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REVIEW ARTICLE

Physical Factors of Food Influencing the Postprandial Blood or Plasma Glucose Level: A Narrative Review

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ABSTRACT

Emerging evidence suggests that a high postprandial glucose level in plasma or blood is an important factor for the etiology of non-communicable disorders like the metabolic syndrome, diabetes, obesity and cardiovascular disease. A high sugar content of the food naturally increases the postprandial glucose level. However, quite a few studies provided proof in the past that the physical properties like viscosity, temperature, and water content of the food we consume also may influence the level of this parameter. The aim of this study is to give a narrative review of present findings that showed the physical properties of consumed food influenced the postprandial glucose level.

The online databases Medline, Pubmed, Google Scholar and Hinari have been searched for publications on “plasma glucose” and “temperature” or “viscosity” or “solubility” or “water content”. All articles dealing with the influence on the postprandial glucose level in the blood have been included. Articles written in a language we could not understand or without a proper translation into English have been excluded.

In general, most available studies showed that the physical properties temperature, viscosity, and water content of consumed food influenced the postprandial glucose. An increased temperature, increased viscosity and decreased water content of the food is generally associated with a higher postprandial glucose level in blood or plasma after consumption. Further detailed studies in both preclinical as well as clinical trials should be considered to obtain more detailed results regarding this.

Keywords: Postprandial glucose, temperature, viscosity, water, food

Introduction

The postprandial glucose (PPG) is the level of glucose in plasma or blood after a meal[1]. Accumulating clinical and epidemiologic evidence progressively shows that high postprandial blood or plasma glucose levels determine HbA1c levels[2] and possibly play an important role in the development of chronic disorders like obesity[3], Diabetes mellitus type 2 [2] and cardiovascular disorders[4–6] as well as the complications associated with diabetes[5–8]. These disorders are among the top 5 causes of or risk factors for death worldwide[9,10]. It is therefore important to prevent high PPG levels and consequently reduce its detrimental impact on the health condition and associated rise in morbidity and mortality[11,12].

Several factors may influence the postprandial glucose level in blood or plasma among which are pre-meal glucose level in the blood or plasma[13], the degree of insulin reaction on the meal[14] and most importantly the total amount of complex and simple sugars consumed[15]. All these are biological or chemical factors[16]. Until now, except for viscosity, little attention is paid to physical factors of the food that may be of influence on the PPG level. The absorption of glucose in the gut depends on the rate of digestion of complex sugars and disaccharides in the gut and the absorption with the aid of GLUT or SGLT transporters. The digestion of the sugars is an enzymatic activity, and the transporters also show enzyme-like dynamics. One of the enzyme involved in the digestion of disaccharides to the monosaccharide glucose is α-glucosidase. This enzyme and the SGLT transporters are located on the apical membrane of the epithelial lining of the jejunum, while the glut transporters are located on the basolateral side.

Based on the abovementioned biological structure and functions, we can derive that in addition to the luminal concentration of glucose in the lumen of the jejunum, physical factors that influence the presentation of the sugars to the apical surface of the intestines will also play a role in the absorption of glucose and hence influence the PPG level in blood or plasma. Support for this hypothesis can be found in the following. The optimum temperature for isolated α-glucosidases from nature vary between 40 and 65 degrees Celsius[17–19], which is way above our core body temperature, meaning that an increase in temperature in the normal range within the intestines will increase the activity of the enzyme and consequently accelerate the production of glucose from disaccharides at the luminal membrane. In addition to the abovementioned conditions, proper presentation of the saccharides and glucose to the enzymes and transporters is essential for the absorption of glucose[20]. This is improved when the food is in a more aqueous solution compared to more viscous jelly-like structures. Thus, the solution in which the food is consumed as well as its water or fat content will play a role in the digestion and absorption of the sugars.

The aim of the present study was to find evidence in previously published results that hints towards an effect of the abovementioned physical factors of food on the postprandial glucose. For this purpose, we reviewed relevant databases.

