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Investigating the Relationship Between Sleep Quality, Inflammatory Biomarkers, and Immune System Function in Young Adults

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Abstract

Background: Poor sleep quality is common among young adults, yet its role in immune health is often underestimated. This paper reviews and synthesises published human evidence on how sleep quality and duration relate to inflammatory biomarkers (C-reactive protein [CRP], interleukin-6 [IL-6], tumour necrosis factor-alpha [TNF-α]) and to functional immune outcomes such as infection susceptibility and vaccine response.
Methods: A narrative evidence synthesis was carried out on one large meta-analysis (72 studies, more than 50,000 participants) and five landmark experimental and prospective studies whose samples include young adults. Reported effect sizes, odds ratios and fold-changes were extracted exactly as published and are presented in two tables and two figures.
Results: Sleep disturbance was linked to higher CRP (effect size 0.12) and IL-6 (0.20). A single night of partial sleep loss raised IL-6 messenger RNA more than three-fold. Short or inefficient sleep before viral exposure raised the odds of a clinical cold by 2.9 to 5.5 times, and sleeping under six hours raised the odds of being unprotected after hepatitis B vaccination by 11.5 times.
Conclusion: Sleep quality, and not only sleep length, is a modifiable risk factor for low-grade inflammation and weaker immune defence. Young adults are a key group for sleep-focused prevention.

Keywords: sleep quality, sleep efficiency, C-reactive protein, interleukin-6, immune function, vaccine response, young adults

Introduction

Sleep is one of the main regulators of the human immune system. During normal night-time sleep, the body shifts its immune activity: pro-inflammatory signalling falls, and processes that support immune memory, such as antigen presentation and T-cell help to B cells, are favoured. When sleep is short, broken or of poor quality, this rhythm is disturbed.

Young adults, roughly 18 to 30 years of age, are a group at special risk. University study, shift work, late-night screen use, social jet lag and caffeine all push sleep later and make it shorter. Many young adults therefore build a chronic sleep debt during the very years when long-term health habits are formed. Because they are usually healthy, the immune costs of poor sleep in this group are easy to miss.

Two kinds of immune outcome matter here. The first is **low-grade systemic inflammation**, measured by circulating biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α). Raised levels of these markers predict later cardiovascular disease, type 2 diabetes and depression. The second is **functional host defence**: how well the body resists a real virus, and how strongly it makes antibodies after a vaccine.

Sleep can be described in two ways. *Sleep duration* is how long a person sleeps. *Sleep quality* covers how continuous and restorative that sleep is; it is often measured as *sleep efficiency*, the percentage of time in bed actually spent asleep. A key question is whether quality matters as much as quantity.

Aim. This paper brings together the best available published human evidence to answer three questions:

1. Is poor sleep quality or abnormal sleep duration associated with higher inflammatory biomarkers?
2. Does sleep loss directly switch on inflammatory pathways in immune cells?
3. Do sleep quality and duration predict real immune outcomes such as infection risk and vaccine response, especially in young and early-middle-aged adults?

Methods

Design. This is a narrative evidence synthesis (secondary analysis of published data). No new participants were recruited. All numbers in the tables and figures are taken directly from the original peer-reviewed papers; none were simulated or estimated.

Source selection. Studies were chosen if they (a) were peer-reviewed human studies, (b) measured sleep duration, sleep quality or sleep efficiency, either objectively (actigraphy, polysomnography, lab-controlled sleep) or by daily diary, and (c) reported either an inflammatory biomarker (CRP, IL-6, TNF-α) or a functional immune outcome (clinical infection after viral challenge, or antibody response to vaccination). Preference was given to studies whose samples included young adults (aged 18 to 30).

Included evidence. Six sources met these criteria:

- one systematic review and meta-analysis of 72 studies on sleep and inflammation[2];
- one experimental partial sleep deprivation study on monocyte cytokine production[3];
- two prospective viral-challenge studies of the common cold[1,5];
- two vaccination studies: one experimental[6] and one naturalistic[4].

Data extraction. For each study, the sample size, age range, sleep exposure, outcome and main effect estimate (effect size, odds ratio or fold-change, with 95% confidence intervals where reported) were recorded. Effect sizes from the meta-analysis are standardised mean differences pooled with a random-effects model, as reported by the original authors.

