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Sleep fragmentation and glucose metabolism

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1. ABSTRACT

The analyzed study evaluated whether repeatedly interrupting sleep, without necessarily reducing its total duration, alters how the body manages glucose and insulin. Eleven healthy volunteers underwent two nights of experimental sleep fragmentation. Following the intervention, both insulin sensitivity and the ability of glucose to independently promote its own clearance from the blood decreased; morning cortisol and sympathetic nervous system activity also increased.In "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, the publication contributes to the narrative that even compromised sleep quality can alter the insulin response in healthy individuals. Independent evidence shows that a reduction in deep sleep and sleep duration restriction can reduce insulin sensitivity. However, a subsequent experiment did not find the same effect after just one night of hourly awakenings. The most prudent conclusion is that sleep continuity and quality are relevant to metabolism, but the intensity of the effect depends on the experimental protocol and the type of sleep disturbance.

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 sleep fragmentation, i.e., repeated interruptions of nocturnal rest, can alter glucose metabolism even when it occurs in healthy adults without a diagnosed sleep disorder. The hypothesis was that broken sleep reduces the effectiveness of insulin, the hormone that facilitates the entry of glucose into cells.The relevance to sections 4.14 and 4.15 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi is specific: the publication documents that poorer sleep quality can be followed by a reduction in the insulin response in clinically healthy subjects. The work also examines cortisol and autonomic nervous system activity, two possible pathways through which disturbed sleep could influence metabolism.Being an acute and very small experiment, the study can show a short-term metabolic variation under the conditions tested. However, it cannot prove that sleep fragmentation causes type 2 diabetes, nor establish that cortisol is the only mechanism responsible for the reduction in insulin sensitivity.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The study involved 11 healthy volunteers, described as normal in the available material. For two nights, sleep was fragmented across all stages using auditory and mechanical stimuli. The design included an assessment before and after exposure; the detailed sequence of conditions, the demographic characteristics of the participants, and the complete inclusion and exclusion criteria are not reported in the available material.The primary outcome was insulin sensitivity, indicated as S(I): a measure of how effectively insulin helps the body remove glucose from the blood. It was measured using an intravenous glucose tolerance test. The authors also evaluated S(G), or glucose effectiveness, which describes the ability of glucose to promote its own clearance independently of insulin, as well as insulin secretion.Secondary outcomes included sympathovagal balance, an indicator of the equilibrium between the activating and restorative components of the autonomic nervous system, serum cortisol, markers of systemic inflammation, and adipokines, which are proteins produced by adipose tissue. Details regarding statistical methods, sampling times, the intensity of the stimuli, and insulin secretion values are not reported in the available material.

4. RESULTS

After two nights of fragmented sleep, insulin sensitivity decreased from 5.02 to 3.76 (mU/L)–1min–1, with $P < 0.0001$. In practical terms, the body was less responsive to the action of insulin in managing glucose compared to the baseline condition.S(G) also decreased, from $2.73 \times 10{-}2$ to $2.16 \times 10{-}2$ min–1, with $P < 0.01$. This indicates a reduced ability of glucose to contribute independently to its own clearance from the bloodstream. The available material does not report the quantitative value of insulin secretion, nor does it clarify whether this changed in a statistically significant way.Fragmented sleep also increased morning cortisol levels and shifted the sympathovagal balance toward greater sympathetic activity, that is, toward the physiological state of activation associated with the stress response. Markers of systemic inflammation and blood adipokines did not change. This result is important because it limits the interpretation: over the two nights considered, the metabolic worsening was not accompanied by a measurable increase in the inflammatory markers examined.

5. INTERPRETATION

The main significance is that interrupted sleep can rapidly affect how the body uses glucose. The reduction in S(I) indicates lower insulin sensitivity, while the reduction in S(G) suggests that a component of glucose regulation not directly dependent on insulin has also worsened.This data connects to the narrative of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi in the segment: worsening sleep quality activation of the stress response reduction in insulin response. The study indeed observes an increase in morning cortisol and sympathetic activity along with a worsening of metabolic indices. However, it does not separately manipulate cortisol or the sympathetic system; therefore, these factors are plausible mechanisms proposed by the authors, not a definitive causal demonstration.The publication does not directly measure the PI3K/AKT/mTOR cellular cascade, which

is a series of intracellular signals that allow insulin to promote glucose utilization. It therefore supports the physiological level of the narrative—namely, the reduction in insulin sensitivity after fragmented sleep—but not the entire molecular chain described in the chapter.