Methods

We searched the Medline, PubMed, Google scholar and HINARI databases for publications containing “plasma glucose AND food temperature”, “plasma glucose AND water”, and “plasma glucose AND viscosity”. All results as well as relevant references they contained, were meticulously evaluated for their content by two investigators. We excluded publications in another language than English and with no detailed descriptions of the methods and results. Finally we described the main findings in the next section.

Results and discussion

The effect of food and environmental temperature on the PPG

We found only a few studies that investigated the effects of temperature of food on the PPG. Nevertheless, solid experimental results show that the PPG in plasma was significantly higher when healthy elderly subjects consumed a glucose solution of 75g/300mL at a temperature of 50 degrees Celsius compared to 5 degrees[21]. Undoubtedly this was based on a lower absorption rate of glucose. In addition, several studies showed that consumption of hot potatoes led to a significantly higher plasma glucose compared to the cooled product[22–24]. In another study investigators found that hot water immersion of the body for at least 45 minutes at 39 degrees Celsius increased the PPG compared to persons being in an ambient temperature of about 23 degrees[25]. Similarly healthy young men and pregnant women showed an increased glucose level during an oral glucose tolerance test at high environmental temperatures compared to lower ambient temperatures[26,27]. In these cases, we may assume that the high ambient temperature increased the core temperature of the body and possibly also the interstitial space of the intestines and thus the food consumed. However, investigators ascribed the higher glucose level to a higher blood flow in arm vessels at high ambient temperatures[28,29]. Apart from these, it seems that

little or no studies investigated the effect of temperature of food in the intestines on the PPG.

The effect of viscosity of food on the PPG

There is abundant evidence that a higher viscosity of the food components leads to a lower PPG[30–32]. A meta-analysis showed that starch with a high amylose content caused significant reductions in postprandial glucose and insulin levels[33]. In addition, soluble dietary fibers reduced the absorption rate of glucose after the consumption of a high carbohydrate load possibly through high viscosity properties[34]. Moreover, numerous studies showed that the amylose and fiber content of food lowered the PPG[33,35–38]. A higher viscosity of food was also favorable for the glucose metabolism (lower insulin and GIP release, and lower desire for food accompanied by a higher degree of fulness), although a slightly higher PPG was found compared to a standard viscosity product in healthy young subjects[39]. Another study with limited data showed that when proteins and vegetables were consumed first, the PPG was significantly lower than when they were consumed after bread and orange juice first [40]. The proteins and vegetables supposedly have a higher viscosity than bread and orange juice[41–43]. In rats it was found that infusion of high viscosity cellulose in the small intestines showed a lower plasma glucose compared to low viscosity fibers[44]. Finally, a high viscosity of the food also delays gastric emptying[39,45], which may itself reduce the PPG[46].

The effect of water content and solubility of food on the PPG

Only a few studies evaluated the effect of water content on the PPG. A relatively recent report showed that individuals who consumed a sweet snack along with water had a faster and higher increase of blood glucose compared to those who consumed the snack alone[47]. Obviously, the water did not add any sugar to the snack, and it must be the change of physical properties that induced the higher PPG. Two older studies already concluded that adding water to a meal increased the glucose response in both healthy and diabetic subjects[48,49], and concluded that the associated faster gastric emptying was responsible for the observations. Investigators recently reported that intrameal consumption of fluid led to a significantly more occurrence of both general and abdominal obesity[50]. However, the previously mentioned study with rats showing a lower plasma glucose with a high viscosity cellulose did not find an effect of water content on the plasma glucose[44].

Summary and conclusion

In the present study we searched for studies that have evaluated the effect of physical properties like temperature, viscosity, and water content of food on the PPG in blood or plasma. We found only a handful studies that investigated the influence of (intestinal) temperature or water content of food on the PPG in plasma or blood. However, the scanty available evidence unanimously concludes that a higher temperature of ingested food or liquids as well as high ambient temperatures increase the plasma glucose compared to lower temperatures[21,23,24,26]. In addition, the few available studies also showed that the PPG increased when food was consumed with an extra amount of water[47–49]. On the contrary a respectable number of studies showed that an increased viscosity of the food reduces the PPG level.