Analysis. Because the studies use different designs and outcome measures, results were not pooled statistically. Instead, they are

compared side by side in Tables 1 and 2 and visualised in Figures 1 and 2.

Results

Characteristics of the included studies

The six sources cover more than 50,000 participants in total. Three studies used objective or laboratory-controlled sleep measures, and two of them directly exposed participants to a live virus. Table 1 summarises their design.

Table 1: Characteristics of the included studies

Study	Design	Sample (n, age)	Sleep measure	Immune outcome
Irwin et al., 2016	Systematic review and meta-analysis of 72 studies	>50,000 adults	Self-report, polysomnography, experimental deprivation	Circulating CRP, IL-6, TNF-α
Irwin et al., 2006	Experimental partial sleep deprivation (awake 23:00–03:00)	30 healthy adults	Laboratory-controlled	Monocyte IL-6 and TNF-α production; mRNA
Spiegel et al., 2002	Experimental sleep restriction (4 h/night for 6 nights)	25 healthy men, mean age 23	Laboratory-controlled	Influenza vaccine antibody titre
Cohen et al., 2009	Prospective viral challenge (rhinovirus)	153 adults, 21–55	14-day sleep diary	Clinical cold
Prather et al., 2012	Prospective vaccination cohort	125 adults, 40–60	Sleep diary; actigraphy (n = 88)	Hepatitis B antibody protection
Prather et al., 2015	Prospective viral challenge (rhinovirus)	164 adults, 18–55	7-day wrist actigraphy	Clinical cold

Sleep and circulating inflammatory biomarkers

Sleep disturbance (poor sleep quality or insomnia complaints) showed the clearest link with inflammation. It was associated with higher CRP and, more strongly, with higher IL-6, with an effect size of 0.20 (95% CI 0.08–0.31). Long sleep (more than 8 hours) was also linked to higher CRP and IL-6. Short sleep was weakly linked to CRP but not to IL-6. Neither sleep disturbance nor sleep duration was associated with TNF-α. Experimental sleep deprivation, tested over short periods, did not change circulating CRP, IL-6 or TNF-α in the pooled data[2].

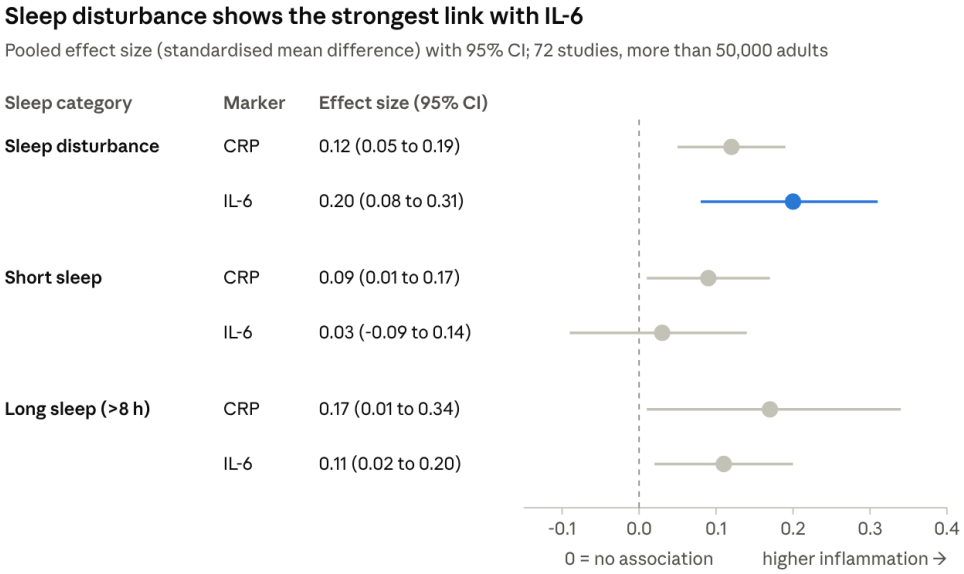


Fig 1: Pooled effect sizes (standardised mean difference with 95% CI) linking sleep disturbance, short sleep and long sleep with CRP and IL-6; 72 studies, more than 50,000 adults. Source: Irwin, Olmstead & Carroll (2016), Biol Psychiatry 80:40–52. TNF-α not shown (no significant association).

