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ORIGINAL RESEARCH

Glycemic index, postprandial glycemia, and curve shape in healthy subjects: analysis of a database of over 1,000 foods - Review of the evidence

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1. ABSTRACT

The analyzed study examined the glycemic response following the consumption of 1,126 carbohydrate-containing foods, evaluated using a standardized methodology in small groups of healthy subjects. The main finding is that a higher glycemic index was strongly associated with higher glucose peaks and wider glycemic oscillations in the early stages after the meal. The research did not evaluate habitual diets, insulin requirements, oxidative stress, inflammation, or vascular damage.In the section "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, the study contributes primarily to the point that the quality of carbohydrates modifies the magnitude of repeated glycemic excursions. Independent evidence shows that greater glycemic variability is associated with markers of oxidative stress and, in controlled experimental models, with a transient worsening of endothelial function. A meta-analysis of clinical studies also indicates small improvements in glycemic control with low glycemic index or low glycemic load dietary patterns in people with diabetes. However, a controlled trial in adults without diabetes did not find consistent benefits on cardiovascular risk factors: the conclusion therefore requires caution and clinical context.

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MISSION

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Through articles, reviews, in-depth analyses and expert contributions, the Journal champions evidence-based medicine and science, fostering a correct interpretation of scientific data and countering misinformation. Every piece of content is developed with the aim of translating the complexity of research into understandable, useful and applicable information, while keeping intact the principles of accuracy, transparency and scientific independence.

Free from political, industrial and commercial influence, the journal stands as an authoritative and independent editorial space, dedicated to promoting scientific culture, professional development and the growth of health awareness among the public.

2. ANALYZED STUDY

The research question was whether the glycemic index (GI)—that is, the measure of how quickly a carbohydrate-containing food raises glycemia compared to a reference glucose—can truly describe the shape of the glycemic response after a meal. Specifically, the authors evaluated whether a higher GI corresponded to a faster and greater increase in blood glucose.The relevance to the section "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi therefore concerns a precise point: foods with carbohydrates of different metabolic quality can produce different glycemic spikes and fluctuations, even before considering the size of the portion consumed. The study documents this acute segment of the chain, namely glycemic index of the food height and amplitude of the postprandial glycemic response.However, it cannot demonstrate that a low-GI diet reduces oxidative stress, inflammation, deterioration of endothelial function, or cardiovascular risk in the long term. These outcomes were not measured in the publication subject to the Evidence Review.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The authors analyzed a database consisting of 1,126 foods. Each food had been tested using a standardized GI methodology in groups of 8-12 healthy subjects. The GI was calculated by comparing the incremental area under the glycemic curve—that is, the overall increase in blood glucose after consuming the food—with the response obtained after an equivalent amount of oral glucose.For each food, absolute and incremental glucose concentrations were compared with the GI at different time points. The authors also constructed average curves for low GI foods (55 or less), medium GI foods (56 to 69), and high GI foods (70 or more) within the main food categories.The analyses used Spearman correlations, a measure indicating how two variables tend to vary in the same direction, without demonstrating a causal relationship. Age, sex, detailed clinical characteristics of the participants, inclusion and exclusion criteria, the data collection period, and further statistical details are not reported in the available material, which consists only of the abstract.

4. RESULTS

In simple terms, foods with a higher GI tended to produce a higher initial increase in glucose and greater fluctuations compared to pre-meal values. The strongest association concerned the incremental glucose peak, with a Spearman correlation of r=0.76, and the absolute peak; both results had P<0.001, making them highly unlikely to be due to chance given the statistical model used.The GI was also strongly associated with glycemia at 60 minutes: r=0.70 for the incremental value and r=0.66 for the absolute value. The maximum amplitude of glycemic excursion also increased as the GI increased. These results indicate that the GI summarizes quite well how quickly and how intensely glycemia rises in the early stages after food ingestion.The relationship with glycemia at 120 minutes was weak: r=0.20 for the incremental data and r=0.16 for the absolute data, albeit with P<0.001. This means that the GI is more informative for the glycemic peak or near-peak than for the value reached two hours after the meal. Within food categories, low, medium, and high GI foods differed significantly in terms of mean GI, glycemia at 30 and 60 minutes, and maximum excursion amplitude, with P<0.001.The most relevant result for the book is therefore directional and specific: a higher GI is associated with higher postprandial glycemic peaks and wider glycemic excursions. No results regarding insulin, glycemic variation throughout the day, insulin therapy, reactive oxygen species, or blood vessel function are reported in the available material.

