LIFE Science Hub
JOURNAL OF SCIENCE
EXPANDING KNOWLEDGE ABOUT NUTRITION AND HEALTH

ORIGINAL RESEARCH
ISSN 2845-7501

Associations between sleep loss and increased risk of obesity and diabetes - Review of the evidence

D.ssa M. Mondini[1]

1 Biologist, Italy

1. ABSTRACT

The analyzed publication examines current knowledge regarding the relationship between insufficient sleep, glucose regulation, and metabolic risk. The available abstract reports that, in healthy volunteers, experimental sleep restriction rapidly reduces insulin sensitivity, i.e., the ability of tissues to respond to the hormone that facilitates glucose entry into cells. It also describes alterations in glucose tolerance, pancreatic beta-cell compensation, and the hormonal signals that regulate appetite and satiety. However, since only the abstract is available, the full design of the review, the individual cohorts considered, or the numerical values cited in the book are not reported in the consultable material.In paragraph 4.15 of “The Choice to Live in Health or Illness” by Dr. A. Colonnese and R. Panzironi, the study contributes to the narrative that sleep loss can rapidly promote alterations compatible with insulin resistance. Independent experimental studies in humans show a reduction in insulin sensitivity after one or more nights of reduced sleep. Two meta-analyses of randomized studies confirm an overall unfavorable effect, but also signal heterogeneity: some peripheral indicators and certain forms of qualitative sleep disturbance do not show a uniform effect. The narrative is therefore moderately supported, especially at a physiological experimental level, without demonstrating that every episode of insomnia causes diabetes in every individual.

ONLINE ARTICLE

ARTICLE INFORMATION

Published: November 20, 2026
Updated: November 20, 2026

DOI 10.5281/zenodo.24393644

MISSION

LIFE Science Hub – Journal of Science was founded with the aim of making scientific knowledge accessible without giving up methodological rigour, quality of sources and authoritativeness of content. The journal promotes the dissemination of knowledge in the health, nutrition, medical and longevity sciences, building a bridge between the world of research and society.

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 habitual or experimental sleep loss might contribute to an increased risk of obesity and type 2 diabetes through alterations in glucose metabolism and appetite regulation. The hypothesis discussed is that sleeping too little reduces insulin sensitivity and makes it more difficult to keep blood glucose stable.This question is relevant to paragraph 4.15 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi because the text links insufficient sleep to the early onset of insulin resistance. The publication subject to this evidence review primarily supports the physiological segment of the chain: sleep loss reduced tissue response to insulin poorer glucose regulation.The analyzed study can summarize a biologically plausible relationship and describe the results of experiments and prospective studies cited by the authors. However, based on the available abstract alone, it cannot establish which numerical data derive from each included study, nor can it demonstrate that insufficient sleep is the sole cause of diabetes or obesity in the general population.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The available material consists only of the abstract. The publication appears to be a literature review on sleep, glucose metabolism, insulin sensitivity, and the neuroendocrine regulation of appetite, rather than a single experiment with a new sample of participants.The abstract reports that the authors considered laboratory studies in healthy volunteers and prospective epidemiological studies in children and adults. Experiments on sleep restriction, assessments of glucose homeostasis, pancreatic beta-cell function, and hormones such as leptin and ghrelin are mentioned. Glucose homeostasis is the set of mechanisms that maintains glycemia within ranges compatible with the normal functioning of the body.The bibliographic search strategy, inclusion and exclusion criteria, number of studies examined, populations involved, observation periods, predefined endpoints, and statistical analyses are not reported in the available material. Consequently, it is not possible to classify this publication with certainty as a systematic review, nor to verify from the abstract the specific quantitative data mentioned in the book's paragraph.

4. RESULTS

The core finding reported in the abstract is that experimental sleep restriction in healthy volunteers impairs glucose control. Simply put, after insufficient sleep, the body may require more insulin to manage the same amount of glucose. Technically, the abstract reports a rapid and marked reduction in insulin sensitivity without an adequate compensatory increase in beta-cell function, i.e., the pancreatic cells that produce insulin.The publication also reports that sleep loss is associated with a reduction in leptin, a signal linked to satiety, and an increase in ghrelin, a hormone that promotes hunger. The authors interpret these variations as a possible contribution to an internal perception of insufficient energy availability. The abstract also mentions a possible involvement of orexins, brain molecules that promote both wakefulness and food intake.The most relevant passage for the book is the indication that reduced sleep can worsen insulin sensitivity even before obvious alterations in glycemia appear. However, the numerical data cited in paragraph 4.15, including the increase of more than 50% in the area under the HOMA-IR curve, the 40% reduction in glucose tolerance, and the 40% reduction in the disposition index, are not reported in the available abstract. Therefore, they cannot be directly attributed to the analyzed publication based on the material provided.

5. INTERPRETATION

The practical significance of this publication is that sleep is not just a break from work or daily activities. It is also a state that helps the liver, muscles, adipose tissue, and pancreas coordinate glucose management. When sleep is reduced, these organs may respond less effectively to insulin; the pancreas may therefore have to increase its effort to keep glycemia stable.In the language of the book's paragraph, the documented segment is: reduced sleep duration reduced insulin sensitivity alterations consistent with insulin resistance. Insulin resistance does not automatically equate to diabetes: it indicates a condition in which tissues respond less to insulin and which can be compensated for variable periods by increased pancreatic secretion.The publication also links

insufficient sleep to changes in cortisol and appetite, but the abstract does not allow us to determine how much each mechanism contributes to the reduction in insulin sensitivity. Furthermore, experimental sleep deprivation studies describe short-term physiological effects under controlled conditions; they do not, by themselves, prove that every person with insomnia will develop a metabolic disease.

