 2018; Teysseire et al., 2024, Agopsowicz et al., 2025). Low-calorie sweetening compounds also include sugar alcohols such as erythritol, which differ from high-intensity NNS in their chemical structure and metabolic fate. Erythritol is a four-carbon polyol that is largely absorbed in the small intestine and excreted unchanged, resulting in minimal systemic metabolism and a negligible effect on blood glucose (Skowrońska-Borsuk et al., 2025). These differences in chemical structure and metabolic fate warrant consideration when interpreting studies investigating the metabolic effects of NNS. Aspartame, for example, is hydrolysed in the gastrointestinal tract into its constituent components, whereas sucralose is only partially absorbed and is predominantly excreted without extensive metabolism. Other NNS, such as acesulfame potassium, are absorbed and subsequently eliminated largely unchanged (Sylvetsky and Rother, 2018; Moriconi et al., 2020). Consequently, physiological responses observed with one NNS cannot necessarily be generalized to other compounds within the same category. The metabolic fate of an individual sweetener may therefore represent an important determinant of its potential effects on glucose regulation, endocrine signaling and substrate metabolism. In addition to the specific compound, factors such as dose, duration of exposure, gastrointestinal exposure and the presence of other dietary components may further influence the observed response (Agopsowicz et al., 2025, Moriconi et al., 2020; Teysseire et al., 2024).

3.2. Glucose and insulin regulation

The acute effects of NNS on glucose and insulin metabolism appear to depend on the type of sweetener, timing of administration, carbohydrate co-ingestion and metabolic characteristics of the individual. A systematic review and network meta-analysis found that NNS-sweetened beverages generally did not produce significant acute changes in postprandial glucose or endocrine responses compared with water or unsweetened beverages (Zhang et al., 2023; Agopsowicz et al., 2025). Individual studies, however, have reported heterogeneous responses. For example, sucralose ingestion before an oral glucose tolerance test altered insulin responses, with differences observed according to baseline adiposity (Nichol et al., 2020, Agopsowicz et al., 2025). Overall, current evidence does not support a uniform adverse effect of NNS on glucose homeostasis but suggests that metabolic responses may be context-dependent (Pang et al., 2021; Nobs and Elinav, 2023).

3.3. Sweet taste receptors and metabolic signaling

One proposed mechanism underlying the metabolic effects of NNS involves activation of sweet taste receptors. The heterodimeric G-protein-coupled receptor TAS1R2/TAS1R3 is responsible for the perception of sweetness in the oral cavity and has also been identified in extra-oral tissues, including the gastrointestinal tract (Moriconi et al., 2020; Sylvetsky and Rother, 2018). Experimental evidence suggests that activation of extra-oral sweet taste receptors is involved in enteroendocrine and metabolic signaling, including pathways involving incretin hormones such as glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) (Moriconi et al., 2020; Sylvetsky and Rother, 2018; Herzog et al., 2025).

Human studies have not consistently demonstrated substantial metabolic or endocrine responses following isolated NNS exposure (Moriconi et al., 2020; Herzog et al., 2025). The metabolic response may also depend on whether NNS are consumed alone or together with other dietary components (Sylvetsky and Rother, 2018; Moriconi et al., 2020). This distinction is important because mechanistic findings obtained in experimental models cannot automatically be extrapolated to physiological effects in humans (Herzog et al., 2025). The presence of sweet taste receptors in extra-oral tissues therefore provides biological plausibility for metabolic effects of NNS but does not establish that receptor activation results in clinically meaningful changes in glucose or insulin regulation (Herzog et al., 2025; Moriconi et al., 2020).

Whether obesity modifies these pathways or alters the response to NNS during physical exercise remains insufficiently investigated. This question merits further investigation because obesity is associated with impaired insulin signaling and reduced metabolic flexibility, potentially creating a distinct physiological context in which NNS exposure occurs (Choo et al., 2026; Oppert et al., 2021).

3.4. Gut microbiota and metabolic signaling

The gut microbiota is another potential pathway linking NNS exposure with metabolic regulation. Experimental and clinical evidence indicates that some NNS can alter microbial composition and metabolite production, although responses vary according to the sweetener, dose, duration of exposure and baseline microbiota (Suez et al., 2014; Conz et al., 2023; Crakes et al., 2025). In the SWEET randomized controlled trial, adults with overweight or obesity consuming sweeteners and sweetness enhancers within a healthy, sugar-reduced diet maintained greater weight loss over one year and showed changes in gut microbiota composition compared with the sugar-control group (Pang et al., 2025). These findings further support the context-dependent nature of NNS effects.

