Measure weight of animal before left alone for 24 hours, weight of 12 pieces of food then again before brain removal

**Stress, eating and the reward system (Adam and Epel, 2007)**

**doi: 10.1016/j.physbeh.2007.04.011.**

-weight loss as a marker of stress in rats BUT when they have HPF stress increases intake of that HPF

-humans: bidirectional, 30% decrease intake during or after stress, the rest increase

-sympathetic adrenomedullary system (SAM) originates in the locus ceruleus and with the HPA build the effector limbs of the stress response

-CRH neurons of the PVN are the principle hypothalamic regulator of the HPA

-CRH stimulates secretion of ATCH from anterior pituitary

-ACTH on the adrenal glands where it stimulates the release of cortisol or corticosterone

-cortisol feedback back to shut off further secretion to protect the organism from prolonged secretion

-predictors of eating more during stress in humans: female, overweight, scoring high on dietary restraint

-human “threat stress” activates the HPA and cortisol which stimulates hunger and feeding

-human “challenge” activates the SAM and adrenaline which shuts down digestion

-excess glucocorticoids part of obesity via increase food intake and visceral fat deposition

-humans: greater basal cortisol or greater cortisol reactively in people with AN, BED, BN.

-naloxone suppressed intake of HPF.

-stress as a type of negative reinforcement for food intake

-rats: physical stress reduced sugar water intake (vs water) and emotional stress increased it

**Effect of restraint stress on feeding behaviours of rats (Ely et al., 1997)**

[**https://doi.org/10.1016/S0031-9384(96)00450-7**](https://doi.org/10.1016/S0031-9384(96)00450-7)

-neural events guide and trigger behavior but there is peripheral physiological input

-products of digestion act on chemoreceptors

-adult male rats (60-90 days)

-1 hr/day

-“control animals were manipulated but not submitted to restraint”

-acute restraint stress did not increase the intake of fruit loops

-chronic model of moderate intensity increase food intake of fruit loops

**The hypothalamic-pituitary-adrenal axis in the regulation of energy balance (Nieuwenhuizen and Rutters, 2008)**

**DOI: 10.1016/j.physbeh.2007.12.011**

-cortisol binds to transporter in the blood

-binds to glucocorticoid and mineralocorticoid receptors

-GR: initiates or represses transcription, negative feedback of HPA axis

-MR: regulates basal HPA activity

-anorectic effects of adrenalectomy can be reversed by glucocortoid replacement

-CRH neurons in the PVN

-A diagram of a food chain

AI-generated content may be incorrect.

**Chronic stress, metabolism, and metabolic syndrome**

**(Tamashiro et al., 2011)**

-stress has adverse effects including inferring with energy homeostasis and resulting in obesity

-responses to acute stress and protective and adaptive

-chronic stress impairs neuroplasticity

**Stress and obesity: the role of the hypothalamic–pituitary–adrenal axis in metabolic disease**

**(Bose et al., 2009)**

-stress is a challenge to the homeostasis of the animal

-respond by producing physiological stress response to regain equilibrium

-ANS and HPA

-acute short-term stress response is necessary for homeostatic recovery, chronic or prolonged stress can be harmful

-CRH from the PVN of the hypothalamus stimulates ACTH from pituitary

-physical stressors activate PVN neurons that express CRH

-ACTH cortisol from adrenal cortex

-adrenalectomy in Cushing’s syndrome (high cortisol) relives obesity

**Palatable foods, stress, and energy stores sculpt corticotropin-releasing factor, adrenocorticotropin, and corticosterone concentrations after restraint**

**(Foster et al., 2009)**

-previous studies show reduced HPA response to acute and repeated stressors in rats

-adult male SD rats

-only rats with highly palatable sucrose ate more after 30 min restraint stress

**Glucocorticoids, chronic stress, and obesity**

**(Dallman et al, 2006)**

-sustained stressors may leave prolonged traces of elevated glucocorticoids

-chronic elevations of glucocorticoids act differently depending on if they are presently elevated in the presence or absence of a chronic stressor

NOT FINISHED

**ENDOCRINEOLOGY OF THE STRESS RESPINSE**

**(Charmandari et al., 2005)**

-when homeostasis is threatened (or perceived to be threatened)  
-central parts of the stress system are in the hypothalamus (HPA) and brainstem (SAM)

-CRH is an anorexigenic peptide

-glucocorticoids inhibit the PVN CRH and NE sympathetic systems

-diurnal variation in secretion of cortisol and ACTH (which is normal) can be disrupted by changed in lighting, feeding, activity, and following stress

-glucocorticoids are the final effectors of the HPA

-neg. feedback of glucocorticoid on CRH and ACTH

-stress influences appetite satiety centers in the hypothalamus

-acute elevation in CRH concentration causes anorexia

-stress response is supposed to be short/limited

-increased HPA axis activity: chronic stress, anorexia, DM, Cushing syndrome, hyperthyroidism

-prolonged activation of HPA suppresses growth hormone secretion

-glucocorticoids induce insulin resistance