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Carbohydrates: friend or foe to post-exercise hunger?

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Introduction

A high percentage of the general population are either overweight or obese in England (men, 69%; women, 59%) (NHS Digital, 2022). Obesity, a chronic disease, is a global epidemic and is associated with many life-limiting health problems such as diabetes, hypertension and cardiovascular disease. This epidemic creates a huge economic burden on the National Health Service (NHS); in 2019 alone, obesity and morbidities cost the NHS £6.1 billion (NHS Digital, 2022). Research into weight loss interventions is paramount to understand how to decrease the burden on our health care systems and create a healthier population.

Obesity is caused by a sustained period in positive energy balance when energy intake exceeds energy expenditure; but energy intake and exercise are not the sole dictating factors. There are numerous additional factors that influence the development of obesity, including genetics, macronutrient composition, ultra-processed food, global industrialisation and the development of technology that encourages sedentary lifestyles. Public health measures to tackle obesity include lifestyle changes (exercise and diet), although often these fail due to compensatory eating behaviours and reliance on continued motivation from the patient. Secondary therapies include pharmaceutical and surgical means, but these are a monetary burden on the NHS.

It is well published that acute, single bouts of exercise have a transient effect on appetite-regulating hormones. Immediately after exercise, appetite is suppressed and the decision to eat is delayed. This indicates that novel, obesity-tackling therapies could be developed through exploring the underpinning metabolic mechanisms that characterise the transient suppression of appetite. To explore the metabolic principles of weight loss, in a recent article in *The Journal of Physiology*, Frampton et al. (2023) investigated the interplay between dietary carbohydrates and exercise with regard to appetite regulation. Here we review Frampton et al. (2023) to understand whether carbohydrate ingestion prior to exercise attenuates post-exercise hunger.

Method

Frampton et al. (2023) conducted a four-condition randomised crossover study in 12 healthy, adult men aged 24 ± 5 years. All participants ingested 75 g maltodextrin or a placebo beverage followed by 30 min of rest or exercise on a cycle ergometer at 75% $\dot{V}_{O_2\max}$. Participants were then asked to rest for 90 min; during the rest period participants were given an *ab libitum* meal to eat within 20 min, until comfortably full. At baseline and every 15 min after carbohydrate ingestion, venous blood samples and visual analogue scale (VAS) scores were collected to measure subjective appetite and blood-borne hormones and metabolites. Finally, energy expenditure and substrate oxidation were measured using pulmonary gas analysis, and energy intake was derived from measuring the difference in mass of the *ad libitum* meal before and after consumption.

Findings

This study reported no significant independent effect of exercise or carbohydrate feeding on energy intake based on the mass consumed of the *ad libitum* meal but did show a significant interaction between carbohydrate and exercise whereby less food was consumed *ad libitum* in the carbohydrate and exercise condition. Only when carbohydrate was paired with exercise did this lead to reductions in energy intake through the *ad libitum* meal. However, the exercise intervention without carbohydrate feeding elicited the lowest acute energy balance. Subjective appetite scores derived from the VAS were not associated with *ad libitum* energy intake.

Carbohydrate and exercise both exhibited similar independent effects on appetite-regulating hormones, whereby glucagon-like peptide-1 (GLP-1) and glucagon increased and (active) ghrelin decreased. Independent effects were evident where insulin concentration increased with carbohydrate. However, opposing independent effects were observed in which insulin concentration was decreased by exercise. Peptide YY increased with carbohydrate ingestion, but no independent effects were observed with exercise. Carbohydrate and exercise demonstrated an interactive effect with increases in insulin concentration, from carbohydrate ingestion, being attenuated by exercise. No other interaction between carbohydrate and exercise was observed. The study identifies GLP-1 as an important mediator of meal energy intake following exercise showing a negative association between GLP-1 and energy intake. A similar negative correlation was found between succinate and *ad libitum* energy intake during the exercise interventions.

Finally, carbohydrate ingestion increased concentrations of high-density lipoprotein cholesterol while exercise increased cholesterol concentrations and intermediate-density lipoprotein particle number.

Discussion

This study reported changes in metabolic, hormonal and behavioural factors surrounding appetite regulation with exogenous carbohydrate feeding and exercise.