3.5. Potential relevance to exercise metabolism

The metabolic pathways potentially influenced by NNS, including glucose and insulin regulation and substrate oxidation, contribute to the metabolic response to physical exercise. A systematic review of human studies found that NNS were associated with changes in carbohydrate and fat oxidation under some experimental conditions, although findings varied across experimental conditions and the clinical significance of these acute responses has not been clearly established (Andersen et al., 2023). In the setting of obesity, impaired insulin action and altered substrate metabolism can affect the metabolic response to exercise, providing a biological rationale for investigating potential NNS-exercise interactions (Oppert et al., 2021; Khalafi et al., 2023). Direct evidence examining the relationship between NNS consumption and exercise responses remains scarce, with few studies specifically addressing populations affected by obesity. These observations provide a biological rationale for further investigation but do not demonstrate a direct effect on exercise performance or tolerance.

4. Metabolic responses to physical activity in individuals with obesity

4.1. Metabolic responses to exercise

Obesity is associated with impaired glucose homeostasis, insulin resistance and alterations in substrate metabolism that may contribute to differences in the physiological response to physical exercise. Excess adiposity, with a prominent contribution from visceral adiposity, is associated with increased fatty acid availability and impaired insulin-mediated glucose disposal, contributing to metabolic dysfunction and reduced metabolic flexibility (Choo et al., 2026). Physical activity is therefore integral to obesity management because it can improve glucose regulation and enhance insulin-mediated glucose uptake independently of substantial weight loss (Oppert et al., 2021).

Exercise training has a well-established beneficial effect on postprandial glucose and insulin responses in individuals with overweight or obesity. A systematic review and meta-analysis including 29 studies and 1,401 participants demonstrated that exercise training significantly reduced postprandial glycemia and insulinemia, with benefits observed following both aerobic and resistance training (Khalafi et al., 2023). The metabolic response may also vary according to the type of exercise performed, with different forms of structured exercise producing beneficial effects on glucose regulation (Oppert et al., 2021; Khalafi et al., 2023; Kang et al., 2023). These adaptations are particularly relevant in obesity, where impaired insulin sensitivity and altered substrate metabolism may limit the ability to appropriately regulate glucose and lipid utilization in response to changing energy demands.

The metabolic response to exercise is influenced by exercise modality, intensity and duration. Aerobic exercise increases energy expenditure and requires coordinated utilization of carbohydrate and lipid substrates, whereas strength-oriented exercise promotes increases in skeletal muscle mass and may consequently increase the amount of metabolically active tissue available for glucose disposal. Repeated exercise training can therefore improve insulin-mediated glucose uptake and metabolic regulation through adaptations in skeletal muscle and whole-body energy metabolism (Oppert et al., 2021; Khalafi et al., 2023).

These adaptations warrant consideration in obesity, where impaired insulin-mediated glucose regulation may compromise the appropriate adjustment of substrate utilization to changing energy demands. In this context, the relationship between NNS and substrate metabolism represents a potential point of interaction with exercise. A systematic review of human studies reported heterogeneous effects of non- and low-calorie sweeteners on substrate oxidation, energy expenditure and catecholamine responses, although the clinical relevance of these acute findings remains unclear (Andersen et al., 2023). AAltered substrate utilization may therefore represent a potential pathway through which dietary exposure to NNS could interact with exercise metabolism, although direct evidence in adults with obesity remains limited.

Obesity is characterized by chronic low-grade inflammation and metabolic disturbances that contribute to impaired insulin action and disrupted glucose regulation. Excess adipose tissue, including visceral adipose tissue, is metabolically active and associated with an altered inflammatory environment that may interfere with normal metabolic signaling (Choo et al., 2026; Walbolt and Koh, 2020).

