Relationship Between Reported Carbohydrate Intake and Fasting Blood Glucose

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Abstract

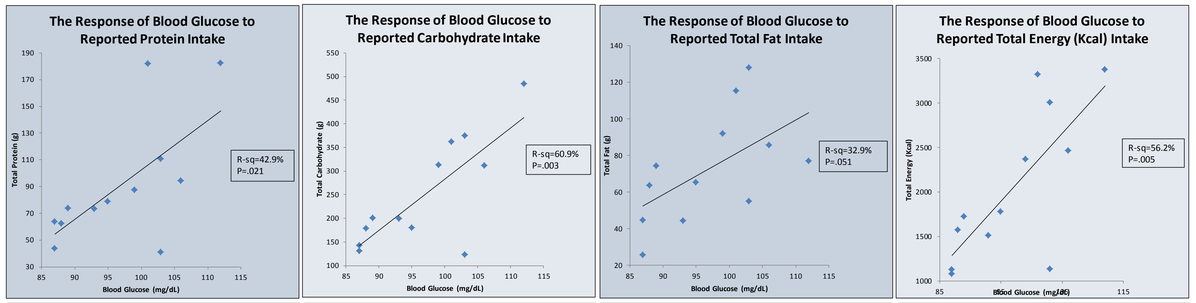
Background: Elevated fasting blood glucose ranges from normal glucose tolerance (under 100 mg/dL) to impaired glucose tolerance (100-125 mg/dL) to diabetes mellitus (above 126 mg/dL). Glucose intolerance is related to the development of chronic diseases, specifically, metabolic syndrome, obesity, type 2 diabetes, and cardiovascular disease. Current National Institutes of Health statistics conclude that an estimated 1 in 6 Americans have insulin resistance. Dietary intake may have a direct influence on glucose metabolism and insulin sensitivity.

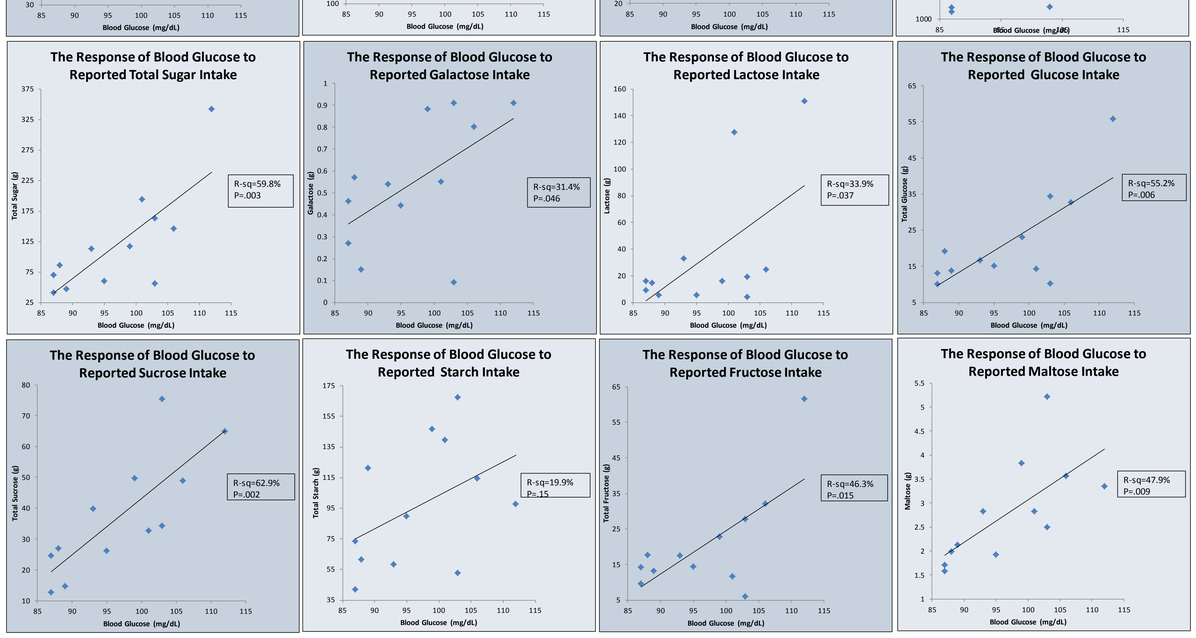
Objective: The purpose of this study was to correlate the result of reported dietary intake to measured fasting blood glucose.

