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ORIGINAL RESEARCH

Loss of postprandial glycemic control precedes the gradual deterioration of fasting glycemia as diabetes worsens - Evidence review

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1. ABSTRACT

The original study analyzed 130 people with type 2 diabetes using 24-hour continuous glucose monitoring. By comparing groups defined by glycated hemoglobin (HbA1c, an indicator of average blood sugar over the previous months), the authors observed that the average increase in daytime blood sugar after meals became evident before the persistent increase in nighttime fasting glycemia.In the narrative of “The Choice to Live in Health or Illness” by Dr. A. Colonnese and R. Panzironi, the study contributes to the passage stating that alterations in blood sugar after a carbohydrate load can appear before abnormalities in fasting glycemia. Independent prospective studies show that high blood sugar one hour after a standard glucose load can predict a higher future risk of diabetes, even in people with normal fasting glycemia. However, other longitudinal data indicate different metabolic pathways across populations and do not allow for a universal "postprandial-first, fasting-later" sequence to be assumed.

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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, in type 2 diabetes, the worsening of post-meal glycemia and fasting glycemia occurs simultaneously or sequentially. The authors' hypothesis was that glycemic control deteriorates initially during the daytime hours following meals, then in the morning, and finally during overnight fasting.The relevance to the book's section is specific: the publication does not prove that high-glycemic index foods cause diabetes, nor does it directly measure glycemic variability in prediabetes. Instead, it documents, in people who already had type 2 diabetes, a cross-sectional sequence in which the average increase in post-meal glycemia was detectable in groups with lower HbA1c compared to the stable increase in nocturnal glycemia. This is the segment of the narrative that it can support.Since this is a comparison between groups observed at a single point in time, the study describes a gradation associated with poorer metabolic control but does not follow the same individuals over time. Therefore, it cannot prove with certainty that every single patient necessarily goes through that same sequence.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The study included 130 people with type 2 diabetes. Participants were divided into five groups based on their HbA1c levels: below 6.5% (30 people), 6.5–6.9% (17 people), 7.0–7.9% (32 people), 8.0–8.9% (25 people), and at least 9.0% (26 people). A higher HbA1c value indicates, on average, greater blood exposure to glucose over the preceding months.For each participant, 24-hour glycemic profiles were obtained using a continuous glucose monitoring system, a device that repeatedly records glucose concentration in the interstitial fluid, which is closely correlated with blood glucose levels. The authors compared average glucose concentrations during postprandial daytime periods, the morning period associated with the dawn phenomenon, and the nocturnal fasting period.The dawn phenomenon refers to the rise in glycemia in the early morning hours, driven by hormonal changes that increase glucose availability. The available material does not report inclusion and exclusion criteria, duration of diabetes, therapies used, age and sex composition, diet during monitoring, recruitment period, clinical endpoints, or further details of the statistical analyses.

4. RESULTS

The overall result was a progressive increase in glucose concentrations moving from the groups with lower HbA1c to those with higher HbA1c. Simply put, poorer long-term glycemic control was associated with higher values throughout the day, but the different times of day did not all change at the same HbA1c level.The first statistically significant difference appeared for daytime periods after meals: average glycemia was 6.4 mmol/L in the group with HbA1c below 6.5% and 7.7 mmol/L in the group with HbA1c 6.5-6.9% (P=0.0004). This means that, when comparing these two groups, average post-meal glycemia was higher in the group with slightly higher HbA1c.The second significant change concerned the morning period: 7.5 mmol/L in the group with HbA1c 6.5-6.9% compared to 9.3 mmol/L in the group with HbA1c 7.0-7.9% (P=0.0003). Finally, the significant difference in the nocturnal fasting period appeared between the group with HbA1c 7.0-7.9% and the one with HbA1c 8.0-8.9%: 6.3 and 8.4 mmol/L, respectively (P<0.0001).The authors therefore interpreted the data as a three-step progression: first an increase in daytime postprandial glycemia, then higher morning glycemia, and finally sustained nocturnal hyperglycemia. The relevant finding for the book is the observed direction: in the groups with lower HbA1c, the alteration in average post-meal glycemia was already present when nocturnal fasting glycemia did not yet show the significant difference detected in the subsequent groups.

