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The consumption of caffeinated coffee and a carbohydrate-rich meal influences the postprandial metabolism of a subsequent oral glucose tolerance test in healthy young men - Evidence review

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1. ABSTRACT

The original study evaluated whether caffeinated coffee, consumed with a carbohydrate-rich meal, could influence the response to a second glucose load three hours later. In a randomized crossover design, ten healthy young men received caffeinated coffee, decaffeinated coffee, or water along with high-glycemic index cereals. After the subsequent oral test with 75 g of glucose, the caffeinated coffee group showed a higher insulin response, greater blood glucose exposure, and a lower insulin sensitivity index.In paragraph 4.16 of “The Choice to Live in Health or Illness” by Dr. A. Colonnese and R. Panzironi, the publication contributes to the narrative that caffeine can acutely interfere with insulin action. Independent studies confirm that caffeine can temporarily reduce insulin sensitivity in healthy individuals. However, the original study did not measure cortisol, and the available evidence does not allow us to consider it proven that cortisol-mediated interference is the predominant mechanism.

ONLINE ARTICLE

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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 caffeinated coffee, consumed simultaneously with a first carbohydrate-rich meal, could alter the body's ability to manage a second glucose load three hours later. The hypothesis evaluated was that the effect of caffeine does not end with the first meal, but persists into the subsequent phase, increasing the demand for insulin and worsening glycemic regulation.This question is relevant to section 4.16 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi because it documents, in healthy young men, a specific segment of the narrative: after consuming caffeinated coffee and carbohydrates, the insulin response to a subsequent glucose load can increase without improving glycemia. The study can therefore show an acute alteration in insulin sensitivity, that is, a lower efficiency with which insulin promotes glucose utilization. However, it cannot demonstrate that this alteration depends on cortisol, as cortisol is not reported among the variables measured in the available material.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The study used a randomized crossover design: each of the ten participants received all three treatments at different times and in random order. The participants were young, healthy men. Their exact age, habitual caffeine consumption, inclusion and exclusion criteria, the washout period between trials, and the randomization method are not reported in the available material.In one condition, participants consumed caffeinated coffee at a dose of 5 mg per kg of body weight; in the others, they received decaffeinated coffee or an equal volume of water. The beverages were ingested along with high-glycemic index whole grains, i.e., foods capable of rapidly raising blood glucose. Three hours later, an oral glucose tolerance test with 75 g of glucose was performed.The outcomes included the area under the curve, or AUC, for glucose and insulin: this indicator summarizes how much glucose or insulin remains in the blood over a defined period. An insulin sensitivity index was also calculated. The laboratory analytical procedures, the instruments used, and the detailed statistical plan are not reported in the available material.

4. RESULTS

After the first meal, the area under the insulin curve and the insulin sensitivity index did not differ between the three conditions. The area under the glucose curve, however, was greater both after caffeinated coffee, 107 mmol/l × 3 hours with a standard error of 18, and after decaffeinated coffee, 74 mmol/l × 3 hours with a standard error of 15, compared to water, −0.2 mmol/l × 3 hours with a standard error of 29; the differences compared to water were statistically significant, with P less than 0.05.The result most relevant to the book's section emerged after the second glucose load. After caffeinated coffee, the area under the insulin curve was 49% higher than after decaffeinated coffee and 57% higher than after water, with P less than 0.01. This means that the body had to produce or maintain more insulin in circulation to manage the glucose.Despite this greater insulin response, the area under the glucose curve was higher after caffeinated coffee, at 217 mmol/l × 2 hours with a standard error of 24, compared to decaffeinated coffee, 126 mmol/l × 2 hours with a standard error of 11, and water, 55 mmol/l × 2 hours with a standard error of 34. The differences were significant with P equal to 0.01 and P less than 0.001, respectively. The insulin sensitivity index was lower after caffeinated coffee, 8.2 with a standard error of 0.9, compared to decaffeinated coffee, 12.4 with a standard error of 1.2, and water, 13.4 with a standard error of 1.4.

5. INTERPRETATION

In simple terms, the results indicate that caffeinated coffee was followed by less efficient glucose management in the subsequent test: both glucose and insulin levels remained higher, while the insulin sensitivity index decreased. This pattern is consistent with a temporary reduction in the effectiveness of insulin in tissues that use glucose, particularly muscle and adipose tissue.The specific sequence documented by the publication subject to this Evidence Review is therefore: intake of caffeinated coffee with carbohydrates lower insulin sensitivity three hours later higher insulin response and higher glycemia after a second glucose load. The comparison with decaffeinated coffee reinforces the hypothesis that caffeine contributes to the observed effect.The decisive limitation concerns the mechanism. The

study did not report measurements of cortisol, ACTH, or other stress hormones. Therefore, it does not allow for the effect to be attributed to cortisol mediation, nor does it define it as predominant. The results describe an acute metabolic response in ten healthy men and do not demonstrate a chronic effect of habitual coffee consumption, nor the development of persistent insulin resistance or type 2 diabetes.