Regular physical activity can modify this metabolic environment and may contribute to reductions in systemic inflammatory activity. However, the relationship between exercise and inflammation should not be interpreted as uniformly anti-inflammatory. Acute exercise represents a physiological stressor and may transiently alter inflammatory signaling, whereas repeated exercise training may promote longer-term adaptations associated with improved metabolic regulation. This distinction is important in obesity, where baseline metabolic and inflammatory disturbances may modify the response to exercise (Oppert et al., 2021).

Oxidative stress represents another component of the metabolic environment associated with obesity. Exercise itself increases metabolic demand and may transiently increase oxidative stress, whereas repeated exercise can induce adaptive responses that improve the capacity of tissues to respond to oxidative challenges. The magnitude and direction of these responses depend on exercise characteristics and individual metabolic status. Oxidative stress should therefore be considered as part of the broader metabolic context of exercise in obesity rather than as an isolated mechanism.

4.2. Exercise tolerance and physical fitness limitations

Obesity may be associated with reduced exercise capacity and cardiorespiratory fitness, as well as limitations in mobility and exercise participation related to excess body mass, musculoskeletal conditions and comorbidities (Oppert et al., 2021). Increased body mass may also increase the physiological demands of weight-bearing activity, potentially contributing to greater perceived exertion during physical exercise. Exercise tolerance is therefore influenced not only by metabolic function but also by cardiorespiratory fitness, physical fitness and the presence of obesity-related functional limitations.

Exercise capacity varies substantially among people with obesity and is influenced by factors such as age, habitual physical activity, physical fitness, musculoskeletal function and comorbidities (Oppert et al., 2021). This heterogeneity needs to be considered when evaluating potential dietary influences on exercise responses, as the same NNS exposure may not produce identical physiological or perceptual responses across individuals with different metabolic and functional characteristics.

Baseline glucose regulation, including the effectiveness of insulin action, together with metabolic flexibility and cardiorespiratory fitness, may therefore act as key modifiers when investigating whether NNS consumption influences exercise tolerance. Individuals with similar levels of adiposity may nevertheless differ substantially in their metabolic response to exercise, which may contribute to variability in the effects of dietary factors. Such heterogeneity warrants consideration when interpreting studies of NNS and exercise, especially when findings from healthy or physically active populations are extrapolated to individuals with obesity.

These individual differences provide an important physiological context for investigating potential NNS-exercise interactions.The effects of NNS consumption on exercise tolerance and performance among people affected by obesity have yet to be established.

5. Potential interactions between non-nutritive sweeteners and physical activity

5.1. Acute NNS exposure and exercise

Few studies have examined NNS exposure and exercise together, and direct evidence in adults with obesity is lacking. Therefore, this section considers both studies directly examining NNS during exercise and indirect evidence from related metabolic research. Available human studies have primarily investigated either oral exposure through mouth rinsing or ingestion of specific NNS with carbohydrates. These approaches differ in the extent of systemic exposure: mouth rinsing provides sensory stimulation without meaningful systemic absorption, whereas ingestion permits gastrointestinal and metabolic effects to occur.

In a randomized crossover study of endurance-trained participants, sucralose mouth rinsing during a 12.8-km running time trial did not improve running performance compared with water, although lower-intensity sucralose rinsing reduced the maximum rating of perceived exertion (Hawkins et al., 2017). These findings suggest that oral exposure to intense sweetness was associated with changes in selected perceptual responses during exercise but did not provide evidence of an ergogenic effect. Moreover, findings from mouth-rinse studies cannot be directly extrapolated to the metabolic effects of ingested NNS.

5.2. NNS, carbohydrate co-ingestion and metabolic responses during exercise

A small randomized crossover study investigated the metabolic response to aspartame consumed during exercise with or without carbohydrate. In nine healthy recreationally active men, insulin concentrations were lower when aspartame was consumed with carbohydrate than with carbohydrate alone, whereas aspartame alone did not produce a comparable response (Siegler et al., 2012). The study included only healthy men without obesity or known metabolic disease and examined a single NNS. These findings therefore provide preliminary evidence of a potential NNS-carbohydrate interaction during exercise but cannot be directly extrapolated to obesity-related exercise responses or to other NNS.

A systematic review of human studies also reported variable effects of non- and low-calorie sweeteners on substrate oxidation, energy expenditure and catecholamine responses (Andersen et al., 2023). These studies were not specifically designed to investigate NNS during exercise in individuals with obesity, limiting their direct applicability to the population considered in this review.

