Neuroendocrine regulation and metabolism of glucose and lipids in primary chronic insomnia: a prospective case-control study

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OBJECTIVES
To investigate the relation between primary chronic insomnia and insulin sensitivity, visceral adiposity, non alcoholic fatty liver disease and neuroendocrine hormones.

MATERIALS AND METHODS
In a case-controlled, prospective clinical trial 13 women with primary chronic insomnia according to DSM-IV criteria were compared to 12 healthy controls matched for age, sex, BMI, body composition and menopausal status. All participants had a sleep assessment including polysomnographic studies and neuropsychiatric evaluation. Insulin sensitivity was evaluated using the euglycaemic hyperinsulinemic clamp. Hepatic fat content, visceral adipose tissue and intramyocellular lipid accumulation were assessed using magnetic resonance imaging and spectroscopy. The hormonal stress axis was evaluated by measurements of midnight and early morning salivary cortisol, urinary catecholamines and plasma metanephrines. Body composition was determined using body impedance analysis and indirect calorimetry.

RESULTS
Although the diagnosis of primary chronic insomnia was made by established clinical criteria, standard polysomnographic studies failed to identify altered sleep continuity and architecture when compared to matched controls. However, women with primary chronic insomnia showed significantly higher midnight salivary cortisol concentrations (1.46 vs. 0.76 nmol/l, p = 0.02), indicating dysregulation of the hypothalamo-pituitary-adrenal (HPA) axis. Plasma glucose and lipid concentrations, insulin sensitivity, hepatic and intramyocellular fat content, visceral adipose tissue mass and body composition did not differ between the two groups.

CONCLUSION
Healthy women with clinically diagnosed primary chronic insomnia demonstrate a dysregulation of circadian cortisol secretion despite normal sleep continuity and architecture. Increased midnight cortisol levels, however, were not
associated with impaired metabolism of glucose and lipids.