inflammation. Mediators of inflammatory resolution mainly derive from omega-3 fatty acids converted to specialized pro-resolving mediators (SPMs), such as resolvins. We investigated SPMs in healthy humans exposed to a novel model of experimental insomnia.
Methods: Twenty-four individuals (age 18–42 years, 12 women) participated in a study consisting of two 19-day in-hospital protocols (insomnia/control). After three nights of baseline sleep (8h/night, 23:00-07:00), participants in the experimental insomnia condition were exposed to three cycles of three nights of disturbed sleep (delayed sleep-onset, hourly sleep disruption, advanced sleep-offset) followed by one night of 8h-recovery sleep. The protocol ended with three additional nights of recovery sleep. In the control condition, participants had an uninterrupted sleep opportunity (8h/night) across the 19-day protocol. Blood samples were taken at 11:00 at baseline, during experimental insomnia exposure, and after recovery sleep. Several SPMs were measured in plasma using liquid chromatography-tandem mass spectrometry (LC-MS/MS). Data were analyzed using linear mixed models.
Results: Exposure to experimental insomnia affected several SPMs compared to control sleep, including a decrease of resolvin D4 and E2 concentrations, which was still evident after the third recovery night ($p<.05$).
Conclusion: This is the first investigation on the effects of experimentally induced sleep disturbance on inflammatory resolution pathways. The results support that SPMs, particularly resolvin D4 and E2, are decreased by sleep disturbances, and do not normalize after a couple of nights of recovery sleep. Targeting these pathways by pharmacologically increasing SPMs may help to limit inflammatory consequences of sleep disturbances.
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DOES LOSING SLEEP UNLEASH ANGER?
Krizan, Z.[1] Miller, A.[1] Hisler, G.[3]
[1]Iowa State University, Ames, IA, [2]Iowa State University, Ames, IA, [3]University of Pittsburgh School of Medicine, Pittsburgh, PA.

Introduction: Sleeping is understood as essential to affective function, yet little is known about how sleep shapes more specific and contextualized emotional responses besides anxiety and depression, such as anger. Anger itself involves arousal and can disrupt sleep. To examine the causal role of sleep in anger, a daily-diary study and an experimental study tested whether shortened sleep amplifies angry feelings, while exploring mediating mechanisms of this influence.
Methods: The daily-diary study (N = 202) collected daily reports of last-night's sleep, daily stressors, and state anger across one month from college students, examining sleep and anger within everyday life. The experimental laboratory study (N = 147 community residents) examined changes in anger experienced during aversive noise following random assignment to either at-home sleep restriction (by about 5 hours across 2 nights), or to individuals' regular schedule.
Results: In the daily-diary study, individuals experienced more anger on days following less sleep than their usual, with half of this effect attributed to the increased frequency of stressors experienced on such days, and somewhat independently from the effect of sleep duration on negative affect more generally. In the experimental study, well-slept individuals adapted to noise and reported less anger and negative affect after 2 days. In contrast, sleep-restricted individuals exhibited higher and increased anger responses. The impact of sleep restriction on anger held even after accounting for negative emotions more generally. Subjective sleepiness accounted for most of the experimental effect of sleep loss on anger.
Conclusion: Together, these results provide compelling evidence that lost sleep amplifies anger in both the laboratory and everyday life, while also pointing to short-term (subjective sleepiness) and mid-term (stress) mediators of these influences. The findings also point to the value of examining specific emotional reactions (and their regulation) in the context of sleep disruption, alongside affect more broadly.
Support: N/A

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SLEEP RESTRICTION DOES NOT ALTER THE TRANSCRIPTION OF ADIPOSE TISSUE INSULIN-SIGNALING REGULATORS
Singh, P.[1] Covassin, N.[2] Bukartyk, J.[2] Davison, D.[2] Svatikova, A.[2] St Louis, E. K.[2] Somers, V. K.[2]
[1]Pennington Biomedical Research Center, Baton Rouge, LA, [2]Mayo Clinic, Rochester, MN.

Introduction: Voluntary sleep curtailment decreases insulin sensitivity. However, molecular mechanisms underlying impaired insulin signaling are not completely understood. To gain molecular insights, we examined the transcription of known cellular insulin-signaling regulators in adipose tissue obtained from subjects undergoing experimental sleep restriction.
Methods: Nineteen healthy subjects (males: 11; age: 19 - 36 years; BMI: 24.5 ± 3.6 kg/m^2) underwent a normal (9 hours/night) and a restricted sleep (4 hours/night) sequence in a random order. Each inpatient stay included a screen visit followed by 4 days of acclimation and 9 days of experimental phase consisting of a controlled sleep opportunity. Eucaloric diet was provided to ensure weight maintenance. Abdominal fat sample was obtained at the screen and end of the experimental phase. mRNA was quantified by RT-PCR. Fasting morning blood draws were obtained at the end of acclimation and experimental phases. Mixed models were used for analysis.
Results: mRNA expression did not differ in normal and restricted sleep condition for SOCS3 [changes in normal Vs. restricted sleep, 0.02 (0.11, -0.01) Vs. 0.01 (0.12, -0.07), p=0.33], PTEN [-0.22 (0.18, -0.39) Vs. -0.10 (0.21, -0.20), p=0.22], PTB1B [-0.003 (0.04, -0.07) Vs. -0.01 (0.06, -0.06), p=0.74] and Cav-1 [-2.45 (0.78, -8.39) Vs. -6.31 (1.68, -8.29), p=0.34]. Further, transcription of insulin-receptor also did not change [0.01 (0.07, -0.06) Vs. -0.03 (0.09, -0.07), p=0.92]. However, within group analysis show significant decreases in Cav-1 mRNA only during sleep restriction (normal: p=0.09; restricted: p=0.003). Importantly, restricting sleep duration was associated with lowering of insulin (0.6 ± 1.9 Vs. -1.6 ± 1.7 uIU/ml; p=0.003), no change in glucose (-1.5 ± 3.8 Vs. -3.7 ± 4.12 mg/dl, p=0.06) and improvement in HOMA-IR index (0.12 ± 0.44 Vs -0.42 ± 0.43, p=0.002).
Conclusion: Chronic sleep restriction does not alter the transcription of cell-signaling regulators in abdominal adipose tissue. Moreover, restricting sleep duration without increasing calorie intake did not seem to decrease insulin sensitivity as determined by HOMA-IR.
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CHANGES IN SLEEP ARCHITECTURE DURING LONG-DURATION SPACEFLIGHT
Piltch, O.[1] Flynn-Evans, E.[2] Stickgold, R.[3]
[1]Harvard College, Cambridge, MA, [2]NASA Ames Research Center, Mountain View, CA, [3]Harvard Medical School, Boston, MA.

Introduction: Previous projects have shown that astronauts sleep significantly worse in mission than on Earth. However, it is unclear