5.3. NNS, obesity and exercise-related outcomes

Evidence from studies outside the exercise setting suggests that metabolic responses to NNS may differ according to metabolic phenotype. In a randomized crossover study, sucralose ingestion before an oral glucose tolerance test produced different insulin responses in participants with normal weight and obesity, despite increased glucose exposure in both groups (Nichol et al., 2020). Although no exercise was performed, these findings suggest that obesity may modify the acute metabolic response to a specific NNS.

Evidence concerning longer-term NNS consumption is also indirect. In the one-year SWEET randomized controlled trial, adults with overweight or obesity consuming sweeteners and sweetness enhancers within a healthy, sugar-reduced diet showed greater maintenance of weight loss and changes in gut microbiota composition compared with the control dietary intervention (Pang et al., 2025). The study was not designed to assess exercise performance, exercise tolerance or physiological adaptation to exercise. Its findings therefore provide information about the broader metabolic context of NNS use in obesity rather than direct evidence of an NNS-exercise interaction.

Taken together, the available human evidence addresses different components of the research question rather than the interaction between NNS consumption, physical exercise and obesity. Studies conducted during exercise have primarily involved healthy, physically active participants, whereas studies including individuals with obesity have generally examined NNS-related metabolic responses outside the exercise setting. This separation of evidence limits the ability to determine whether NNS consumption modifies exercise responses in adults with obesity.

6. Discussion

6.1. Interpretation of the evidence

The evidence reviewed in this paper does not currently support a clear conclusion that NNS consumption modifies the metabolic or functional response to exercise in individuals with obesity. The available studies suggest that NNS can influence selected metabolic or perceptual responses under specific experimental conditions, but these effects are not consistent across NNS, modes of administration or study populations (Andersen et al., 2023; Hawkins et al., 2017; Siegler et al., 2012; Nichol et al., 2020). The evidence directly addressing NNS use during exercise is substantially more limited than the evidence examining either NNS exposure or exercise independently (Hawkins et al., 2017; Siegler et al., 2012; Andersen et al., 2023).

The literature reviewed here can be understood as arising from two largely separate areas of research: the metabolic effects of NNS and the physiological responses to exercise under conditions of obesity. Exercise modifies glucose disposal, insulin sensitivity and substrate metabolism, while NNS are associated with changes in selected aspects of glucose regulation, endocrine signaling and substrate oxidation under specific experimental conditions (Oppert et al., 2021; Khalafi et al., 2023; Moriconi et al., 2020; Andersen et al., 2023). A mechanistic overlap between pathways influenced by NNS and pathways involved in exercise metabolism does not, by itself, demonstrate a physiological interaction between NNS consumption and exercise. The potential interaction is likely to depend on several contextual factors, including the specific sweetener, dose, timing of exposure, carbohydrate availability, exercise modality and intensity, and baseline metabolic phenotype (Hawkins et al., 2017; Siegler et al., 2012; Nichol et al., 2020; Andersen et al., 2023). To date, the studies identified in this review have not examined these factors simultaneously in adults with obesity. These findings are therefore better interpreted as providing a biological rationale for further investigation rather than demonstrating a clinically meaningful NNS–exercise interaction.

The main limitation of the current evidence is therefore not the absence of plausible biological mechanisms, but the lack of studies directly integrating NNS exposure, exercise and obesity within the same experimental design. Existing findings are difficult to compare because they differ in NNS type, dose, route and timing of administration, exercise protocol and participant characteristics (Hawkins et al., 2017; Siegler et al., 2012; Nichol et al., 2020; Andersen et al., 2023). Consequently, findings from healthy, physically active populations cannot be assumed to reflect physiological responses in individuals with obesity.

6.2. Relevance to individuals with obesity

NNS-exercise interactions may be of particular relevance in obesity because disturbances in insulin action, altered substrate metabolism and impaired metabolic flexibility can affect responses to both dietary exposures and physical exercise (Choo et al., 2026; Oppert et al., 2021). Metabolic phenotype may influence responses to specific NNS, as suggested by differences in insulin responses to sucralose among participants with normal weight and obesity (Nichol et al., 2020). Whether such differences have functional consequences during exercise remains unclear.