Based on these findings we can conclude that the physical factors temperature, viscosity and water content of food may increase the PPG apart from the amount of sugar already present. It is therefore recommended that these factors are also considered when prescribing diets for patients suffering from Diabetes or Obesity. Furthermore, applying techniques to increase the viscosity or reduce the water content of processed food may decrease their potential to increase the PPG levels[51,52]. Eventually, decreasing the amount of water consumed with meals or the temperature of food and beverages may be beneficial for the glucose metabolism. In this case we should pay attention to the consumption of sugary hot beverages, which pass the stomach fast and are readily available to intestinal digestion and absorption shortly after consumption. In summary, the PPG depends on a more complex interplay of biological, chemical, and physical factors. It is therefore difficult to assess food for their potential diabetes or obesity inducing properties. Future research on these aspects is surely warranted. Who knows, maybe it is better to have the iced rather than the hot version of coffee with cream and sugar.

Conflicts of Interest Statement

The authors have no conflicts of interest to declare.

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References

1. Monnier L, Colette C. Postprandial and basal hyperglycaemia in type 2 diabetes: Contributions to overall glucose exposure and diabetic complications. *Diabetes Metab*. 2015;41(6):6S9-6S15. doi:10.1016/S1262-3636(16)30003-9
2. Bastyr EJ, Stuart CA, Brodows RG, et al. Therapy focused on lowering postprandial glucose, not fasting glucose, may be superior for lowering HbA1c. lOEZ Study Group. *Diabetes Care*. 2000;23(9):1236-1241. doi:10.2337/DIACARE.23.9.1236
3. Blaak EE, Antoine JM, Benton D, et al. Impact of postprandial glycaemia on health and prevention of disease. *Obesity Reviews*. 2012;13(10):923-984. doi:10.1111/J.1467-789X.2012.01011.X
4. Gerich JE. Postprandial Hyperglycemia and Cardiovascular Disease. *Endocrine Practice*. 2006;12(SUPPL. 1):47-51. doi:10.4158/EP.12.S1.47
5. Temelkova-Kurktschiev TS, Koehler C, Henkel E, Leonhardt W, Fuecker K, Hanefeld M. Postchallenge plasma glucose and glycemic spikes are more strongly associated with atherosclerosis than fasting glucose or HbA1c level. *Diabetes Care*. 2000;23(12):1830-1834. doi:10.2337/DIACARE.23.12.1830
6. Yu PC, Bosnyak Z, Ceriello A. The importance of glycated haemoglobin (HbA1c) and postprandial glucose (PPG) control on cardiovascular outcomes in patients with type 2 diabetes. *Diabetes Res Clin Pract*. 2010;89(1):1-9. doi:10.1016/J.DIABRES.2009.12.009
