Adaption to sleep restriction:

changes in prolactin and thyrotropin (TSH) suggests compensatory mechanisms

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Objective

To evaluate how the regulation of TSH and prolactin adapt to restricted sleep (both acutely and in the long-term) and subsequent recovery.

Conclusion

Our data indicates that TSH and prolactin are regulated by highly dynamic systems with the likely intention to retain the homeostasis of metabolism at general as well as cellular levels.

Introduction

Lack of sleep is a strong threat to homeostasis and believed to be harmful if continued over time. However, little is known of how our biological systems adapt to chronic restrictions of sleep. The main aims were to evaluate how the regulation of thyrotropin (TSH) and prolactin adapt to restricted sleep and subsequent recovery.

Results

Both variables varied significantly across days (p's<.0001), across time within days (p's<.0001), and interacted between day and time (p's<.001). The sleep dependent prolactin surge was shorter (03-07h), but the maximum increased with continued sleep restriction, +33% the fifth night. Prolactin remained slightly higher the first recovery night, but returned to baseline thereafter (see Fig 1).

Restricted sleep increased TSH acutely (+30% the first 24h), followed by a reduction with ongoing sleep restriction, +6% the fifth day. TSH was substantially reduced the first recovery night, -30% compared to baseline, but had already returned to baseline by the subsequent evening and continued rising above baseline the second and third recovery days; +18 and 24%, respectively (see Fig 2).

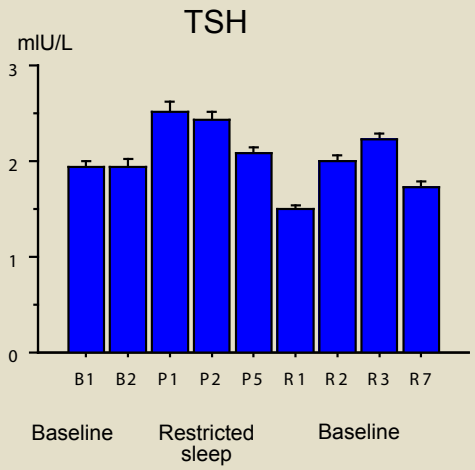

Figure 1. Mean TSH-levels for each day during Baseline (B1, B2), Restricted sleep (P1, P2, P5) and Recovery (R1, R2, R3, R7).

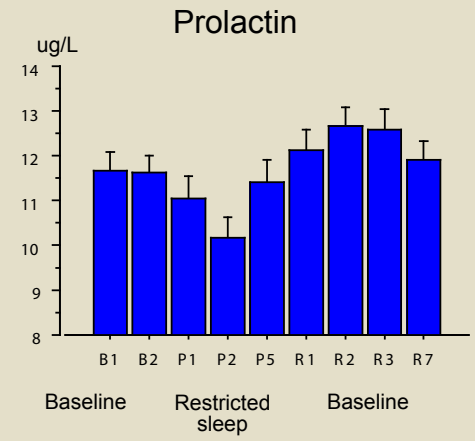

Figure 2. Mean prolactin-levels for each day during Baseline (B1, B2), Restricted sleep (P1, P2, P5) and Recovery (R1, R2, R3, R7).

Methods

Nine healthy males (age 23-27 yrs) participated in the study that included two baseline days with 8 h sleep (23-07 h), 5 days with sleep restricted to 4 h /night (03-07 h); and four recovery days (23-07 h). For nine of those days (see figures), blood was drawn every hour 23-08 h and every 3rd hour 08-23 h.

Discussion

The results add to previous knowledge of a homeostatic relation between sleep (loss) and the endocrine system, but also suggests that other mechanisms are involved in long-term adaptation to restricted sleep. Although compensatory, the homeostatic responses to sleep loss are still part of an allostatic-load, that may relate to future morbidity.

A part of these long-term compensatory mechanisms may be altered sensitivity in the receptor systems, as indicated by recent findings of a desensitization of the serotonin (5HT) 1A receptor system of restricted sleep in rodents (1).

References:

(1) Roman V, Walstra I, Luiten PG, Meerlo P. Too little sleep gradually desensitizes the serotonin 1A receptor system. Sleep. 2005 Dec 1;28 (12):1505-10.

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