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Review Article

Carbohydrates Only Wear the "Metabolic black-hat" if you Don't Exercise

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Abstract

Carbohydrates have been much maligned for raising triglycerides, increasing fat mass, and causing insulin resistance. Clearly this a possibility when in positive carbohydrate balance. On the other hand, if one participates in regular physical exercise these negative manifestations of excessive carbohydrate intake are greatly attenuated and are likely completely blocked.

Keywords: Carbohydrates, Exercise, De Novo Lipogenesis, Insulin Resistance.

Background

Three areas where carbohydrate gets a bad reputation is in the synthesis of triglycerides, the deposition of carbohydrate as adipose tissue, and the development of insulin resistance. Hepatic de novo lipogenesis results in triglyceride synthesis from carbohydrate. In a thorough study on glycogen storage capacity and de novo lipogenesis, [1] reported that ~500g (~15 g/kg) of carbohydrate has to be ingested prior to de novo lipogenesis occurring. It must be stated that prior to the ingestion of carbohydrate, the subjects depleted their muscle glycogen stores by a low carbohydrate diet and exercise. Only exercise can reduce muscle glycogen concentrations as there is no phosphatase in skeletal muscle to degrade glucose-6-phosphate to glucose for release into the blood as there is in the liver. Thus, prior depletion of muscle glycogen through exercise is essential in creating a "muscle sink" where glucose can be disposed of. None of the subjects were obese or had insulin resistance.

Congruent with the glycogen storage capacity theme of Acheson [1,2] reported that a 60-65% carbohydrate diet that was 60% complex carbohydrates, 40% simple carbohydrates, and that was hyperenergetic (~+24% from the weight maintenance diet) resulted in +44 mM increase in triglycerides and a ~165% increase in de novo lipogenesis. The subjects in this study undertook no prior exercise to reduce muscle glycogen [3] reported that overfeeding (ie. 4362 kcal/day for a 73 kg subject, 88% of which was carbohydrate) resulted in 109.5 g of Fat synthesized on day 1 and probably 500g of muscle glycogen synthesized on day 1. So, for the total of 2985.5 kcals of synthesized glycogen or fat ~18% was fat and ~82% was muscle glycogen on day 1 of the massive overfeeding of carbohydrate. Once the glycogen stores were full on day 4 all of the synthesis was fat (163.52 g/day) and none glycogen. It must be stated that prevous exercise was not undertaken in this study and therefore the glycogen stores would have been somewhat "full" to begin the study on day 1.

From the studies of [1-3] we conclude that the effect of carbohydrate on de novo lipogenesis depends on prior exercise and therefore the storage capacity of muscle for carbohydrate ie. The "muscle sink". That is, if you exercise, you reduce muscle glycogen and increase the storage capacity for carbohydrate and thus muscle acts as a "muscle sink" for carbohydrate to be disposed of from

the blood. The excess carbohydrate likely goes to muscle first and liver second. The signals, however, that result in this muscle first phenomenon have yet to be elucidated. The lower insulin during exercise reduces hepatic glucose uptake but not of skeletal muscle glucose uptake as exercise has an insulin like effect on skeletal muscle but not liver [4]. An alternative but less likely hypothesis is that it may be that the increase in glucagon from exercise [5] and possibly the low carbohydrate high fat diet used by Acheson [1] reduces hepatic first pass liver glycogen synthesis (via the hepatic portal vein) in lieu of the skeletal muscle sink as liver but not skeletal muscle is responsive to glucagon [5]. High fructose or fructose related products such as high fructose corn syrup likely induce de novo lipogenesis and non-alcoholic fatty liver disease at a high rate [6]. However, [7] have found that moderate exercise of moderate duration (2 X30 minute bouts in one day) while ingesting a 4-day diet that was 30% fructose (a very high level of fructose) greatly attenuated the rise in triglycerides and associated lipogenic molecules in healthy young subjects. Thus, it appears that exercise is very important in preventing the negative effects of fructose consumption.

With regard to the mechanism for the effect of carbohydrate on adipose tissue storage capacity, hyperinsulinemia resulting from carbohydrate ingestion is the scapegoat for this effect [8]. The Carbohydrate-Insulin Model of Obesity [8] suggests that high insulin resulting from carbohydrate ingestion results in greater body fat deposition. Although, high insulin levels will attenuate lipolysis [9] they may have a stimulatory effect on metabolic rate [10] reported that a variable rate insulin infusion (3.0 mU/kg/min for lean and 6.6 mU/kg/min for obese) and euglycemia resulted in significant ~12% increase in metabolic rate for lean and ~10% increase in metabolic rate for obese. Others [11] have found similar results during euglycemic clamps. Therefore, it appears that insulin has antilipolytic effects but does not influence overall metabolic rate negatively (may increase it when infused with carbohydrate) which is contrary to the Carbohydrate-Insulin Model of Obesity [8] where insulin is proposed to increase body fat storage.