6. INDEPENDENT SUPPORTING EVIDENCE

Direct confirmation of the transition from reduced sleep to poorer glucose control comes from a controlled study of 11 young men. After six nights with four hours in bed per night, compared to the recovery phase with twelve hours in bed, glucose tolerance was reduced and evening cortisol concentrations were increased. Glucose tolerance expresses the body's ability to return glycemia to appropriate levels after a sugar load. The study does not demonstrate long-term clinical risk, but it shows that repeated restriction can rapidly alter metabolism in healthy subjects [1].A second crossover experiment, in which the same nine participants underwent both conditions, strengthened the link. After just one night with four hours of sleep, compared to an unrestricted night, the ability of tissues to clear glucose during the euglycemic-hyperinsulinemic clamp decreased from 40.7 to 32.5 micromoles per kilogram of lean mass per minute. The clamp is a test that measures in a controlled manner how much glucose must be infused to maintain stable glycemia during insulin administration. The observed reduction indicates lower peripheral insulin sensitivity; hepatic glucose production also increased, consistent with a lower liver response to insulin [2].The picture does not depend solely on individual small experiments. A systematic review with meta-analysis of 41 randomized studies in healthy adults found that sleep restriction reduced insulin sensitivity, with a standardized mean difference of –0.70 and a P-value of less than 0.01. A negative standardized mean difference indicates, in this comparison, that insulin sensitivity was lower during restricted sleep compared to normal sleep. The synthesis also observed increased hunger and higher caloric intake, but not strong evidence of uniform average variations in leptin or ghrelin [3].This evidence is independent of the analyzed publication and confirms its central narrative with human experiments. However, important limitations remain: often small samples, very different protocols regarding the duration and method of restriction, predominantly healthy participants, and short-term evaluations. Therefore, the acute physiological effect is more solidly documented than the prediction of individual long-term diabetes risk.

7. CONFLICTING EVIDENCE

A more recent meta-analysis of 35 randomized trials confirms that reduced sleep duration worsens several markers of insulin sensitivity, including those derived from glucose tolerance tests and HOMA-IR. HOMA-IR is an index calculated using fasting glycemia and insulin: higher values are generally consistent with greater insulin resistance. However, the same analysis did not find a statistically uniform overall effect on peripheral sensitivity when evaluated in a limited number of studies using specific methods.The meta-analysis also found that sleep fragmentation and REM sleep disturbance—the phase characterized by intense brain activity—did not show a uniform effect on insulin sensitivity. These results do not negate the effect observed in sleep restriction studies, but they indicate that duration, quality, circadian timing, measurement method, and participant characteristics can modify the magnitude of the result [4].No directly conflicting evidence sufficiently relevant to the fact that marked experimental sleep restriction can reduce certain indicators of insulin sensitivity has been identified; this does not prove the absence of contrary results.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication subject to the review from the independent research used to evaluate the transition from reduced sleep → insulin resistance.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Narrative synthesis, based only on the available abstract	Laboratory and prospective studies cited by the authors; sample size not reported	Reports that sleep loss reduces insulin sensitivity and impairs glucose control	biologically consistent evidence	moderate
[1]	Controlled experiment	11 young men	Six nights with four hours in bed reduced glucose tolerance and increased evening cortisol	direct confirmation	moderate
[2]	Experimental crossover study	9 healthy adults, five men and four women	One night with four hours of sleep reduced peripheral glucose uptake and increased hepatic glucose production	direct confirmation	moderate
[3]	Systematic review and meta-analysis of RCTs	41 randomized studies in healthy adults	Sleep restriction reduced overall insulin sensitivity	direct confirmation	high
[4]	Systematic review and meta-analysis of RCTs	35 eligible articles; 21 sleep restriction studies in the meta-analysis	Confirms overall adverse effects, but not a uniform effect on peripheral sensitivity or sleep fragmentation	discordant evidence	high

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The publication analyzed consistently supports the link between sleep loss, reduced insulin sensitivity, and poorer glucose control. This is the segment of the book's narrative that is most strongly supported. Independent experiments and meta-analysis confirm that even short-term sleep restriction can produce measurable metabolic alterations in healthy individuals.However, the evidence does not demonstrate that a single sleepless night causes diabetes, nor that cortisol alone explains all the observed effects. The framework is moderately supported at a physiological level and requires further long-term studies, in larger and more diverse populations, to define the impact on individual clinical risk.

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.

11. RIFERIMENTI BIBLIOGRAFICI

[1] **Impact of sleep debt on metabolic and endocrine function**
Spiegel et al., 1999
The Lancet
DOI: https://doi.org/10.1016/S0140-6736(99)01376-8

[2] **A Single Night of Partial Sleep Deprivation Induces Insulin Resistance in Multiple Metabolic Pathways in Healthy Subjects**
Donga et al., 2010
The Journal of Clinical Endocrinology & Metabolism
DOI: https://doi.org/10.1210/jc.2009-2430

[3] **Effects of sleep restriction on metabolism-related parameters in healthy adults: A comprehensive review and meta-analysis of randomized controlled trials**
Zhu et al., 2019
Sleep Medicine Reviews
DOI: https://doi.org/10.1016/j.smrv.2019.02.002

[4] **Effects of sleep manipulation on markers of insulin sensitivity: A systematic review and meta-analysis of randomized controlled trials**
Sondrup et al., 2022
Sleep Medicine Reviews
DOI: https://doi.org/10.1016/j.smrv.2022.101594