



High-glucose ingestion and acute exercise elicit dynamic and individualised responses in systemic markers of redox homeostasis.

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Background. Oxidation-reduction (redox) reactions are involved in regulating numerous aspects of human health and disease. As such, systemic markers of redox homeostasis in humans are commonly used to assess whether a redox stimulus (for example high-glucose ingestion or acute exercise) leads to a state of oxidative eustress (beneficial for health) or oxidative distress (detrimental to health). However, evidence now suggests that redox responses in humans are largely variable, yet few studies have reported individual responsiveness to common redox stimuli such as exercise and meal-ingestion. Furthermore, whether systemic markers of redox homeostasis measured in whole tissue samples (i.e., blood) can reliably reflect oxidative eu/distress following more complex stimuli (for example when a high-glucose meal is ingested after acute exercise) is unclear. **Methods.** We examined the effects of aerobic exercise (1 h of cycling at 70-75% $\text{VO}_{2\text{peak}}$), high-glucose mixed-nutrient meal ingestion (45% carbohydrate [$1.1 \text{ g glucose}\cdot\text{kg}^{-1}$], 20% protein, and 35% fat), and the meal when ingested both 3 h and 24 h post-exercise, on an array of commonly studied redox biomarkers in plasma/serum. Eight recreationally active healthy men (age: 28 ± 1 years, BMI: $24\pm 1 \text{ kg/m}^2$; mean \pm SEM) completed the randomised crossover study. **Results.** Acute exercise increased markers of oxidative stress and antioxidant activity (hydrogen peroxide, 8-isoprostanes, catalase activity, superoxide activity, and nitrate). However, not all markers showed individual homogeneity, for example thiobarbituric acid reactive substances (TBARS) exhibited large inter-individual variability in the direction (4 participants increased and 4 participants decreased) and magnitude of responses (small to large effects). High-glucose ingestion at rest, and when ingested 3 h and 24 h post-exercise, also led to alterations in redox homeostasis as indicated by changes in TBARS, catalase activity, superoxide activity, hydrogen peroxide, 8-isoprostanes, and nitric oxide activity. However, postprandial responses also exhibited large individual responsiveness and varied depending on when the meal was ingested and the postprandial timepoint measured. Responses also varied largely between the different markers of oxidative stress and antioxidant activity. **Conclusion.** Systemic redox homeostasis is dynamically altered after exercise, high-glucose ingestion, and high-glucose ingestion after acute exercise. However, limitations exist when using systemic markers to assess redox homeostasis, especially when oxidative eustress and distress are likely to co-exist. Findings also suggest individual redox responsiveness to redox stimuli which may be of physiological relevance and should be investigated in future human studies.