Obesity represents a heterogeneous metabolic condition, with differences in insulin sensitivity, glucose regulation and physical fitness that may contribute to variability in responses to both dietary interventions and exercise. Differences in insulin sensitivity, glucose regulation and physical fitness may contribute to variability in responses to both dietary interventions and exercise. Future studies should therefore consider baseline metabolic characteristics when evaluating potential NNS-exercise interactions rather than treating individuals with obesity as a homogeneous population.

6.3. Clinical and public health implications

The current evidence does not justify recommending NNS as an ergogenic aid, nor does it provide sufficient evidence to recommend avoiding NNS specifically because of concerns regarding exercise tolerance under conditions of obesity. Evidence regarding NNS as substitutes for caloric sweeteners suggests that their effects on body weight depend on the comparator and overall dietary context (Laviada-Molina et al., 2020; Madjd et al., 2018; Pang et al., 2025; Wen et al., 2026).

For exercise-based obesity management, structured physical activity remains a fundamental component of treatment, while NNS should be considered within the broader dietary context rather than as an independent determinant of exercise performance or metabolic adaptation (Oppert et al., 2021).

From a public health perspective, the potential role of NNS should therefore be considered in relation to their intended dietary use rather than isolated metabolic effects. When NNS replace caloric sugars within an overall energy-controlled dietary pattern, they may represent one possible strategy for reducing sugar intake and supporting dietary adherence. Their use should therefore be evaluated according to the broader dietary pattern, individual preferences and metabolic characteristics rather than as a specific strategy for improving exercise capacity.

7. Future directions

The most important priority for future research is to directly investigate the interaction between NNS consumption and physical exercise in individuals with obesity. Randomized crossover or controlled intervention studies should compare specific NNS with appropriate control conditions and distinguish between acute exposure and habitual consumption. Studies should examine NNS consumed alone and in combination with carbohydrate, with standardized timing relative to exercise (Siegler et al., 2012; Oppert et al., 2023).

Future studies would benefit from greater methodological standardization. Clear reporting of the specific NNS, dose, frequency, duration and route of administration is needed, while exercise modality, intensity, duration and nutritional status can be standardized or systematically varied. The selection of control conditions also needs to reflect the research question, as comparisons with caloric sugars address substitution effects, whereas comparisons with water or unsweetened products address potential effects beyond the absence of caloric sugars (Laviada-Molina et al., 2020; Madjd et al., 2018).

Individual metabolic phenotype is a key consideration when investigating NNS-exercise interactions. Baseline insulin sensitivity, glucose tolerance, habitual NNS consumption and cardiorespiratory fitness may influence responses to both NNS and exercise. Future trials would benefit from characterizing participants according to baseline metabolic and functional characteristics and investigating whether specific subgroups respond differently (Nichol et al., 2020).

Finally, future studies should prioritize clinically meaningful outcomes, including glucose and insulin responses, substrate oxidation, perceived exertion, exercise performance and cardiorespiratory exercise tolerance. Longer-term randomized trials are also needed to determine whether habitual NNS consumption modifies the metabolic adaptations normally associated with structured exercise training. Mechanistic measures such as incretin responses and gut microbiota may provide additional information but should be interpreted alongside functional outcomes rather than as evidence of clinical benefit. At present, well-controlled studies addressing NNS type, dose, timing, carbohydrate co-ingestion, metabolic phenotype and exercise modality are required before firm conclusions regarding NNS-exercise interactions in obesity can be drawn.

Disclossure

Supplementary Materials

Not applicable

Author Contributions

All authors have read and agreed to the published version of the manuscript.

Conceptualization: Maria Berek

Methodology: Kacper Kmieciak, Jagoda Stańska, Klaudia Borycka, Aleksandra Dobrzańska, Anna Hajduk, Piotr Szymański, Maria Berek

Investigation: Aleksandra Szymańska, Filip Kostanek, Aleksandra Dobrzańska, Anna Hajduk, Jagoda Stańska, Klaudia Borycka

Formal analysis: Filip Kostanek, Piotr Szymański, Kacper Kmieciak

Writing - original draft preparation: Maria Berek, Aleksandra Baranowska, Kacper Kmieciak, Anna Hajduk, Aleksandra Szymańska,

Data curation: Klaudia Borycka, Aleksandra Baranowska,

Visualization: Aleksandra Baranowska, Maria Berek, Aleksandra Szymańska, Filip Kostanek

Project administration: Maria Berek, Jagoda Stańska

Funding: The study received no specific funding

Institutional Review Board Statement: Not Applicable

Informed Consent Statement: Not applicable

Data Availability Statement: The data presented in this study is available upon request from the corresponding author.