6. INDEPENDENT SUPPORTING EVIDENCE

A confirmation particularly close to the book's narrative comes from a crossover experiment on nine healthy young adults. The selective suppression of slow-wave sleep, the deepest phase of non-REM sleep, for three nights reduced insulin sensitivity without reducing total sleep time. Glucose tolerance decreased, and the body did not adequately increase compensatory insulin secretion. The reduction in insulin sensitivity was greater in participants who had lost a larger amount of deep sleep. This study directly confirms that the architectural quality of sleep, not just its quantity, can influence glucose metabolism [1]. However, the sample was very small, and the intervention specifically concerned deep sleep, not the widespread fragmentation of all phases.A second experiment evaluated nine healthy adults after a single night limited to four hours of sleep. Using the hyperinsulinemic-euglycemic clamp, the gold-standard technique for measuring how much insulin promotes glucose utilization, the authors found a 19% to 25% reduction in insulin sensitivity across multiple metabolic pathways: hepatic glucose production, peripheral glucose uptake, and suppression of lipolysis, i.e., the release of fats from adipose tissue [2]. This evidence does not concern fragmentation without a reduction in duration, but it reinforces the book's more general point that acute sleep disturbances can worsen the insulin response even in healthy people.The overall picture was examined by a systematic review with meta-analysis of 35 randomized controlled trials on sleep manipulation. The 21 studies included in the meta-analysis of sleep restriction showed a reduction in insulin sensitivity measured with oral or intravenous glucose tests and an increase in HOMA-IR, an index that estimates insulin resistance, especially at the hepatic level. The review also noted adverse effects of slow-wave sleep suppression and circadian misalignment [3]. The strength of this synthesis is the breadth of the experimental evidence; the limitation is the heterogeneity of the protocols and the small number of studies specifically on fragmentation.

7. CONFLICTING EVIDENCE

A randomized crossover experiment conducted on 29 young adults yielded a different result. After a night with five-minute awakenings every hour, the participants showed no changes in glucose tolerance, the Matsuda index of insulin sensitivity, or HOMA-IR compared to a normal night [4]. The protocol differed from that of the analyzed study: the fragmentation lasted only one night, took place at home, and the metabolic assessment was based on an oral glucose tolerance test.This conflicting evidence does not invalidate the result of the original study, but it shows that the metabolic effect of fragmentation is not uniform. Factors such as the number of nights, the type and intensity of the awakenings, the laboratory or home setting, the test used to measure insulin sensitivity, and the characteristics of the participants may all play a role. Consistently, the available meta-analysis indicates that there were few studies on fragmentation and they did not show a statistically clear overall effect [3].

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication that is the subject of the review from the independent research used to verify the book's narrative.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Pre-post controlled experiment	11 healthy volunteers; two nights of fragmented sleep	Reduction in S(I) and S(G); increase in morning cortisol and sympathetic activity	direct confirmation	moderate
[1]	Crossover experiment	9 healthy young adults; three nights of slow-wave sleep suppression	Reduction in insulin sensitivity and glucose tolerance without reduction in total sleep time	direct confirmation	moderate
[2]	Crossover experiment	9 healthy adults; one night limited to four hours	19-25% reduction in insulin sensitivity in hepatic, peripheral, and adipose pathways	indirect confirmation	moderate
[3]	Systematic review and meta-analysis of RCTs	35 randomized studies; 21 in the sleep restriction meta-analysis	Sleep restriction reduces indicators of insulin sensitivity; inconclusive evidence for fragmentation	biologically consistent evidence	moderate
[4]	Crossover RCT	29 young adults; one night with hourly five-minute awakenings	No change in insulin sensitivity or glucose tolerance after fragmentation alone	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The study analyzed shows that two nights of experimentally fragmented sleep were associated with a reduction in insulin sensitivity and the ability of glucose to contribute to its own regulation in healthy volunteers. The result is consistent with a concomitant increase in morning cortisol and sympathetic activity, but it does not prove that these factors are the sole cause of the effect.The narrative that sleep quality contributes to maintaining the insulin response is moderately supported by independent experiments on deep sleep suppression and sleep duration restriction. Caution remains necessary when generalizing the data to every form of interrupted sleep, as some short fragmentation protocols have not produced measurable metabolic alterations.

Aggiornato al 24/11/2026. 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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