As has been repeatedly shown, exercise reduces insulin concentrations and improves insulin sensitivity [12]. The exact mechanism for this improvement in insulin sensitivity has yet to be elucidated completely. However, roles for synthesis of Glut-4 protein, glycogen degradation and the utilization of intramuscular lipid have been proposed. [13-15].

It is commonly accepted that a sedentary lifestyle combined with a high calorie diet results in a positive energy balance and will lead to obesity and insulin resistance. What is also apparent is that even moderate intensity exercise of moderate duration can activate skeletal muscle lipoprotein lipase [16], reduce muscle glycogen levels [17], and reduce intramuscular triglyceride levels [15] and therefore act as a "muscle sink" to prevent the deleterious effects of these diets.

A hypothesis for an increase in the propensity for obesity in insulin resistant individuals is that insulin resistance must ensue and thus reduces the potential disposal of glucose as muscle glycogen or the oxidation of the glucose by skeletal muscle. The second step is that the circulating glucose has few fates but to go to the liver for de novo lipogenesis to triglyceride or to the fat cell to be deposited in adipocytes. This is supported by [18] who reported that aging reduced muscle glycogen storage (ie. Insulin resistance), increased hepatic de novo lipogenesis, increased hyperlipidemia, and hepatic steatosis. Therefore, a logical intervention to increase insulin sensitivity and dispose of circulating glucose would be regular physical exercise, as the muscle would become a "muscle sink" for glucose disposal.

The key interplay for improvement of glucose and fat uptake by muscle with exercise appears to be between exercise carbohydrate oxidation and post-exercise carbohydrate repletion [19,17]. Wang [19] found that a 1-hour brisk walk improved insulin sensitivity by about 12% in older women when they ingested ~110g of carbohydrate post exercise and 12 hours prior to the euglycemic-clamp procedure. It must be noted that this was only one bout of exercise (lower muscle glycogen concentrations would be seen with daily exercise) and of relatively low intensity (lower muscle glycogen concentrations would be seen with higher intensity). Harrison [17] reported that the ingestion of 367g of carbohydrate (to match energy expenditure during the exercise bout that was 90 min at 70% of maximal oxygen uptake) abolished the improvements in triglyceride synthesis seen when given a high fat meal and 90 min of exercise. Thus, the ability to dispose of glucose or fat by muscle is dependent on the negative carbohydrate balance from previous exercise and lack of dietary replacement. So, it would behoove individuals wishing to improve their insulin sensitivity to determine how many grams of carbohydrate they oxidise during exercise and rest and not replace the full amount of carbohydrate oxidized by carbohydrate replacement ie. Stay in negative carbohydrate balance (and likely negative muscle glycogen balance [3].

The question arises as to how much and what intensity of exercise is enough and how much carbohydrate should be ingested? From three studies [20,7,19] it appears that 60 minutes of moderate intensity (being able to talk during exercise) exercise daily, is the answer. It also appears that a 15-20 minute Cross Fit workout 3 times per week is not enough to improve insulin sensitivity [21]. Thus, one should exercise for 60 minutes daily at a moderate intensity [7, 19-20] or extend the Cross Fit style workout to ~60 minutes 3 times per week. This later recommendation on Cross

Fit requires confirmation with experimental data. With regard to purely resistance exercise these recommendations cannot be made due to the variable nature of the intensity, rest periods, repetition schemes and overall volume of these types of workouts. However, this would appear to provide an area of fertile research potential as the higher the intensity of the resistance exercises the greater the rate of glycogen depletion [22].

Conclusion

Exercise is of paramount importance for reducing the synthesis of triglycerides from carbohydrate, the deposition of fat mass, and improvements in insulin sensitive. Exercise is the only nonpharmacological means for reducing muscle glycogen (which provides storage space for the deposition of glucose and other ingested carbohydrate forms eg. fructose) and this along with the synthesis of Glut-4 proteins provided by regular physical exercise appear to be the operative mechanisms for the reduction in triglyceride synthesis from carbohydrate, the reduced deposition of fat mass from carbohydrate, and the reduction in insulin resistance from carbohydrate. Additionally, diet alone will have little effect if any effect on reducing muscle glycogen concentrations and increasing insulin sensitivity [20] as diet alone without exercise has no effect on muscle glycogen concentrations or Glut-4 protein.

Ethics Approval and Consent to Participate

None

Consent for Publication

Not applicable.

Availability of Data and Materials

There are no supporting data that are not already published.

Competing Interests

The authors declare they have no competing interests.

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Authors' Contributions

Charles Paul Lambert wrote the manuscript. Lori Van Liere obtained articles and edited the manuscript.

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