Acknowledgements: Not applicable

Conflicts of Interest: The authors deny any conflict of interest

Declaration of generative AI and AI-assisted technologies in the writing process:

The authors utilized ChatGPT (OpenAI) exclusively for language editing and grammatical proofreading. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References

1. Agopsowicz, K., Blicharz, K., Piwowar, M., Bąk, A., Zdziebło, A., Biernacka, M., Stolarczyk, D., Sitko, M., Biernacki, I. and Mikołajczyk, P. (2025) 'Artificial Sweeteners and Glycemic Control: Implications for Metabolic Health', Quality in Sport, 42, pp. 61161. Available at: https://doi.org/10.12775/QS.2025.42.61161.
2. Andersen, S.S.H., Zhu, R., Kjølbæk, L. and Raben, A. (2023) 'Effect of Non- and Low-Caloric Sweeteners on Substrate Oxidation, Energy Expenditure, and Catecholamines in Humans: A Systematic Review', Nutrients, 15(12), p. 2711. Available at: https://doi.org/10.3390/nu15122711.
3. Azad, M.B., Abou-Setta, A.M., Chauhan, B.F., Rabbani, R., Lys, J., Copstein, L., Mann, A., Jeyaraman, M.M., Reid, A., Fiander, M., MacKay, D., McGavock, J., Wicklow, B. and Zarychanski, R. (2017) 'Nonnutritive sweeteners and cardiometabolic health: a systematic review and meta-analysis of randomized controlled trials and prospective cohort studies', CMAJ, 189(28), pp. E929-E939. Available at: https://doi.org/10.1503/cmaj.161390.

4. Buszek, J., Czerniak, P., Antoszewska, A., Bargiel, W., Bąk, D., Wajdowicz, H., Warzocha, M., Medygrał, M., Buszek, S., Sokołowska, A., Rumian, M. and Kosel, A. (2025) 'Are Artificial Sweeteners a Healthier Alternative to Sugar? - Systematic Review', Quality in Sport, 38, pp. 58150. Available at: https://doi.org/10.12775/QS.2025.38.58150.
5. Choo, Y.N., Ravi, R.N. and Subramaniyan, V. (2026) 'Insulin resistance induced by obesity: Mechanisms, metabolic implications and therapeutic approaches', Molecular Biology Reports, 53(1), p. 357. Available at: https://doi.org/10.1007/s11033-026-11509-3.
6. Conz, A., Salmona, M. and Diomede, L. (2023) 'Effect of Non-Nutritive Sweeteners on the Gut Microbiota', Nutrients, 15(8), p. 1869. Available at: https://doi.org/10.3390/nu15081869.
7. Crakes, K.R., Questell, L., Soni, S. and Suez, J. (2025) 'Impacts of non-nutritive sweeteners on the human microbiome', Immunometabolism, 7(2), p. e00060. Available at: https://doi.org/10.1097/IN9.0000000000000060.
8. Hawkins, K.H., Krishnan, S., Ringos, L., Garcia, V. and Cooper, J.A. (2017) 'Running Performance with Nutritive and Non-Nutritive Sweetened Mouth Rinses', International Journal of Sports Physiology and Performance, 12(8), pp. 1105-1110. Available at: https://doi.org/10.1123/ijspp.2016-0577.
9. Herzog, H., Zhang, L., Fontana, L. and Neely, G.G. (2025) 'Impact of non-sugar sweeteners on metabolism beyond sweet taste perception', Trends in Endocrinology & Metabolism, 36(6), pp. 563-576. Available at: https://doi.org/10.1016/j.tem.2024.10.008.
10. Kang, J., Fardman, B.M., Ratamess, N.A., Faigenbaum, A.D. and Bush, J.A. (2023) 'Efficacy of Postprandial Exercise in Mitigating Glycemic Responses in Overweight Individuals and Individuals with Obesity and Type 2 Diabetes—A Systematic Review and Meta-Analysis', Nutrients, 15(20), p. 4489. Available at: https://doi.org/10.3390/nu15204489.
11. Khalafi, M., Symonds, M.E., Ghasemi, F., Rosenkranz, S.K., Rohani, H. and Sakhaei, M.H. (2023) 'The effects of exercise training on postprandial glycemia and insulinemia in adults with overweight or obesity and with cardiometabolic disorders: A systematic review and meta-analysis', Diabetes Research and Clinical Practice, 201, p. 110741. Available at: https://doi.org/10.1016/j.diabres.2023.110741.

