Blood glucose control in the diabetic coffee drinker

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Abstract

Introduction: Approximately 9.4% of the US population has diabetes, while about 85% of the US population consumes over a 100mg of caffeine daily. Whereas several epidemiological studies indicate that long-term coffee consumption reduces the risk of Type 2 diabetes mellitus (T2DM), the short-term effect of its consumption on glycemic control and diabetic complications warrants further elucidation. This is the focus of our research inquiry.

Methods: The blood glucose levels of 21 healthy individuals were measured using a glucometer after a carbohydrate-rich meal. For the control trial, each individual's blood glucose levels was taken before the meal and then 15 mins, 45 mins, and 60 mins after eating the meal. For the experimental trial, each individual repeated the control trial but 30 mins after consuming caffeine. The meal was composed of a bagel, cream cheese, and a glucose drink.

Results: There was a significant time-dependent change in blood glucose levels during the control trial, $F(2.980, 59.602) = 21.492$, $p < 0.0005$. There was a statistically significant increase in blood glucose levels with the caffeine trial - $F(1, 20) = 5.830$, $p = 0.025$.

Conclusion: Caffeine consumption with or about meal time acutely impairs glucose tolerance by increasing the rate of spike of blood glucose levels, decreased insulin sensitivity and potentially increases the risk of β-cell dysfunction. In a diabetic patient, this effect could make a tight blood glucose control difficult to achieve therefore requiring higher doses of hypoglycemic drugs and increasing the risk of diabetic complications like diabetic retinopathy, neuropathy or ischemic heart disease.

Introduction

The postprandial spike in blood glucose levels initiates a glucose-dependent secretion of insulin by the pancreatic beta cells which promotes the rapid uptake of glucose by skeletal muscles and adipose tissues consequently re-establishing euglycemia. These responses are impaired in T2DM patients owing to a sustained elevation of counter-regulatory hormones (like glucagon, epinephrine and cortisol) which causes persistent hyperglycemia. Impaired glucose tolerance is worsened by high glycemic index foods which cause a very rapid postprandial spike in blood glucose levels. Caffeine, the major component of coffee is a methylxanthine that has antagonistic properties against adenosine receptors (A1 and A2) and has been shown to cause elevations of plasma epinephrine levels. Consequently, caffeine has the acute effects of promoting lipolysis (increases release of free fatty acids into the bloodstream) and gluconeogenesis. This elevation of epinephrine promotes insulin resistance and in the context of the hyperglycemia and hyperinsulinemia in the immediate postprandial period, how will these counteracting biochemical activities affect the attainment of a good blood glucose control and the development or prevention of diabetic complications in a diabetic coffee drinker? This is the research question that we sought to investigate in this study.

Results

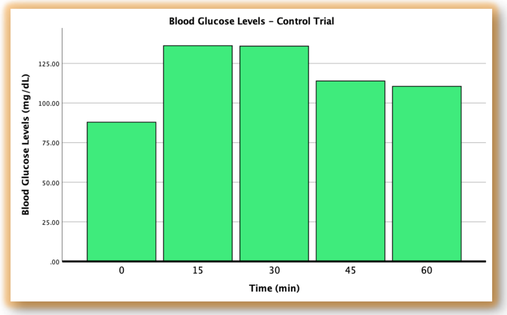

Figure 1. The blood glucose levels changed over time during the control trial, $F(2.980, 59.602) = 21.492$, $p < 0.0005$. The blood glucose levels increase significantly before they gradually decline – the decline is due to the activity of insulin secreted in response to a sudden spike in blood glucose level after food.

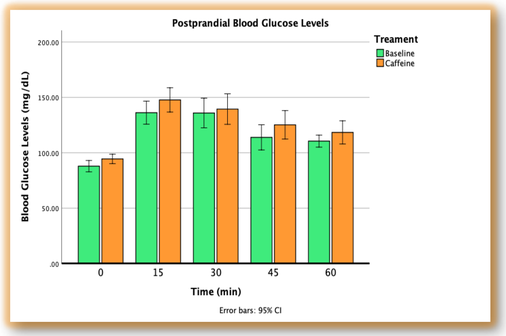

Figure 2. Caffeine significantly increased blood glucose levels over a 60 minute post prandial period - $F(1, 20) = 5.830$, $p = 0.025$.