6. INDEPENDENT CONFIRMING EVIDENCE

A systematic review with meta-analysis gathered seven randomized clinical trials conducted on healthy individuals, in which caffeine was the primary difference between the treatment and control groups. Compared to the placebo, caffeine significantly reduced the insulin sensitivity index, with a standardized mean difference of –2.06; the 95% confidence interval ranged from –2.67 to –1.44. This directly confirms the section regarding the acute reduction of insulin sensitivity. The quality of the synthesis is useful, but the included studies were small and moderately heterogeneous, with an I^2 of 49% [1].A previous randomized, double-blind, crossover study evaluated twelve healthy volunteers using a hyperinsulinemic-euglycemic clamp, a procedure that directly measures the amount of glucose used by the body during a controlled level of insulin. Intravenously administered caffeine reduced insulin sensitivity by 15% compared to the placebo. In the same experiment, adrenaline increased approximately fivefold. The result confirms the acute effect on insulin sensitivity but also suggests that sympathetic activation, rather than cortisol alone, may contribute to the response [2].Another randomized, double-blind, crossover study involved twenty-four healthy adults, twelve men and twelve women. After doses of caffeine ranging between 1 and 5 mg/kg and an oral glucose tolerance test, an increase of 1 mg/kg of caffeine increased the area under the curve for insulin by 5.8% and for C-peptide by 8.7%; C-peptide is a substance released along with insulin and helps estimate its secretion. Glucose exposure also increased with the dose. The effect was present in both sexes and reinforces the biological coherence between caffeine, increased insulin demand, and poorer postprandial glucose control [3].

7. CONFLICTING EVIDENCE

No directly conflicting evidence sufficiently relevant to the acute effect of caffeine on insulin sensitivity has been identified; most of the selected experimental studies converge toward a temporary reduction in insulin sensitivity. However, there is significant conflicting evidence regarding the explanation proposed in the paragraph, namely the predominant role of cortisol.In a randomized, double-blind, placebo-controlled crossover study, sixteen young adults consumed 200 mg of caffeine twice daily for seven days. Compared to the placebo, fasting insulin increased by 1.80 microU/mL, and the HOMA index of insulin sensitivity, an estimate derived from fasting glycemia and insulin, decreased by 35%. However, serum cortisol did not increase in a statistically significant manner: the difference was 1.8 microg/dL, with a confidence interval from –1.9 to 5.4 and a P-value of 0.32. The study therefore confirms the alteration in insulin sensitivity but does not support cortisol as a necessary or predominant mediator [4].This observation does not exclude the possibility that cortisol may contribute in certain contexts, for example depending on the time of day, dosage, sleep, or stress. It does, however, indicate that the causal link caffeine cortisol reduced insulin sensitivity is not demonstrated by the original study and requires experiments that measure both phenomena and directly verify their relationship.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication subject to the Evidence Review from the independent research used to confirm or define the book's narrative progression.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Crossover RCT	10 healthy young men	Caffeinated coffee increased glycemia and insulin during the second glucose load and reduced the insulin sensitivity index.	direct confirmation	moderate
[1]	Systematic review and meta-analysis of RCTs	7 RCTs in healthy adults	Caffeine significantly reduced the insulin sensitivity index compared to placebo.	direct confirmation	moderate
[2]	Double-blind crossover RCT	12 healthy volunteers	Intravenous caffeine reduced insulin sensitivity by 15% and increased adrenaline.	direct confirmation	moderate
[3]	Double-blind crossover RCT	24 healthy adults, 12 men and 12 women	Increasing doses of caffeine increased insulin, C-peptide, and glucose during the oral test.	direct confirmation	moderate
[4]	Double-blind crossover RCT	16 healthy young adults	Caffeine reduced insulin sensitivity without significantly increasing serum cortisol.	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The study analyzed directly supports the finding that, in healthy young men, caffeinated coffee consumed with a high-carb meal can impair glucose management during a subsequent load, increasing both glycemia and the insulin response while reducing estimated insulin sensitivity. This result is moderately supported by independent evidence in humans.However, this conclusion must remain limited to the acute effects observed in small experimental studies. It cannot be inferred that all habitual coffee consumption produces persistent insulin resistance, nor that cortisol represents the predominant mechanism. To clarify this, larger studies are needed that simultaneously measure caffeine, cortisol, catecholamines, sleep, stress, and insulin sensitivity.

Aggiornato al 25/11/2026. 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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