professionals in the community, hospitals, and clinics are the most effective and profound solutions in both the prevention and treatment of childhood diarrhea. However, management of childhood diarrhea was not independently associated with knowledge from educational training particularly in rural areas [6] and developing countries including Nigeria [7] and the Gambia [2]. Thus, translating public health knowledge into practice is a critical need among caregivers who are the first step in the management of childhood diarrhea. Additionally, attention should be given to caregivers who do not have any experience in the use of oral rehydration solution and less frequent contact with health professionals [8].

Diverse knowledge in caregiver practice with regard to prevention and management of childhood diarrhea may involve breastfeeding, hand-washing with soap, safe treatment and storage of water and foods, proper disposal of human waste, and correct use of oral rehydration solution [5]. The implementation of such programs as well as the evaluation and monitoring of systems should be established as a network among governments, nongovernmental organizations, health care providers and clinical facilities, and communities to effectively reduce childhood deaths caused by diarrhea.

Table
Causes of childhood deaths from diarrhea and AIDS in different world regions in 2010 [2]

<table>
<thead>
<tr>
<th>World regions</th>
<th>Cause of childhood deaths (% cause-specific deaths)</th>
<th>Diarrhea</th>
<th>AIDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Africa (3.552 million)</td>
<td></td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Americas (0.284 million)</td>
<td></td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Eastern Mediterranean (1.062 million)</td>
<td></td>
<td>12</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Europe (0.161 million)</td>
<td></td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Southeast Asia (2.096 million)</td>
<td></td>
<td>11</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Western Pacific (0.467 million)</td>
<td></td>
<td>5</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>

Assessment of the post-exercise glycemic response to food: Considering prior nutritional status

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 coingestion, 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 for 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 coingested 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 before 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 coingestion) is dependent on the prior nutritional state (fasted versus postprandial), is highly pertinent.

We recently conducted a study to address this [6]. Young, healthy men 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 with rest [6]. Although Roberts et al alluded to some of the potential reasons for a similar glycemic response after exercise compared with 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 splanchic glucose output are enhanced. The effect that exercise has in the postprandial state (after breakfast) could be explained by elevated splanchic 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.

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It is clear that studying the postprandial response to ingested food is a useful tool for understanding the effect 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.

References


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Fed or fasted? New considerations for exercise and glycemic response testing

Author’s response re.: Assessment of the post-exercise glycemic response to food: considering prior nutritional status

To the Editor:

I write in response to the article by Gonzalez and Stevenson [1], commenting on our recently published study investigating the effects of prior exercise and protein coingestion on the glycemic response to carbohydrates. In our study, protein coingestion lowered the glycemic response to carbohydrates; however, prior aerobic exercise had no effect [2]. Although we discussed a number of factors that could potentially explain this result, Gonzalez and Stevenson suggest that an important factor was omitted—participants’ nutritional state before exercise [1]. Gonzalez et al. conducted a study investigating the effects of breakfast and exercise on the glycemic response to a test drink (chocolate milk) containing carbohydrates, protein, and fat [3]. The authors found that in the fasted state, there was no difference in the glucose area under the curve (AUC) between rest and exercise trials, which confirms the results of our study. However, they found that in the fed state (i.e., when breakfast was consumed), the glucose AUC was 15% higher after exercise than at rest [3]. This appears to be a novel study and raises many important scientific questions, such as the splanchnic effects (blood flow, intestinal glucose absorption, glycojenolysis, etc.) and the hormonal effects involved with this observed increase in glucose AUC. It also challenges the customary fasted condition that is generally used for standardization in clinical exercise trials.

In our study, participants were fasted to ensure consistency within and between participants. Although this may not always be reflective of every eating occasion, we felt this was a prudent control measure to ensure the best chance of answering the research question. We did not allude to prior nutritional intake as a potential explanatory factor for the lack of effect observed on exercise on glycemic response, due to the limited evidence for this in the literature. The study by Gonzalez et al. [3] is the first, to our knowledge, to investigate this effect, and the results highlight the need for further work in the area. Some further areas of research could include investigating differences in macronutrient distributions of the breakfast meal, and more specifically, whether there are differences in the glycemic response depending on the amount of carbohydrates consumed per kg body weight at this meal, as this may affect glycogen stores and circulating blood glucose. The research should incorporate both males and females to elucidate whether there are any sex differences in hormonal or splanchnic effects on the glycemic response. The macronutrient composition of the test drink is also important, as differing proportions of these are likely to affect gastric emptying and insulin secretion, and in turn, glycemic response.

References


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