

Human growth hormone before and after sleep deprivation

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Total sleep deprivation (TSD) has been shown to have a non-pharmacological antidepressant effect in depressed patients. From endocrine profiles a noradrenergic way of therapeutic action has been proposed and could additionally be supported by higher human growth hormone (HGH) levels in responders after sleep and elevated HGH in responders after TSD compared to non-responders (Baumgartner *et al*, 1990).

In this study we therefore tested the HGH response to TSD in 11 male patients with major depression, melancholic type (APA, 1987). All probands had the same antidepressant medication for 2 weeks (150–225 mg amitriptyline without additional medication).

Patients were examined twice. One investigation was started after a night's sleep at 7 am, the other after a night of TSD at the same time of day. Thirty min after inserting a catheter, nine baseline blood samples were drawn in 15-min intervals for 2 h. The probands remained in bed throughout the procedure. Patients were rated after sleep and after TSD with a modified version of the Hamilton scale HDRS (Hamilton, 1967). Items 4, 5, 6, 16, 18 were omitted. TSD responders were defined having a 30% decrease of HDRS.

Specimens were assayed by synchron enzyme linked immuno sorbent assay (Synelisa).

The means of the nine measures were analyzed by analysis of variance (Anova) with repeated measures and *post hoc* U-test. Six patients were responders, five non-responders. The two groups did not differ in age (mean 35 ± 6.5 years), body weight (76 ± 2.1 kg), HDRS-scores (28 ± 5).

There were no significant differences in mean HGH levels between sleep and sleep-deprivation procedures or responders and non-responders, possibly due to the small size of the sample (table 1).

However, trends for lower HGH in responders

compared to non-responders after sleep and after TSD and for lower HGH in responders after TSD compared to measures after sleep do not confirm previous results (Baumgartner *et al*, 1990). Our data could support interpretations that TSD downregulates dopamine or noradrenaline release in responders, but one should be aware of complex mechanisms of HGH release and problems of reliable assessment through pulsatile release. Therefore, for future research, only drug challenging studies before and after TSD should be recommended.

Table 1. Mean HGH levels (from nine measures) before and after TSD in responders and non-responders. No significant interactions (Anova) or U-tests (sleep procedure \times response \times HGH levels).

	Responders (n = 6)		Non-responders (n = 5)	
	After sleep	After TSD	After sleep	After TSD
HGH ng/ml (m \pm SD)	3.10 \pm 5.34	0.93 \pm 1.27	3.32 \pm 4.98	1.78 \pm 1.53

References

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