Figure 1 shows that every confidence interval except short sleep with IL-6 sits to the right of zero, so most sleep problems go with slightly higher inflammation.

Sleep loss and cellular inflammation

Circulating levels may not change after one bad night, but immune cells do. In 30 healthy adults kept awake from 23:00 to 03:00, morning monocyte production of IL-6 and TNF-α was significantly higher than after a full night of sleep. Sleep loss also produced a more than 3-fold increase in IL-6 messenger RNA and a 2-fold increase in TNF-α messenger RNA[3]. In other words, a single night of partial sleep loss is enough to switch on inflammatory gene programmes.

Sleep and functional immune outcomes

All five functional studies point in the same direction: shorter or less efficient sleep goes with weaker host defence (Table 2).

Table 2: Sleep exposure and functional immune outcomes

Study	Sleep exposure vs reference	Outcome	Main result
Cohen et al., 2009	Duration <7 h vs ≥8 h	Clinical cold after rhinovirus	OR 2.94 (95% CI 1.18–7.30)
Cohen et al., 2009	Efficiency 92–98% vs >98%	Clinical cold after rhinovirus	OR 3.94
Cohen et al., 2009	Efficiency <92% vs ≥98%	Clinical cold after rhinovirus	OR 5.50 (95% CI 2.08–14.48)
Prather et al., 2015	Duration 5–6 h vs >7 h	Clinical cold after rhinovirus	OR 4.2
Prather et al., 2015	Duration ≤5 h vs >7 h	Clinical cold after rhinovirus	OR 4.5
Prather et al., 2012	Duration <6 h vs >7 h	Unprotected after 3-dose hepatitis B vaccine	11.5 times more likely unprotected
Spiegel et al., 2002	4 h/night for 6 nights vs 7.5–8.5 h	Influenza antibody titre at day 10	Less than half the titre of controls; no difference by 3–4 weeks

In the Cohen study[1], sleep **efficiency** was a stronger predictor than sleep **duration**. Losing as little as 2–8% of time in bed to wakefulness, about 10–38 minutes for an 8-hour sleeper, was tied to 3.9 times the odds of a cold. How rested people *felt* did not predict illness. In the Prather study[5], short sleep was a stronger predictor of catching a cold than age, stress, income or smoking.

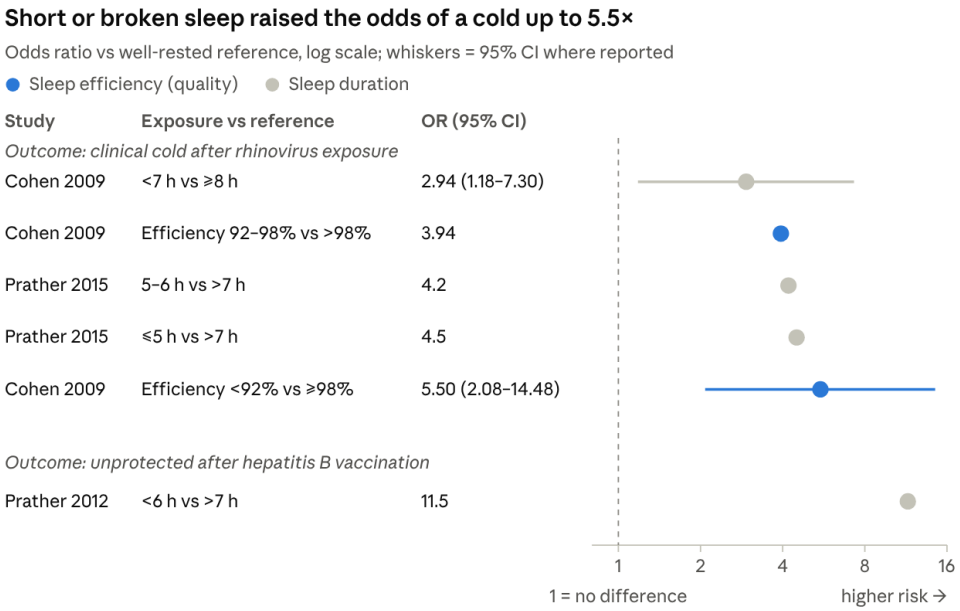


Fig 2: Odds of a clinical cold after rhinovirus exposure, and of being unprotected after hepatitis B vaccination, by sleep exposure versus a well-rested reference (log scale; whiskers = 95% CI where reported). Sources: Cohen et al. (2009), Prather et al. (2012), Prather et al. (2015). Spiegel et al. (2002) reported titres, not odds, and is shown in Table 2 only.

