Editorial

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On the beneficial effects of exercise on hyperglycemia.

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ABSTRACT

Glucose disposal ie. insulin sensitivity is impaired in sedentary individuals, impaired glucose tolerance, and type 2 diabetes. Exercise increases carbohydrate oxidation during and glycogen storage after exercise in these populations. These effects are short-lived and are prior to changes in enzyme activities. Fat oxidation is relatively minor on a gram basis compared to carbohydrate oxidation at an intensity of 65% of VO2 peak. From deductive reasoning this leads to the conclusion that carbohydrate oxidation during exercise and rest is elevated above carbohydrate ingestion and that this muscle and liver "sink" leads to improved glucose disposal/insulin sensitivity. In this editorial I propose guidelines for exercise duration/intensity and carbohydrate ingestion to realize these effects and explain the calculations that resulted in these conclusions.

Keywords : Carbohydrate balance; Insulin Sensitivity; Glucose Disposal; Oxidative glucose disposal; Non-oxidative glucose disposal

Type 2 diabetics have impaired insulin stimulated glucose disposal [1, 2]. This is despite similar Glut-4 translocation during acute exercise[3] . Short-term exercise (1 [4], 7 [5], 10 [6]days) improves indices of glucose disposal in sedentary individuals, in Type 2 diabetics and individuals with impaired glucose tolerance. Therefore, it is unlikely that these improvements were due to changes in enzyme activities or cytokines as sufficient days of training did not ensue. Likewise, the effects of exercise training are short-lived and exercise must be conducted every day or every other day (personal communication John Oliver Holloszy, MD) to keep blood glucose within normal limits. Because of this acute effect of exercise and based on metabolic calculations of carbohydrate balance in the current paper, it appears that the impairments in insulin sensitivity are due to positive carbohydrate balance and the improvements with exercise are due to negative carbohydrate balance during exercise and in post-exercise period (CHO oxidation during exercise + CHO non-oxidative disposal after exercise > CHO intake). The "sink" (deficit in muscle and liver glycogen (carbohydrate) created by exercise during exercise (carbohydrate oxidation) and after exercise (non-oxidative glucose disposal aka glycogen storage) in skeletal muscle lowers blood glucose and increases liver and muscle glycogen stores. It is unlikely that intramuscular triglyceride oxidation is responsible for this effect as the contribution to total energy expenditure as a result of exercise is very minor when compared to muscle glycogen and blood glucose [7]. Although, it is possible that a negative fat balance contributes to this energy deficit ("sink"), albeit and minor effect.

Based on limited data, [5, 6] and requiring validation, I propose 60 min of aerobic exercise (~65% VO2peak) a day and ~< 2.35 g of CHO/kg bodyweight/day or 30 min of aerobic exercise (~65% VO2peak) a day and ~<1.15 g of CHO/kg bodyweight/day. These two scenarios will lead to negative carbohydrate balance and improvements in insulin sensitivity in type 2 diabetics and sedentary individuals. These guidelines are based on the data of Arciero et al [6] where subjects weight was 110.3 kg, age was 53 y , and they ingested 2074 kcals/day (50% carbohydrate) and total carbohydrate oxidation-total carbohydrate intake = carbohydrate balance. Total energy expenditure was calculated by the Mifflin-St. Jeor equation using vigorous activity 5-6 days per week and was =3289 kcals/day. Total carbohydrate oxidation (resting + exercise) was calculated using an estimate of RER of 0.90 (combined exercise and resting). Total carbohydrate ingested 259 g/day - total carbohydrate oxidized 411g/day=-152 g/day (carbohydrate deficit).

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