

## Introduction

Sleep exerts modulatory effects on components of the endocrine system; depending on the endocrine axis hormonal secretion may be stimulated or inhibited during sleep. Reciprocally, several hormones have effects on sleep architecture (**Chokroverty, 2006**).

The sleep-wake cycle is under dual control of a centrally generated endogenous circadian signal and of a homeostatic mechanism relating sleep duration and intensity to the duration of prior wakefulness. This dual control affects the temporal organization of hormonal release (**Kales, 2007**).

The physiological response of hormonal secretions to sleep is disturbed during sleep deprivation and when sleep is shallow and/or fragmented. Part of the hormonal and metabolic changes observed in older adults could be secondary to the decrease in sleep duration and quality that characterize aging (**Knutson, 2007**).

During the first half of the night, the growth hormone (GH) surge preponderates, whereas Adeno-corticotrophic hormone (ACTH) and cortisol levels are low; during the second half of the night ACTH and cortisol are high whereas GH is low (**Grunstein, 2005**).

Slow-wave sleep has inhibitory influence on Thyroid stimulating hormone (TSH), whereas Luteinizing Hormone (LH) and Follicular stimulating hormone (FSH) have nocturnal surge with puberty (**Jha, 2006**).

Obstructive sleep apnea is associated with obesity, type 2 Diabetes mellitus (DM), hypothyroidism and excess growth hormone (**Aurora, 2007**).