When focusing on the behavioural responses to the *ad libitum* meal, it was unclear which was the driving factor between carbohydrate and exercise when energy intake decreased in response to the significant interactive effect. The absence of reported *post hoc* results made the determinant of energy intake difficult to elucidate. This absence could be attributed to the authors focusing on the metabolic

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and hormonal mechanisms at play during appetite regulation, rather than exploring the behavioural response demonstrated through energy intake. In addition, the results indicate that the lower *ad libitum* meal intake in the carbohydrate and exercise condition was not powerful enough to affect overall energy balance. This suggests that the interactive effect of carbohydrate and exercise may not be powerful enough to facilitate weight loss and that exercise independent of carbohydrate feeding is still more effective overall. However, as suggested by Frampton et al. (2023), these findings could encourage future research to establish if exercise in the fasted state leads to greater weight loss.

Pharmaceuticals such as Semaglutide can be used to treat obesity by increasing levels of the anorexigenic hormone GLP-1 to suppress appetite. However, the administration of pharmaceuticals is costly and can lead to negative side effects. The finding of Frampton et al. (2023) of the appetite-suppressive effects of GLP-1 following carbohydrate feeding and exercise makes a useful contribution to support non-pharmaceutical weight loss interventions.

Subjective appetite, as reported through VAS scores, did not show any correlation with *ad libitum* energy intake across all study conditions. Therefore, the results suggest that the drive to eat is not a reliable determinant of appetite regulation. That said, VAS scores poorly predict energy intake (Holt et al., 2017), possibly because of poor ecological validity surrounding *ad libitum* feeding. The extreme controlled environment whereby the *ad libitum* meal was provided potentially distorted true eating behaviours as a result of a triggered feeding response.

A notable strength of this study is the novel finding that succinate, a circulating metabolite generated from gut microbial metabolic activity (Macfarlane & Macfarlane, 2003), was a key predictor of energy intake during exercise conditions, supporting the requirement for future research investigating mechanisms to increase succinate levels and its potential impact on energy intake. For example, future research could investigate the supplementation of succinate and its role in appetite suppression. An additional novel finding was the characterisation of the acute lipoprotein response to exercise. This understudied acute response could address an exciting gap in the research for future weight loss interventions. However, the literature reports associations between chronic changes in lipoprotein parameters and high cardiovascular disease risk, and appropriate evidence is required before promoting the increase in lipoproteins to a population that already hold a high cardiovascular disease risk. Frampton et al. (2023) conducted their study in healthy men. We speculate that healthy males may have a different plasma metabolome and hormone profiles to obese and/or female populations, and findings should be interpreted with caution when extrapolating to clinical populations subject to weight-loss interventions.

Frampton et al. (2023) provide valuable insight into appetite suppression following a single aerobic exercise bout, in healthy individuals. However, obesity is a disease caused by chronic behaviour patterns. Therefore, it is important for future studies to investigate the long-term effects of carbohydrate feeding and aerobic exercise on energy intake, particularly in overweight and obese individuals. Furthermore, longitudinal studies could improve the understanding of compensatory eating behaviours notably succeeding energy expenditure, which may not have been captured in the present study. In addition to manipulating the time course of the present study, future research could explore the effect of resistance exercise on post-exercise energy intake. A similar study by Ballard et al. (2007) found that carbohydrate feeding during resistance exercise produced similar results in *ad libitum* meal energy intake as the aerobic exercise in the present study. Follow-up research could allow for the conjunction of resistance and aerobic exercise in weight loss programmes, therefore giving overweight or obese individuals greater choice when managing their weight using exercise.

In conclusion, the research performed by Frampton et al. (2023) highlights a knowledge gap in determinants of post-exercise energy intake, and their findings inform future research to support weight loss interventions for obesity.

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Additional information

Competing interests

None.

Author contributions

All authors: conception or design of the work, and drafting the work or revising it critically for important intellectual content. All authors have read and approved the final version of this manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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Keywords

ad libitum, appetite suppression, carbohydrate, exercise, hormones, metabolism

Supporting information

Additional supporting information can be found online in the Supporting Information section at the end of the HTML view of the article. Supporting information files available:

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