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Sleep loss leads to increased cortisol levels the following evening - Evidence review

Dr.ssa M. Bitonti[1]

1 Nutritional Biologist, Italy

1. ABSTRACT

The analyzed study evaluated whether acute sleep deprivation, whether partial or total, alters the profile of cortisol, a hormone essential for the stress response and energy regulation. In healthy young men, after a night of reduced or absent sleep, cortisol was higher in the following evening hours, and the onset of the normal phase of reduced secretion was delayed. The sample size is not reported in the available material.The research contributes to the passage from "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, according to which the evening increase in cortisol is one of the first observable signs after sleep loss and may be part of a chain of metabolic alterations. Independent studies confirm that sleep restriction can increase daytime cortisol and worsen indicators of glucose metabolism. However, a direct causal link between evening cortisol alone and reduced peripheral glucose uptake is not demonstrated by the original work and appears to be influenced by other hormones and mechanisms as well.

ONLINE ARTICLE

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MISSION

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2. ANALYZED STUDY

The research question was whether losing a night of sleep, either partially or totally, produces effects that continue into the following day on the hypothalamic-pituitary-adrenal axis, the system that coordinates the hormonal response to stress. The hypothesis was that acute sleep loss does not only alter nighttime rest, but also modifies the normal daily pattern of cortisol.The relevance to paragraph 4.15 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi is specific: the publication documents the increase in cortisol on the evening following a night of reduced or absent sleep. This is the starting point of the book's narrative. The study did not directly measure glucose uptake in muscles or adipose tissue and therefore cannot prove that the evening rise in cortisol, on its own, causes insulin resistance.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The research analyzed healthy young men subjected to three experimental protocols: normal sleep from 11:00 PM to 7:00 AM, partial sleep deprivation with sleep from 4:00 AM to 8:00 AM, and total sleep deprivation. Plasma cortisol profiles were measured for 32 hours, from 6:00 PM on the first day to 2:00 AM on the third day.The main comparison concerned the cortisol concentration between 6:00 PM and 11:00 PM on the first day and that observed in the same time frame after the experimental night. The onset time of the cortisol secretion quiescence period—the evening phase during which this hormone normally decreases—was also evaluated. The sample size, inclusion and exclusion criteria, randomization methods, detailed statistical analysis, and other aspects of the protocol are not reported in the available material, which consists only of the abstract.

4. RESULTS

After a normal night's sleep, plasma cortisol measured between 6:00 PM and 11:00 PM was similar on both the first and second day. In other words, normal nocturnal rest did not result in an evening increase in cortisol the following day.After partial sleep deprivation, however, cortisol levels in the 6:00 PM–11:00 PM window increased by 37% on the second day compared to the first, with a P-value of 0.03. After total sleep deprivation, the increase was 45%, with a P-value of 0.003. The P-value indicates how compatible the observed result is with chance according to the statistical model used; in both comparisons, the authors considered the increase to be statistically significant.Sleep loss also delayed the onset of the evening period of low cortisol secretion by at least one hour. For the reader, the practical significance is that cortisol remained elevated for longer during the time of day when, following an adequate night's rest, it should begin to decrease. The study does not report absolute cortisol values in the available material, nor does it provide direct data on insulin, glycemia, glucose tolerance, or tissue glucose uptake.

5. INTERPRETATION

The central biological result is clear: a single night of reduced or absent sleep can delay the evening decline of cortisol. Since cortisol promotes energy availability during stress, more prolonged exposure can theoretically make the action of insulin—the hormone that facilitates the entry of glucose into cells—less efficient.The authors interpreted the delay in the cortisol decline as a possible sign of incomplete recovery of the hypothalamic-pituitary-adrenal axis from morning circadian stimulation. They also hypothesized an alteration in the negative feedback of glucocorticoids, the mechanism by which cortisol limits its own production. However, this is a physiological interpretation: the study did not directly measure the feedback nor did it isolate the causes of the change.Within the scope of the book, the work supports the transition: lost sleep evening increase in cortisol. The subsequent transition: higher cortisol lower peripheral glucose uptake requires further evidence. It is biologically plausible, but it was not directly demonstrated in this experiment.

