**Obesity or EDs (1.1) ~half a page**

-how the world has changed in terms of stress and food availability

**Stress (1.2) ~one page**

Physiologically, stress is a challenge to the homeostasis of an organism (Bose et al., 2009), or the perceived threat to homeostasis (Charmandari et al., 2005). The organism then responds to regain equilibrium (Bose et al., 2009). In humans, stress activates the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic adrenomedullary system (SAM), which originates in the locus ceruleus (LC) to different degrees (Adam and Epel, 2007; Charmandari et al., 2005). Activation of the SAM activates epinephrine which suppresses appetite and stops digestion (Adam and Epel, 2007). The HPA includes corticotrophin releasing hormone (CRH) release from the paraventricular nucleus (PVN) of the hypothalamus which stimulates adrenocorticotrophin hormone (ACTH) release from the pituitary gland which stimulates cortisol release from the adrenal cortex (Bose et al., 2009; Adam and Epel, 2007). In an acute, short-term stressor, cortisol should negatively feedback on CRH and ACTH to prevent prolonged secretion of cortisol (Adam and Epel, 2007; Charmandari et al., 2005) since the acute stress response is necessary for homeostatic recovery, but chronic or prolonged stress can be harmful (Bose et al., 2009).

**Stress and appetite (1.2.1) ~two pages (**sex difference can be here: pandemic example**)**

As part of the normal response to an acute stressor in a stressor-naïve animal, there is afferent activation of the HPA, with glucocorticoid levels increases within 2-5 minutes of the stimulus (Dallman et al., 2006). This rapid action is critical to shorten the duration of ACTH secretion and the HPA so that the threat can be responded too, but not so much that there could be negative consequences (examples) (Dallman et al., 2006). In the periphery, glucocorticoids act catabolically to mobilize energy stores, ensuring enough fuel for tissues such as the heart and muscles, allowing for the energy to escape stressors (Dallman et al., 2006). In contrast, glucocorticoids act anabolically in the brain, driving caloric intake (Dallman et al., 2006). Meaning of that. Chronic elevation of glucocorticoids in the absence of chronic stress inhibits basal HPA activity and HPA activity stimulated by an acute stressor, but this is likely due to inhibition at the pituitary, not central inhibition (Dallman et al., 2006). As LC lesions decrease HPA response to acute stress, it is likely that LC NE neurons activate the HPA (Dallman et al., 2006).

-highly palatable foods leads into sex differences

Davies et al. (2023) found females were at higher risk for pandemic stress-induced binge eating, and females ages 10 to 19 showed the greatest increase in eating disorder released hospitalizations (Auger et al., 2023) during this time. In contrast, acute stressors tend to suppress appetite (Torres et al., 2007).

-brain region transition to next subsection

Cortisol, a glucocorticoid, stimulates hunger and feeding (Adam and Epel, 2007). The balance between SAM and HPA, effectors of the stress response, results in… Chronic stress and excess glucocorticoids play a role in obesity by interfering with energy homeostasis (Tamashiro et al., 2011) and increasing food intake and visceral fat deposition (Adam and Epel, 2007).

But behaviour following an acute stressor is highly variable (Klatzkin et al., 2023).

**-The hypothalamus (1.3) ~paragraph**

**The DMH (1.4) ~half page**

**DMH and food intake (1.4.1) ~one page**

**DMH and stress (1.4.2) ~paragraph**

**-CRH receptors and glucocorticoid**

**Synapses (1.5) ~one page**

**-Glutamate (1.5.1) ~one page**

**Stress on synaptic transmission (1.5.2) ~half page**

**Current Study (1.6) ~one page**

**-females under researched (1.6.1)**