Design: Participants (n=12) selected were a subset of "The Glycemic Effect of Honey" study. Recruited individuals exhibited both normal and impaired glucose tolerance. At baseline, fasting blood glucose levels were determined by Accu-Chek (mg/dL). Participants completed the Diet History Questionnaire II (DHQ II) online and the resultant energy, carbohydrate, protein, fat, total sugar, fructose, glucose, sucrose, galactose, lactose, maltose, and starch were used for comparison. Reported nutrient intake was correlated to blood glucose using the fitted line regression on Minitab (version 15).

Results: The overall reported total energy intake (P=.005) was statistically significantly correlated to fasting blood glucose. The macronutrients, carbohydrate (P=.003), protein (P=.021), and fat (P=.051) were statistically significantly correlated to fasting blood glucose. The sugars, including total sugar (P=.003), fructose (P=.015), glucose (P=.006), sucrose (P=.002), galactose (P=.046), lactose (P=.037), and maltose (P=.009) were statistically significantly correlated to fasting blood glucose. The reported starch intake (P=.15) was not statistically significantly correlated to fasting blood glucose.

Conclusions: The reported energy, carbohydrate, fat, and protein intake correlated to fasting blood glucose levels. Reported total sugar intake including, fructose, glucose, sucrose, galactose, lactose, and maltose also directly correlated to fasting blood glucose levels. The reported starch intake did not correlate with fasting blood glucose levels. The results illustrate a correlation between reported macronutrient and total sugar consumption with fasting blood glucose levels.





Demographic	Total- mean(SE)	Men- mean(SE)	Women- mean(SE)
Age (years)	49.1(3.9)	52.3(4.7)	48.0(5.16)
Body Mass Index (BMI)	26.9(1.4)	28.2(0.6)	26.8(1.8)
Height (cm)	171.1(2.1)	179.9(2.9)	168.2(1.8)
Weight (kg)	79.1(4.4)	91.3(2.7)	75.1(5.1)
N=	12	3	9
Blood Glucose (mg/dL)	97(2.40)	104(3.84)	94(2.46)

Introduction

Glucose intolerance can be associated with serious chronic diseases. The most common are metabolic syndrome, obesity, type 2 diabetes, and cardiovascular disease. Normal glucose tolerance is defined as blood glucose under 100 mg/dL, impaired glucose tolerance is defined as blood glucose of 100-125 mg/dL, and diabetes mellitus is defined as blood glucose of 126 mg/dL or greater. Impaired glucose tolerance is rising around the world and many people are being diagnosed with diabetes.

Materials and Methods

AccuChek* - The use of AccuChek by nursing staff determined blood glucose measured in mg/dL

DHQ II- The food frequency questionnaire was administered by a registered dietitian. The results were based on the participants' diet for the past year.

Conclusion

- Reported higher dietary intake of energy, carbohydrates, protein, fat, total sugar, fructose, glucose, sucrose, galactose, lactose, and maltose correlated with a higher fasting blood glucose level.
- Starch intake did not correlate with fasting blood glucose.

References

Costacou T, Mayer-Davis EJ. Nutrition and prevention of type 2 diabetes. AnnRevNutr 2003;23:147-170

Haffner S. The Metabolic Syndrome: inflammation, diabetes mellitus, and cardiovascular disease. AM J Cardiol 2006;97:3-11

National Diabetes Information. (2011, February). Retrieved August 8, 2011, fromNational DiabetesStatistics, 2011: http://www.diabetes.niddk.nih.gov/dm/pubs/statistics/