5. INTERPRETATION

From a biological perspective, the result suggests that the GI is not just a theoretical number assigned to foods: it describes quite accurately a food's tendency to generate a blood glucose spike in healthy individuals. A higher spike does not automatically equate to harm, because the postprandial response is physiological. However, if these excursions are large and repeated in people with impaired glucose metabolism, they can represent a significant metabolic stimulus.The publication subject to the Evidence Review therefore supports the starting point of the book's narrative: the physiological quality of carbohydrates modifies the blood glucose trend after a meal. This data alone does not prove the subsequent steps, namely that repeated

oscillations increase oxidative stress, activate inflammatory processes, or deteriorate the endothelium, the layer of cells that lines the inside of blood vessels and helps regulate their function.It is also important to distinguish the GI from the glycemic load. The GI describes the response relative to a standardized amount of available carbohydrates; the glycemic load also incorporates the quantity actually consumed. Therefore, a high-GI food consumed in a small portion and an intermediate-GI food consumed in a large portion can have a different overall impact on glycemia.

6. INDEPENDENT SUPPORTING EVIDENCE

A first independent study examined whether daily glycemic fluctuations are associated with oxidative stress in people with type 2 diabetes. In a case-control study involving 21 people with diabetes and 21 age- and sex-matched controls, the mean amplitude of glycemic excursions, measured via continuous monitoring, was positively associated with the urinary excretion of 8-iso-prostaglandin F2a, an indicator of oxidative stress. The association with the amplitude of excursions was strong, r=0.86 and P<0.001, and persisted after adjustment for other indicators of glycemic control. This confirms, at an observational level and in people with diabetes, the link between greater glycemic instability and increased oxidative stress, but it does not prove that dietary GI is the direct cause of such instability [1].A second study addressed the next step with a controlled experiment. Twenty-seven people with newly diagnosed type 2 diabetes and 22 healthy subjects were exposed to either constantly high or oscillating blood glucose conditions using a glycemic clamp, a technique that allows for external regulation of blood glucose. Oscillations between baseline values and 15 mmol/L every six hours for 24 hours produced a greater worsening of flow-mediated dilation, an indicator of endothelial function, and a greater increase in markers of oxidative stress compared to blood glucose levels maintained constantly at 10 or 15 mmol/L. In subjects with diabetes, the excretion of 8-iso-prostaglandin F2a was 536±51 pg/mg of creatinine during the oscillating profile, compared to 476±48 with constant blood glucose at 15 mmol/L and 432±47 with constant blood glucose at 10 mmol/L. The experimental design strengthens the biological link, but the duration was short and the model does not reproduce habitual eating [2].To link GI to glycemic management over time, a systematic review with meta-analysis brought together 27 randomized trials, covering 29 comparisons and 1,617 participants with type 1 or type 2 diabetes. Compared to diets with higher GI or glycemic load, low GI or low glycemic load patterns reduced glycated hemoglobin, an indicator of average blood glucose over the previous months, by 0.31 percentage points; 95% confidence interval from –0.42 to –0.19; P<0.001. Reductions in fasting glycemia, LDL cholesterol, triglycerides, body weight, and C-reactive protein, an inflammatory marker, were also observed. The average effect on blood glucose was small but considered clinically relevant by the authors; however, heterogeneity for glycated hemoglobin was substantial, with I^2=75%, signaling differences between the included studies [3].Overall, these publications build a convergent but incomplete chain in humans: higher GI foods tend to generate larger spikes; wider glycemic fluctuations are associated with greater oxidative stress; experimental oscillations can transiently worsen endothelial function; low GI or glycemic load dietary patterns modestly improve some glycemic indicators in people with diabetes [1,2,3]. It remains unproven that reducing GI alone directly prevents clinical cardiovascular events.

