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Title: Assessment of the post-exercise glycemc response to food: considering prior nutritional status.

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To the editor:

We read with interest a recent article in *Nutrition* by Roberts et al. [1], demonstrating the effects of prior exercise, and of protein co-ingestion, on postprandial glycemia. We agree with the authors that this is an important area of research, given the close links between postprandial glucose excursions and the risk of type 2 diabetes, cardiovascular disease and mortality [2-4]. Roberts et al. report that prior exercise (cycling at 60% of age-predicted maximum heart rate for 45 min) does not influence the glycemic response to 50 g of glucose ingestion [1]. Furthermore, this effect was similar with, and without, 21.5 g of protein co-ingested with the glucose.

The authors highlight some putative explanations for the absence of an effect of prior exercise on postprandial glycemia; including the exercise intensity, the timing of test drink administration relative to exercise, and the residual influence of habitual exercise routines. An important point however, was omitted; that of the nutritional state prior to exercise. Participants performed the exercise bout after an overnight fast [1]. In western society, the majority of the day is spent in the postprandial state [5]. Therefore, understanding whether the response of an intervention (in this instance, exercise and protein co-ingestion) is dependent upon the prior nutritional state (fasted vs. postprandial), is highly pertinent.

We recently conducted a study to address this [6]. Young, healthy males completed four trials, comprising of a fasted rest trial; a fasted exercise trial (running at 60% of peak oxygen consumption for ~60 min); a breakfast rest trial; and a breakfast exercise trial. The glycemic response to consumption of a mixed-macronutrient test drink (chocolate milk; 16 g protein, 56 g carbohydrate and 8 g fat) was determined following the exercise, and equivalent rest periods. In the fasted state, glucose tolerance [assessed by the area under the curve (AUC)] was remarkably similar between rest and exercise trials, corroborating the findings of Roberts et al. [1]. When breakfast had been consumed however, we were surprised to note a

15% *increase* in the glucose AUC with exercise, compared to rest [6]. Although Roberts et al. alluded to some of the potential reasons for a similar glycemic response after exercise, compared to rest, we believe some key components were overlooked.

The change in blood glucose concentration following food ingestion is influenced by the rate of appearance and the rate of disappearance of blood glucose. As it is well-documented that acute exercise usually enhances both insulin-independent, and insulin-mediated glucose uptake [7], it is somewhat unexpected that glucose tolerance is unaffected by prior exercise. Furthermore, whole-body rates of glucose disappearance are elevated following exercise [8]. This can however, be offset by an even greater increase in the rate of glucose appearance [8], principally due to an increase in the rate of appearance of the orally-ingested glucose, suggesting that rates of intestinal glucose absorption, and splanchnic glucose output, are enhanced. The effect that exercise has in the postprandial state (after breakfast), could be explained by elevated splanchnic perfusion with exercise in the fed state [9], and/or greater hepatic glycogenolysis (and subsequent glucose output) stimulated by higher liver glycogen stores following breakfast consumption [10]. These ostensible mechanisms however, need clarification.

It is clear that studying the postprandial response to ingested food is a worthy tool to understand the impact of exercise and nutritional interventions on risk factors for morbidity and mortality. In order to gain the most appropriate insight for translation into practical guidelines for daily living, it is important to consider the prescribed intervention in the context of the fasted or postprandial state.

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