5. INTERPRETATION

In lay terms, the work suggests that in existing type 2 diabetes, the body may begin to struggle with managing glucose from meals before it starts maintaining high glycemia throughout the night. This is biologically plausible because post-meal glucose management requires a rapid insulin response, good muscle sensitivity to insulin, and an adequate reduction in glucose production by the liver.However, the result does not equate to proof that glycemic oscillations cause diabetes. The study compared average concentrations across time slots; it did not directly quantify the amplitude and frequency of the

oscillations, nor did it study subjects before their diagnosis. Furthermore, higher HbA1c levels may reflect many clinical differences not reported in the available material, including disease duration, therapy, residual pancreatic beta-cell function, and dietary habits.Compared to the statement in the book, the documented finding is narrower but recognizable: greater postprandial glycemic exposure may appear in a less advanced stage of deterioration compared to nocturnal fasting hyperglycemia. The aspect not directly documented concerns the dietary origin of this change, its presence before the onset of overt diabetes, and the fact that it represents the first signal in every individual.

6. INDEPENDENT SUPPORTING EVIDENCE

Independent confirmation of the early value of glycemia after a glucose load comes from the San Antonio Heart Study. In 1,611 people without diabetes at baseline, followed for 7-8 years, glycemia one hour after an oral glucose load of more than 155 mg/dL identified a higher future risk of diabetes, even among people with normal glucose tolerance. In this subgroup, the risk of developing diabetes was 15.3% with one-hour glycemia above the threshold and 2.9% below the threshold; the difference was statistically significant. The one-hour measurement is not identical to glycemia after a regular meal or continuous monitoring, but it confirms that an early alteration in the glucose response can be informative when fasting glycemia is not necessarily altered [1].The Whitehall II study added a longitudinal perspective. In 6,538 adults initially without diabetes, 505 developed diabetes during a median follow-up of 9.7 years. In future cases, both fasting glycemia and two-hour post-load glycemia showed a rapid increase in the three years preceding the diagnosis. Two-hour glycemia rose from 7.60 to 11.90 mmol/L, while fasting glycemia increased from 5.79 to 7.40 mmol/L. This result confirms that post-load abnormalities are part of the trajectory toward diabetes, but it does not confirm a clear temporal precedence of postprandial glycemia: in the final acceleration, both measures worsened [2].Taken together, this evidence supports the relevance of glycemic measurements obtained after a glucose load and shows that fasting glycemia alone may not capture all people at risk. However, the level of confirmation remains observational: the data associate higher post-load values with a greater future probability of diabetes, without demonstrating that the selective reduction of such values necessarily prevents the progression of the disease.

7. CONFLICTING EVIDENCE

There is relevant data suggesting that the sequence proposed in the original study should not be considered universal. In a longitudinal analysis of the Whitehall II cohort, conducted on 120 South Asian participants and 867 white participants who developed type 2 diabetes, South Asian participants showed a more rapid increase in fasting glycemia before diagnosis compared to white participants, while two-hour post-load glycemia trajectories did not differ between the groups. The difference in the slope of the increase in fasting glycemia was 0.22 mmol/L per decade, with a 95% confidence interval from 0.02 to 0.42 and P=0.03 [3].These results do not deny that post-meal alterations can be early or prognostically relevant. However, they show that metabolic history can depend on the population, insulin sensitivity, and the ability of pancreatic beta cells to compensate. Consequently, the relationship between postprandial glycemia and fasting glycemia should not be interpreted as a rigid path identical for everyone.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication under review from independent evidence and indicates how much each contributes to the specific step of the narrative.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Cross-sectional study with continuous glucose monitoring	130 people with type 2 diabetes	The average increase in glycemia after meals appeared among groups with lower HbA1c compared to the significant increase in nocturnal fasting glycemia.	direct confirmation	moderate
[1]	Prospective observational	1,611 adults without diabetes, San Antonio Heart Study	Glycemia at one hour greater than 155 mg/dL during a load test was associated with a higher future incidence of diabetes, even with normal glucose tolerance.	indirect confirmation	moderate
[2]	Longitudinal prospective cohort	6,538 adults initially without diabetes; 505 incident cases	Fasting glycemia and two-hour post-load glycemia increased rapidly in the three years prior to diabetes diagnosis.	biologically consistent evidence	moderate
[3]	Longitudinal prospective cohort	120 South Asian and 867 white participants with incident diabetes	In South Asian participants, fasting glycemia increased more rapidly, with no differences in two-hour glycemia trajectories.	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The study analyzed directly supports the observation that, in type 2 diabetes, the average increase in glycemia after meals may emerge before fasting nocturnal hyperglycemia across groups with increasing HbA1c. This evidence is moderately supported by prospective studies in which elevated glycemic values after a glucose load predicted a higher future probability of diabetes.However, this conclusion requires caution. The work does not demonstrate that all subjects follow the same sequence, does not directly measure the causal role of specific foods, and does not prove that postprandial glycemic variability alone is the first universal sign of the disease. The narrative is well-founded as a mechanism and observational association, but it needs to be contextualized within the individual variability of metabolic progression.

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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