12. Laviada-Molina, H., Molina-Segui, F., Pérez-Gaxiola, G., Cuello-García, C., Arjona-Villicaña, R., Espinosa-Marrón, A. and Martinez-Portilla, R.J. (2020) 'Effects of nonnutritive sweeteners on body weight and BMI in diverse clinical contexts: Systematic review and meta-analysis', Obesity Reviews, 21(7), p. e13020. Available at: https://doi.org/10.1111/obr.13020.
13. Li, D., Han, L., Yu, Z., Teng, X., Ma, Y. and Wang, D. (2026) 'Effects of non-nutritive sweeteners on body weight: a systematic review and meta-analysis of randomized controlled trial (RCT) studies', Journal of Endocrinological Investigation, 49, pp. 11-24. Available at: https://doi.org/10.1007/s40618-025-02654-w.
14. Madjd, A., Taylor, M.A., Delavari, A., Malekzadeh, R., Macdonald, I.A. and Farshchi, H.R. (2018) 'Effects of replacing diet beverages with water on weight loss and weight maintenance: 18-month follow-up, randomized clinical trial', International Journal of Obesity, 42(4), pp. 835-840. Available at: https://doi.org/10.1038/ijo.2017.306.
15. Moriconi, E., Feraco, A., Marzolla, V., Infante, M., Lombardo, M., Fabbri, A. and Caprio, M. (2020) 'Neuroendocrine and Metabolic Effects of Low-Calorie and Non-Calorie Sweeteners', Frontiers in Endocrinology, 11, p. 444. Available at: https://doi.org/10.3389/fendo.2020.00444.
16. Nichol, A.D., Salame, C., Rother, K.I. and Pepino, M.Y. (2020) 'Effects of Sucralose Ingestion versus Sucralose Taste on Metabolic Responses to an Oral Glucose Tolerance Test in Participants with Normal Weight and Obesity: A Randomized Crossover Trial', Nutrients, 12(1), p. 29. Available at: https://doi.org/10.3390/nu12010029.
17. Nobs, S.P. and Elinav, E. (2023) 'Nonnutritive sweeteners and glucose intolerance: Where do we go from here?', Journal of Clinical Investigation, 133(10), p. e171057. Available at: https://doi.org/10.1172/JCI171057.
18. Oppert, J.M., Bellicha, A., van Baak, M.A., Battista, F., Beaulieu, K., Blundell, J.E., Carraça, E.V., Encantado, J., Ermolao, A., Pramono, A., Farpour-Lambert, N., Woodward, E., Dicker, D. and Busetto, L. (2021) 'Exercise training in the management of overweight and obesity in adults: Synthesis of the evidence and recommendations from the European Association for the Study of Obesity Physical Activity Working Group', Obesity Reviews, 22(S4), p. e13273. Available at: https://doi.org/10.1111/obr.13273.
19. Oppert, J.M., Ciangura, C. and Bellicha, A. (2023) 'Physical activity and exercise for weight loss and maintenance in people living with obesity', Reviews in Endocrine and

Metabolic Disorders, 24(5), pp. 937-949. Available at: https://doi.org/10.1007/s11154-023-09805-5.

