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

-stressor-glucocorticoid-induced plastic effects on the brain that result from persistent stressor may have “deleterious consequences for the chronically stressed organism”

-at high daily doses of glucocorticoids for 3 weeks, it takes days-weeks to return to basal HPA activity after the treatment is stopped

-“in absence of concurrent stress, prolonged treatment with glucocorticoids reduces activity in the HPA axis and blunts responses to acute stressors in proportion to the dose”

-normal response to acute stressor in a stressor-naïve animal: afferent activation of the hypothalamic CRF neuron, secretion to the median eminence to activate the pituitary corticotrope, then ACTH secretion into general circulation, then adrenal cortex where glucocorticoids increase within 2-5 minutes of the stimulus, and soon act to inhibit the CRF and ACTH “secretory responses”

-rapid action of glucocorticoids shorten the duration but not the peak magnitude of stimulus-induced ACTH secretion which is key to limiting the duration of action of the HPA to be able to respond to the threat but not so much that it could be harmful

-(concurrent stress) “sensitization of HPA activity in response to a novel stimulus in chronically stressed rats” only if the glucocorticoids levels are elevated above the normal daily mean

-“sustained treatment with glucocorticoids in the absence of concurrent stress inhibits both basal and acutely stimulated activity in the HPA axis… likely that the inhibition is at the pituitary with **less central inhibition**”

-“however, many low-intensity repeated stressors like restraint, cold, noise, and ethanol provoke habituation rather than sensitization in the HPA”

-highly likely that LC noradrenergic activate the HPA and that LC lesions decrease HPA response to acute stress

-glucocorticoids act catabolically in the periphery, and anabolically in the brain/centrally

-in the brain glucocorticoids promote caloric intake (opposite to in the periphery)

-“in the absence of stress, glucocorticoids strongly stimulate in a dose-related fashion, the ingestion of substances that are pleasurable to the animal”

-incidence of chronic social stress is increase, high calorie HPF are readily available and physical effort needed to acquire them is decreased

-the system of glucocorticoids needed to survive in chronic stress has not yet adapted to our modern climate with ease of access to these foods

**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