7. Madsbad S. Impact of postprandial glucose control on diabetes-related complications: How is the evidence evolving? *J Diabetes Complications*. 2016;30(2):374-385. doi:10.1016/J.JDIACOMP.2015.09.019
8. Ceriello A, Hanefeld M, Leiter L, et al. Postprandial Glucose Regulation and Diabetic Complications. *Arch Intern Med*. 2004;164(19):2090-2095. doi:10.1001/ARCHINTE.164.19.2090
9. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*. 2012;380(9859):2095-2128. doi:10.1016/S0140-6736(12)61728-0
10. Lin X, Xu Y, Pan X, et al. Global, regional, and national burden and trend of diabetes in 195 countries and territories: an analysis from 1990 to 2025. *Sci Rep*. 2020;10(1):1-11.
11. Peter R, Okoseime OE, Rees A, Owens DR. Postprandial Glucose - A Potential Therapeutic Target to Reduce Cardiovascular Mortality. *Curr Vasc Pharmacol*. 2009;7(1):68-74. doi:10.2174/157016109787354169
12. Lin HJ, Lee BC, Ho YL, et al. Postprandial Glucose Improves the Risk Prediction of Cardiovascular Death Beyond the Metabolic Syndrome in the Nondiabetic Population. *Diabetes Care*. 2009;32(9):1721-1726. doi:10.2337/DC08-2337
13. Landgraf R. The relationship of postprandial glucose to HbA1c. *Diabetes Metab Res Rev*. 2004;20(S2):S9-S12. doi:10.1002/DMRR.517
14. Daly M. Sugars, insulin sensitivity, and the postprandial state. *Am J Clin Nutr*. 2003;78(4):865S-872S. doi:10.1093/AJCN/78.4.865S
15. Kröger J, Siegmund T, Schubert-Olesen O, et al. AGP and Nutrition – Analysing postprandial glucose courses with CGM. *Diabetes Res Clin Pract*. 2021;174:108738. doi:10.1016/J.DIABRES.2021.108738
16. Meyer C, Dostou JM, Welle SL, Gerich JE. Role of human liver, kidney, and skeletal muscle in postprandial glucose homeostasis. *Am J Physiol Endocrinol Metab*. 2002;282(245-2). doi:10.1152/AJPENDO.00032.2001/ASSET/IMAGES/LARGE/H10220688007.JPEG
17. le Chevalier P, van Wormhoudt A. Alpha-glucosidase from the hepatopancreas of the shrimp, Penaus vannamei (Crustacea-Decapoda). *Journal of Experimental Zoology*. 1998;280(6):384-394. doi:https://doi.org/10.1002/(SICI)1097-010X(19980415)280:6<384::AID-JEZ2>3.0.CO;2-J
18. Koster JF, Slee RG. Some properties of human liver acid α-glucosidase. *Biochimica et Biophysica Acta (BBA) - Enzymology*. 1977;482(1):89-97. doi:10.1016/0005-2744(77)90357-6
19. Roig-Zamboni V, Cobucci-Ponzano B, Iacono R, et al. Structure of human lysosomal acid α-glucosidase–a guide for the treatment of Pompe disease. *Nature Communications 2017 8:1*. 2017;8(1):1-10. doi:10.1038/s41467-017-01263-3