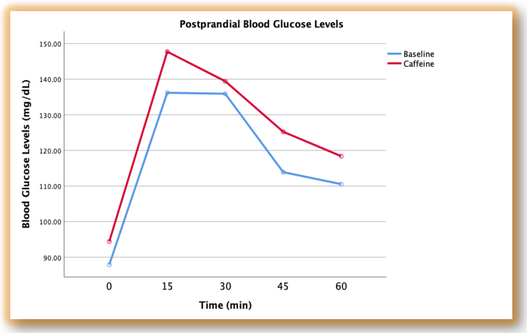

Figure 3. Blood glucose levels peaked at the 15 minute mark which coincides with the peak activity and plasma concentration of caffeine (45 minutes from the time of caffeine consumption). This peak point also indicates the peak of insulin secretion.
Also note that the blood glucose level at the start and end of the experiment is higher for the caffeine trial than baseline

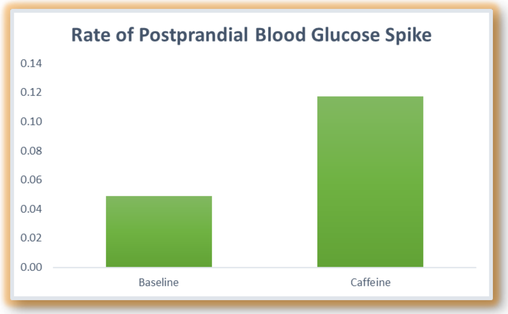

Figure 4. Caffeine consumption with a carbohydrate-rich meal produced a rate of postprandial blood glucose spike that was twice that of the baseline trial. The implication being that the strain on the pancreatic beta cells to secrete insulin will be much higher with caffeine.
Prolonged pancreatic strain potentially induces beta cell apoptosis and insulin deficiency.

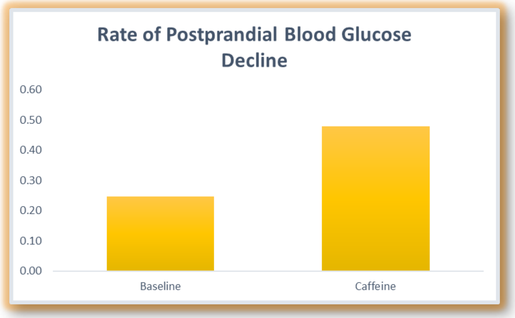

Figure 5. The rate of blood glucose decline from the peak values in the caffeine trial was 2 times higher than the baseline trials. This suggests that caffeine consumption with the meal elicited a much higher level of insulin secretion than the baseline trials.
This wide fluctuation in blood glucose levels poses a huge challenge to maintaining euglycemia.

Methods

The blood glucose of 21 healthy individuals were measured using a glucometer. For the control trial, each individual's blood glucose levels was taken before the meal and then 15 mins, 45 mins, and 60 mins after eating the meal. For the experimental trial, each individual repeated the control trial but only 30min after the consumption of caffeine. The meal was composed of a bagel, cream cheese, and a glucose drink.

Conclusion

As opposed to the findings of several epidemiological studies on the long-term effects of coffee consumption on insulin sensitivity and risk of T2DM, our study suggests that;

1. Caffeine consumption with a carbohydrate-rich meal causes a more rapid spike in blood glucose.
2. Caffeine acutely impairs glucose tolerance necessitation the use of higher doses of oral hypoglycemic in T2DM patients.
3. Caffeine acutely increases insulin secretion but reduces insulin sensitivity. This potentially increases the risk of diabetic complications in T2DM patients.

Future Studies

The following studies will help consolidate our understanding of the clinical implications of coffee consumption in a T2DM patient;

1. An evaluation of the incidence of complications in T2DM coffee drinkers.
2. The acute and long-term effects of the other ingredients found in coffee
3. The actual doses of oral hypoglycemic used to maintain glycemic control in T2DM coffee drinkers.

References

1. B. Keijzers, Bastiaan E. De Galan, Cees J. Tack, and Paul Smits. Caffeine Can Decrease Insulin Sensitivity in Humans. *Diabetes Care*. 2002;25(2): 364-369.
2. Ribeiro JA, *Sebastiao AM*. Caffeine and Adenosine. Journal of Alzheimer's Disease. 2010;20(1): 3-15.
3. Deibert DC, DeFronzo RA: Epinephrine -Induced Insulin Résistance in Man. *J Clin Invest.* 1980;65:717-721.
4. James A. Greenberg, Carol N. Boozer, Allan Geliebter, Coffee, diabetes, and weight control, The American Journal of Clinical Nutrition, 2006;84(4):682–693.

Acknowledgments

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