6. INDEPENDENT SUPPORTING EVIDENCE

Independent confirmation of hormonal elevation and metabolic alterations has come from an experimental study conducted on 14 healthy young men. After five nights with four hours in bed per night, compared to the baseline condition of ten hours in bed, daytime cortisol, glycemia, and insulin levels increased. Cortisol increased with $P = 0.002$, while glucose and insulin increased with $P < 0.001$, respectively. This

data confirms that more prolonged restriction can be accompanied by greater cortisol exposure and a less favorable metabolic profile. However, the fasting HOMA-IR index, an indirect estimate of hepatic insulin resistance, did not change significantly; therefore, the study does not prove that cortisol is the sole cause of the observed variations [1].A second randomized, double-blind, crossover study explored the hormonal contribution more directly. Thirty-four healthy young men underwent four nights with four hours of sleep. When cortisol and testosterone were artificially maintained at baseline levels using a double hormonal clamp, the development of overall insulin resistance and hyperinsulinemia was attenuated by 50% compared to the placebo. The effect on the worsening of insulin sensitivity had a P = 0.046. This result supports the idea that changes in cortisol and testosterone contribute to the metabolic response to sleep restriction, but it does not attribute the phenomenon to cortisol in isolation, because both hormones were controlled together [2].These studies form a chain consistent with the book's narrative: sleep loss can increase cortisol during a part of the day when it should decrease and, simultaneously or subsequently, be associated with lower insulin sensitivity. The level of confirmation is more solid for the effects of sleep restriction on cortisol and glucose metabolism indicators than for the specific claim that evening cortisol alone inhibits peripheral glucose uptake.

7. CONFLICTING EVIDENCE

One element that calls for caution comes from a controlled study of 20 healthy men subjected to one week of five hours of sleep per night. The restriction reduced insulin sensitivity by 20% according to an intravenous glucose tolerance test and by 11% according to the hyperinsulinemic-euglycemic clamp, a method that measures how much glucose must be administered to keep glycemia stable during insulin infusion. In the same study, salivary cortisol increased by an average of 51%, but the change in cortisol was not correlated with the change in insulin sensitivity [3].This result does not contradict the rise in cortisol following sleep loss. However, it scales back the interpretation that cortisol alone explains the worsening of insulin action. The relationship may depend on the duration and timing of the restriction, testosterone, sympathetic nervous system activity, diet, circadian rhythms, and individual differences. No directly conflicting evidence sufficiently relevant to the evening increase in cortisol observed in the original study has been identified; this does not prove the absence of contrary results.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication being reviewed from the independent research used to evaluate the specific passage of the book.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Acute physiological experiment	Healthy young men; sample size not reported in available material	Following partial or total deprivation, evening cortisol increased by 37% and 45% respectively; the reduction phase was delayed by at least one hour.	direct confirmation	moderate
[1]	Controlled experimental study	14 healthy young men; five nights with four hours in bed	Daytime cortisol, glucose, and insulin increased; fasting HOMA-IR did not change significantly.	direct confirmation	moderate
[2]	Double-blind crossover RCT	34 healthy young men; four nights with four hours of sleep	Joint control of cortisol and testosterone attenuated sleep restriction-induced insulin resistance and hyperinsulinemia by 50%.	indirect confirmation	moderate
[3]	Crossover controlled study	20 healthy men; one week with five hours of sleep per night	Insulin sensitivity decreased and salivary cortisol increased, but the two changes were not correlated.	discordant evidence	moderate

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The study analyzed documents that acute sleep loss can increase cortisol the following evening and delay its normal decline. This point is well-supported and precisely explains why the research was cited in the book's section. Independent evidence also indicates that sleep restriction can worsen insulin sensitivity and that hormonal changes contribute to this effect. However, it remains to be validated to what extent evening cortisol alone directly determines reduced peripheral glucose uptake. The chain is therefore moderately supported in humans, but not fully proven as a sole causal relationship.

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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