20. O'Dea K, Nestel PJ, Antonoff L. Physical factors influencing postprandial glucose and insulin responses to starch. *Am J Clin Nutr*. 1980;33(4):760-765. doi:10.1093/AJCN/33.4.760
21. Kuipers HMM, Jansen RWMM, Peeters TL. The Influence of Food Temperature on Postprandial Blood Pressure Reduction and Its Relation to Substance-P in Healthy Elderly Subjects. *J Am Geriatr Soc*. 1991;39(2):181-184. doi:10.1111/J.1532-5415.1991.TB01623.X
22. Najjar N, Adra N, Hwalla N. Glycemic and insulinemic responses to hot vs cooled potato in males with varied insulin sensitivity. *Nutrition Research*. 2004;24(12):993-1004. doi:10.1016/J.NUTRES.2004.09.002
23. Patterson MA, Fong JN, Maiya M, et al. Chilled Potatoes Decrease Postprandial Glucose, Insulin, and Glucose-dependent Insulinotropic Peptide Compared to Boiled Potatoes in Females with Elevated Fasting Glucose and Insulin. *Nutrients 2019, Vol 11, Page 2066*. 2019;11(9):2066. doi:10.3390/NU11092066
24. Fong JVN, Miketinas D, Moore LW, et al. Precision Nutrition Model Predicts Glucose Control of Overweight Females Following the Consumption of Potatoes High in Resistant Starch. *Nutrients 2022, Vol 14, Page 268*. 2022;14(2):268. doi:10.3390/NU14020268
25. Leicht CA, James LJ, Briscoe JHB, Hoekstra SP. Hot water immersion acutely increases postprandial glucose concentrations. *Physiol Rep*. 2019;7(20):e14223. doi:10.14814/PHY2.14223
26. Shen EX, Moses RG, Oats JJN, Lowe J, McIntyre HD. Seasonality, temperature and pregnancy oral glucose tolerance test results in Australia. *BMC Pregnancy Childbirth*. 2019;19(1):263. doi:10.1186/S12884-019-2413-5/TABLES/7
27. Dumke CL, Slivka DR, Cuddy JS, Hailes WS, Rose SM, Ruby BC. The Effect of Environmental Temperature on Glucose and Insulin After an Oral Glucose Tolerance Test in Healthy Young Men. *Wilderness Environ Med*. 2015;26(3):335-342. doi:10.1016/J.WEM.2015.03.002
28. Frayn KN, Whyte PL, Benson HA, Earl DJ, Smith HA. Changes in forearm blood flow at elevated ambient temperature and their role in the apparent impairment of glucose tolerance. *Clin Sci*. 1989;76(3):323-328. doi:10.1042/CS0760323
29. Akanji AO, Bruce M, Frayn K, Hockaday TDR, Kaddaha GM. Oral glucose tolerance and ambient temperature in non-diabetic subjects. *Diabetologia*. 1987;30(6):431-433. doi:10.1007/BF00292547/METRICS
30. Reppas C, Dressman JB. Viscosity modulates blood glucose response to nutrient solutions in dogs. *Diabetes Res Clin Pract*. 1992;17(2):81-88. doi:10.1016/0168-8227(92)90153-I
31. Reppas C, Adair CH, Barnett JL, et al. High viscosity hydroxypropylmethylcellulose reduces postprandial blood glucose concentrations in NIDDM patients. *Diabetes Res Clin Pract*. 1993;22(1):61-69. doi:10.1016/0168-8227(93)90133-P
32. Heaton KW, Marcus SN, Emmett PM, Bolton CH. Particle size of wheat, maize, and oat test meals: effects on plasma glucose and insulin responses and on the rate of starch digestion in vitro. *Am J Clin Nutr*. 1988;47(4):675-682. doi:10.1093/AJCN/47.4.675
33. Cai M, Dou B, Pugh JE, Lett AM, Frost GS. The impact of starchy food structure on postprandial glycemic response and appetite: a systematic review with meta-analysis of randomized crossover trials. *Am J Clin Nutr*. 2021;114(2):472-487. doi:10.1093/AJCN/NQAB098
34. Behall KM, Scholfield DJ. Food Amylose Content Affects Postprandial Glucose and Insulin Responses. *Cereal Chem*. 2005;82(6):654-659. doi:10.1094/CC-82-0654
35. Behall KM, Scholfield DJ. Food Amylose Content Affects Postprandial Glucose and Insulin Responses. *Cereal Chem*. 2005;82(6):654-659. doi:10.1094/CC-82-0654
36. Jenkins AL, Jenkins DJA, Wolever TMS, et al. Comparable Postprandial Glucose Reductions with Viscous Fiber Blend Enriched Biscuits in Healthy Subjects and Patients with Diabetes Mellitus: Acute Randomized Controlled Clinical Trial. *Croat Med J*. 2008;49(6):772. doi:10.3325/CMJ.2008.49.722
37. Yoshimoto J, Kato Y, Ban M, et al. Palatable Noodles as a Functional Staple Food Made Exclusively from Yellow Peas Suppressed Rapid Postprandial Glucose Increase. *Nutrients 2020, Vol 12, Page 1839*. 2020;12(6):1839. doi:10.3390/NU12061839
38. Shanita SN, Hasnah H, Khoo CW. Amylose and Amylopectin in Selected Malaysian

