



POSTER NO 11

Chronic Stress and Obesity: A New View of "Comfort Food"

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ABSTRACT

The effects of adrenal corticosteroids on subsequent adrenocorticotropin secretion are complex. Acutely (within hours), glucocorticoids (GCs) directly inhibit further activity in the hypothalamo-pituitary-adrenal axis, but the chronic actions (across days) of these steroids on brain are directly excitatory. Chronically high concentrations of GCs act in three ways that are functionally congruent:

1. GCs increase the expression of corticotrophin-releasing factor (CRF) mRNA in the central nucleus of the amygdala, a critical node in the emotional brain. CRF enables recruitment of a chronic stress-response network.
2. GCs increase the salience of pleasurable or compulsive activities (ingesting sucrose, fat, and drugs, or wheel-running). This motivates ingestion of "comfort food."
3. GCs act systemically to increase abdominal fat depots. This allows an increased signal of abdominal energy stores to inhibit catecholamines in the brainstem and CRF expression in hypothalamic neurons regulating adrenocorticotropin.

Chronic stress, together with high GC concentrations, usually decreases body weight gain in rats; by contrast, in stressed or depressed humans chronic stress induces either increased comfort food intake and body weight gain or decreased intake and body weight loss. Comfort food ingestion that produces abdominal obesity, decreases CRF mRNA in the hypothalamus of rats. Depressed people who overeat have decreased cerebrospinal CRF, catecholamine concentrations, and hypothalamo-pituitary-adrenal activity. We propose that people eat comfort food in an attempt to reduce the activity in the chronic stress-response network with its attendant anxiety. These mechanisms, determined in rats, may explain some of the epidemic of obesity occurring in our society (emphasis added).

EDITOR'S COMMENT

The association of chronic adrenal stress with modern obesity is an important perspective to understand. Adrenal stress occurs in two primary situations: when liver glycogen stores are depleted due to prolonged exercise (or fasting) and near the end of the night fast when one goes to bed without replenishing liver glycogen stores. In addition, modern life styles frequently expose one to 24 hour day increased light pollution both within and without the home (cortisol is the only hormone directly stimulated by light). These conditions all militate against restful or restorative sleep.

Over time (months or years), this potent combination produces chronic adrenal stress and results in chronically distressed sleep, poor sleep architecture, compromised recovery and restoration of body tissues during rest, and inhibition of recovery fat metabolism, resulting in adult and childhood obesity.

Eating honey before bedtime insures adequate liver glycogen stores, improves sleep by virtue of the HYMN Cycle (see Poster NO 14), and improves sleep architecture. Individuals who consume a tablespoon or two of honey prior to bed often report increased dream memories. Dreams are a vital aspect of human sleep physiology and offline memory processing (see Poster NO 15). Honey before bedtime, it may be argued, is effective in promoting restorative sleep and reducing obesity by the mechanism of reducing or eliminating chronic adrenal stress.