7. CONFLICTING EVIDENCE

A controlled crossover trial with all food provided to participants showed that the effect of GI depends on the dietary context and the population studied. One hundred and sixty-three overweight adults, with systolic blood pressure between 120 and 159 mmHg but without known diabetes, followed four DASH-style diets for five weeks each, with varying amounts of carbohydrates and different GI.When the diet was high in carbohydrates, the low-GI version compared to the high-GI version reduced insulin sensitivity by 20%, from 8.9 to 7.1 units, with P=0.002, and increased LDL cholesterol by 6%, from 139 to 147 mg/dL, with P≤0.001. When carbohydrate intake was lower, reducing the GI did not change the primary outcomes, except for a 5% reduction in triglycerides. In the main comparison, the low-GI and low carb diet did not improve insulin sensitivity, systolic blood pressure, or LDL and HDL cholesterol compared to the high-GI and high-carbohydrate diet; however, it did reduce

triglycerides by 23% [4].This result does not contradict the acute data from the original study, according to which a higher GI predicts higher glycemic peaks. Instead, it scales back the idea that lowering the GI, in isolation and in every context, necessarily produces improvements in cardiovascular risk factors or insulin sensitivity. Differences between healthy individuals and those with diabetes, total carbohydrate amount, diet composition, duration of the intervention, and dietary adherence are decisive factors. No directly conflicting evidence sufficiently relevant to the relationship between food GI and the height of the glycemic peak was identified; this does not prove the absence of contrary results.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the acute data on foods, which is the subject of the Evidence Review, from the independent evidence on the subsequent stages of the book's narrative.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Experimental database analysis	1,126 foods tested in groups of 8-12 healthy subjects	Higher GI associated with higher glycemic spikes and greater excursion amplitude	direct confirmation	moderate
[1]	Observational case-control	21 people with type 2 diabetes and 21 controls	Greater amplitude of glycemic excursions associated with higher oxidative stress	indirect confirmation	moderate
[2]	Experimental study with glycemic clamp	27 people with type 2 diabetes and 22 healthy subjects	Fluctuating glycemia associated with higher oxidative stress and worse endothelial function compared to constant glycemia	biologically consistent evidence	moderate
[3]	Systematic review and meta-analysis of RCTs	1,617 people with type 1 or 2 diabetes	Low GI or glycemic load diets associated with an average reduction in glycated hemoglobin of 0.31 percentage points	indirect confirmation	high
[4]	Controlled crossover RCT	163 overweight adults without known diabetes	Reducing GI did not consistently improve insulin sensitivity or cardiovascular risk factors	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The study analyzed convincingly shows that the glycemic index is a good indicator of the initial glycemic response to a food: higher values generally correspond to higher glucose spikes and wider fluctuations. This supports the first part of the book's narrative, but does not in itself demonstrate vascular damage or clinical benefits of low-GI diets.Independent evidence makes the link between greater glycemic variability, oxidative stress, and transient worsening of endothelial function biologically plausible. In people with diabetes, low-GI or low-glycemic load dietary patterns are associated with small improvements in glycemic control. The effect on cardiovascular risks and the superiority of the GI alone compared to an overall dietary pattern remain, however, to be further validated.

Aggiornato al 17/11/2026. Questo contenuto riflette una sintesi divulgativa delle evidenze scientifiche al momento della pubblicazione e non sostituisce il parere medico.

10. TRANSPARENCY AND SCIENTIFIC RESPONSIBILITY

Artificial Intelligence was used solely as a tool for editorial and documentary support. All content, interpretations, bibliographic citations and conclusions have been verified, reviewed and approved by the author, who assumes full scientific responsibility for them.

The manuscript was also subjected to independent peer review prior to publication.

To ensure the greatest transparency, the complete history of the article, including the dates of submission, revision, acceptance and publication, as well as information on the peer review process, is available on the dedicated Article History page.

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