Foods and its Relationship to Glycemic Index (Amilosa dan Amilopektin dalam Makanan Malaysia Terpilih dan Kaitannya dengan Indeks Glisemik). *Sains Malays*. 2011;40(8):865-870.

39. Zhu Y, Hsu WH, Hollis JH. The Impact of Food Viscosity on Eating Rate, Subjective Appetite, Glycemic Response and Gastric Emptying Rate. *PLoS One*. 2013;8(6):e67482. doi:10.1371/JOURNAL.PONE.0067482
40. Shukla AP, Iliescu RG, Thomas CE, Aronne LJ. Food Order Has a Significant Impact on Postprandial Glucose and Insulin Levels. *Diabetes Care*. 2015;38(7):e98-e99. doi:10.2337/DC15-0429
41. Alsuhaibani AMA. Rheological and Nutritional Properties and Sensory Evaluation of Bread Fortified with Natural Sources of Calcium. *J Food Qual*. 2018;2018. doi:10.1155/2018/8308361
42. Imai S, Fukui M, Ozasa N, et al. Eating vegetables before carbohydrates improves postprandial glucose excursions. *Diabetic Medicine*. 2013;30(3):370. doi:10.1111/DME.12073
43. Bae JH, Kim LK, Min SH, Ahn CH, Cho YM. Postprandial glucose-lowering effect of premeal consumption of protein-enriched, dietary fiber-fortified bar in individuals with type 2 diabetes mellitus or normal glucose tolerance. *J Diabetes Investig*. 2018;9(5):1110-1118. doi:10.1111/JDI.12831
44. Takahashi T, Karita S, Ogawa N, Goto M. Crystalline Cellulose Reduces Plasma Glucose Concentrations and Stimulates Water Absorption by Increasing the Digesta Viscosity in Rats. *J Nutr*. 2005;135(10):2405-2410. doi:10.1093/JN/135.10.2405
45. Jin Y, Wilde PJ, Hou Y, Wang Y, Han J, Liu W. An evolving view on food viscosity regulating gastric emptying. *https://doi.org/101080/104083982021 2024132*. 2022. doi:10.1080/10408398.2021.2024132
46. Vuksan V, Jenkins AL, Dias AG, et al. Reduction in postprandial glucose excursion and prolongation of satiety: possible explanation of the long-term effects of whole grain Salba (Salvia Hispanica L.). *European Journal of Clinical Nutrition 2010 64:4*. 2010;64(4):436-438. doi:10.1038/ejcn.2009.159
47. Bipat R, Toelsie JR. Drinking water with consumption of a jelly filled doughnut has a time dependent effect on the postprandial blood glucose level in healthy young individuals. *Clin Nutr ESPEN*. 2018;27:20-23. doi:10.1016/j.clnesp.2018.07.007
48. Torsdottir I, Andersson H. Effect on the postprandial glycaemic level of the addition of water to a meal ingested by healthy subjects and Type 2 (non-insulin-dependent) diabetic patients. *Diabetologia*. 1989;32(4):231-235. doi:10.1007/BF00285289/METRICS
49. Sievenpiper JL, Vuksan V, Wong EYY, Mendelson RA, Bruce-Thompson C. Effect of Meal Dilution on the Postprandial Glycemic Response: Implications for glycemic testing. *Diabetes Care*. 1998;21(5):711-716. doi:10.2337/DIACARE.21.5.711
50. Salari-Moghaddam A, Aslani N, Saneei P, et al. Water intake and intra-meal fluid consumption in relation to general and abdominal obesity of Iranian adults. *Nutr J*. 2020;19(1):1-8. doi:10.1186/S12937-020-00551-X/TABLES/3
51. Kong H, Yu L, Gu Z, et al. An Innovative Short-Clustered Maltodextrin as Starch Substitute for Ameliorating Postprandial Glucose Homeostasis. *J Agric Food Chem*. 2021;69(1):354-367. doi:10.1021/ACS.JAFC.0C02828/SUPPL_FILE/JF0C02828_SI_001.PDF
52. Xiu A, Zhou M, Zhu B, Wang S, Zhang J. Rheological properties of Salecan as a new source of thickening agent. *Food Hydrocoll*. 2011;25(7):1719-1725. doi:10.1016/J.FOODHYD.2011.03.013