20. Pang, M.D., Goossens, G.H. and Blaak, E.E. (2021) 'The Impact of Artificial Sweeteners on Body Weight Control and Glucose Homeostasis', Frontiers in Nutrition, 7, p. 598340. Available at: https://doi.org/10.3389/fnut.2020.598340.
21. Pang, M.D., Kjølbæk, L., Bastings, J.J.A.J., Andersen, S.S.H., Umanets, A., Sost, M.M., Navas-Carretero, S., Reppas, K., Finlayson, G., Hodgkins, C.E., del Álamo, M., Lam, T., Moshoyiannis, H., Feskens, E.J.M., Adam, T.C.M., Goossens, G.H., Halford, J.C.G., Harrold, J.A., Manios, Y., Martinez, J.A., Blaak, E.E. and Raben, A. (2025) 'Effect of sweeteners and sweetness enhancers on weight management and gut microbiota composition in individuals with overweight or obesity: the SWEET study', Nature Metabolism, 7(10), pp. 2083-2098. Available at: https://doi.org/10.1038/s42255-025-01381-z.
22. Siegler, J., Howell, K., Vince, R., Bray, J., Towlson, C., Peart, D., Mellor, D. and Atkin, S. (2012) 'Aspartame in conjunction with carbohydrate reduces insulin levels during endurance exercise', Journal of the International Society of Sports Nutrition, 9(1), p. 36. Available at: https://doi.org/10.1186/1550-2783-9-36.
23. Skowrońska-Borsuk, J., Narożniak, M., Pękacka, A.E., Czerwiec, B., Borsuk, A., Pergoł, J.K., Wojtas, M., Borkowska, J., Krupa, Z. and Sposób, J. (2025) 'Erythritol as a Next-Generation Sweetener: properties, metabolism, cardiovascular risk and gut health implications - A literature review', Quality in Sport, 43, pp. 62351. Available at: https://doi.org/10.12775/QS.2025.43.62351.
24. Suez, J., Korem, T., Zeevi, D., Zilberman-Schapira, G., Thaiss, C.A., Maza, O., Israeli, D., Zmora, N., Gilad, S., Weinberger, A., Kuperman, Y., Harmelin, A., Kolodkin-Gal, I., Shapiro, H., Halpern, Z., Segal, E. and Elinav, E. (2014) 'Artificial sweeteners induce glucose intolerance by altering the gut microbiota', Nature, 514(7521), pp. 181-186. Available at: https://doi.org/10.1038/nature13793.
25. Sylvetsky, A.C. and Rother, K.I. (2018) 'Nonnutritive Sweeteners in Weight Management and Chronic Disease: A Review', Obesity, 26(4), pp. 635-640. Available at: https://doi.org/10.1002/oby.22139.
26. Teysseire, F., Bordier, V., Beglinger, C., Wölnerhanssen, B.K. and Meyer-Gerspach, A.C. (2024) 'Metabolic Effects of Selected Conventional and Alternative Sweeteners: A Narrative Review', Nutrients, 16(5), p. 622. Available at: https://doi.org/10.3390/nu16050622.

27. Toews, I., Lohner, S., Küllenberg de Gaudry, D., Sommer, H. and Meerpohl, J.J. (2019) 'Association between intake of non-sugar sweeteners and health outcomes: systematic review and meta-analyses of randomised and non-randomised controlled trials and observational studies', BMJ, 364, p. l156. Available at: https://doi.org/10.1136/bmj.l156.
28. Walbolt, J. and Koh, Y. (2020) 'Non-nutritive Sweeteners and Their Associations with Obesity and Type 2 Diabetes', Journal of Obesity & Metabolic Syndrome, 29, pp. 114-123. Available at: https://doi.org/10.7570/jomes19079.
29. Wen, J., Wu, Q., Li, S., Liu, Y. and Wen, D. (2026) 'Effects of Non-Nutritive Sweeteners on Weight Loss and Maintenance, Metabolic Improvement, and Appetite Regulation in Weight Management Programs: A Systematic Review and Meta-Analysis of Randomized Controlled Trials', Nutrition Reviews, 84(2), pp. 318-332. Available at: https://doi.org/10.1093/nutrit/nuaf094.
30. World Health Organization (2025) ‘Obesity and overweight’, 8 December. WHO - Obesity and overweight.
31. Zhang, R., Noronha, J.C., Khan, T.A., McGlynn, N., Back, S., Grant, S.M., Kendall, C.W.C. and Sievenpiper, J.L. (2023) 'The Effect of Non-Nutritive Sweetened Beverages on Postprandial Glycemic and Endocrine Responses: A Systematic Review and Network Meta-Analysis', Nutrients, 15(4), p. 1050. Available at: https://doi.org/10.3390/nu15041050.