Figure 2 shows that the lowest sleep-efficiency group (blue) had higher odds of a cold than any sleep-duration group, which supports the idea that sleep quality matters most.

Discussion

Main findings

The evidence supports a clear conclusion: **sleep quality is at least as important as sleep quantity for immune health.** Across a meta-analysis of more than 50,000 people, sleep disturbance was a more consistent predictor of raised CRP and IL-6 than short sleep. In the viral-challenge data, low sleep efficiency carried the highest odds of a clinical cold (OR 5.50). Even small, regular losses of sleep continuity, of the kind many students do not notice, were linked to measurable risk.

The results also show that **the time scale matters**. Short-term laboratory sleep loss did not raise circulating inflammatory markers in pooled analyses, yet a single night of partial sleep loss switched on inflammatory genes inside monocytes. This suggests that cellular activation comes first. Raised blood levels of CRP and IL-6 may appear only when poor sleep becomes chronic.

The U-shaped pattern for duration is worth noting. Both short and long sleep were linked to higher CRP. Long sleep in observational studies may partly reflect underlying illness, depression or low activity, rather than sleep itself causing inflammation.

Possible mechanisms

Several biological pathways can explain these links:

1. **Sympathetic nervous system activation.** Sleep loss raises sympathetic outflow and noradrenaline, which activate the NF-κB transcription pathway in immune cells and increase IL-6 and TNF-α production[8].
2. **HPA-axis disruption.** Poor sleep alters the normal daily rhythm of cortisol, weakening its usual control over inflammation.
3. **Loss of sleep-dependent immune memory.** Deep slow-wave sleep supports the hormonal environment (high growth hormone and prolactin, low cortisol) that helps antigen-presenting cells activate T-helper cells. This step is key for building antibody responses after vaccination.
4. **Weaker antiviral defence.** Natural killer cell activity and antiviral cytokine responses are reduced after sleep loss, which may explain the higher cold rates in short and inefficient sleepers.

Relevance to young adults

The Spiegel trial[6] was run in young men with a mean age of 23. Six nights of 4-hour sleep, a pattern similar to an exam week, cut their 10-day influenza antibody titre to less than half that of well-rested controls. Titres caught up after 3–4 weeks, but the early weeks after vaccination are when protection matters most during an outbreak. Both cold-challenge studies included participants from age 18 or 21.

For young adults, this has practical meaning. Protecting sleep in the days around a vaccination, during exam periods and during flu season is a low-cost way to support immune defence. Interventions such as cognitive behavioural therapy for insomnia (CBT-I), fixed wake times, less evening screen time and limited late caffeine target sleep **quality**, which this evidence suggests is the more important lever.

Limitations

- **Mixed designs.** Most observational studies show association, not causation. Only the laboratory studies manipulated sleep directly, and their samples were small (25 and 30 participants).
- **Age coverage.** Only one study[6] was limited to young adults. The hepatitis B cohort included adults aged 40–60, so its effect size may not transfer directly to people under 30.
- **Sex differences.** The Spiegel trial included only men. A 2023 meta-analysis of seven vaccine studies[7] found the sleep–antibody link was strong in men but weaker and not significant in women.
- **Measurement.** Several studies used self-reported sleep, which is less accurate than actigraphy. In the vaccine meta-analysis, the link held only when sleep was measured objectively.
- **No pooled estimate.** Because outcomes differ (effect sizes, odds ratios, titres), the results could not be combined into one statistic.

Future research should run large, objectively measured cohorts of 18–30-year-olds, with repeated CRP and IL-6 sampling, and randomised trials testing whether improving sleep quality (for example with CBT-I) lowers inflammation and improves vaccine response.

Conclusion

Poor sleep quality in young adults is associated with higher inflammatory biomarkers, activation of inflammatory genes in immune cells, greater susceptibility to viral infection and weaker vaccine responses. Sleep efficiency, the continuity of sleep, appears to matter as much as or more than total sleep time. Sleep should be treated as a modifiable immune risk factor alongside diet and physical activity, and young adults are a priority group for sleep-health education and intervention.

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