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Effect of sleep deprivation on overall 24 h growth-hormone secretion

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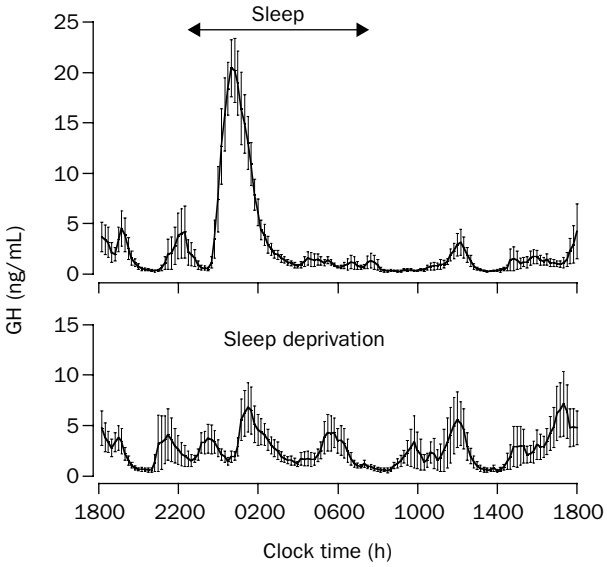
After sleep deprivation, the blunting of the normal sleep-related growth-hormone (GH) pulse is compensated during the day. Consequently, the amount of GH secreted during a 24 h period is similar whether or not a person has slept during the night. These results argue against the belief that sleep disorders in children can inhibit growth through a daily GH deficit.

Growth hormone (GH), an important metabolic hormone that is essential for growth,[1] is secreted in a series of pulses throughout a 24 h period. A major GH pulse occurs shortly after sleep onset, in temporal association with the first slow-wave sleep (SWS) episode.[2] This pulse is attenuated when sleep is altered, and a reversal of the sleep-wake cycle inhibits GH release.[2] Pharmacological stimulation to enrich SWS results in increased GH release.[3] A reciprocal interaction between the stimulating GH-releasing hormone, and the inhibiting somatostatin is primarily responsible for GH release, and weaker somatostatinergic influence during sleep than during awakening has been proposed.[4] We investigated 24 h GH secretion in sleep-deprived people.

Plasma GH concentrations were measured every 10 min over 24 h (1800–1800 h) in ten people aged 20–26 years old, once after night-time sleep from 2300–0700 h and once after complete sleep deprivation for 24 h. The participants gave written informed consent to participate. The study was approved by the local ethics committee. Participants were assessed under constant conditions—continuous enteral nutrition and bed rest in sound-proofed, air-conditioned rooms, with light at 100 lux during waking hours. We measured GH secretory rates from corresponding plasma concentrations by a deconvolution procedure. A one-compartment model for hormone distribution and degradation was used with a participant-adjusted half-life of between 21 min and 18 min. The distribution volume was assumed to be 7% of the participant's bodyweight.

In night-time sleep conditions, the GH rate followed the recognised pattern with a major pulse just after onset that accounted for 58% of the 24 h secretion. During sleep deprivation, the GH pulses were more equally distributed throughout the 24 h and large individual pulses occurred during the day (figure). These pulses could not be related to external events and were not synchronised among participants. The amount of GH secreted during the night



Effect of sleep deprivation on mean (SE) 24 h GH profiles

(2300–0700 h) was significantly lower during sleep deprivation (mean 44·4 [SE 7·7] *vs* 82·1 μg [10·7], p<0·001). However, the amount of GH secreted during the day (0700–1800 h) in awake participants was significantly increased (68·0 [13·8] *vs* 29·5 μg [5·2], p<0·02), so that the total amount of GH secreted during the 24 h period was similar in both conditions (132·4 [12·6] *vs* 136·5 μg [21·8], p=0·20).

Acute sleep deprivation does not induce deficiency of GH release in 24 h. In sleep-deprived participants, GH secretion was increased during the day, which compensated for the blunting of the major sleep-related pulse. Whether a threshold of GH secretion is needed to produce its physiological effect is unclear. Nevertheless, these results argue against the common belief that sleep disorders in children, in pathological cases or in response to adverse environmental influences, can inhibit growth through a daily GH deficit. Care should be taken, however, in extrapolating these results to children, in whom sleep-deprivation tests such as ours cannot be done for ethical reasons. Nevertheless, the results challenge this much publicised concept, based on case reports[5] rather than on epidemiological studies.

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