HOW EMOTIONS INFLUENCE APPETITE REGULATION

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APPETITE REGULATION

Balance of energy metabolism is the main regulator of appetite

CENTRAL REGULATION (CNS)

HYPOTHALAMUS: regulating center of appetite and energy homeostasis.

Main Hypothalamic nuclei

- Lateral hypothalamic area (**LHA**): **hunger center**
- Ventromedial nucleus (**VMN**): **satiety center**
- Arcuate nucleus (**ARC**): two distinct neuronal populations expressing:

OREXIGENIC NEUROPEPTIDES
- NPY
- AgRP
- Orexin

ANOREXIGENIC NEUROPEPTIDES
- α-MSH
- CART
- POMC

BRAINSTEM: metabolic signals primarily relay to the solitary tract nucleus (NTS) a major neuronal link between the gut and the brain.

MIDBRAIN: brain rewarding system is involved in the control of hedonic feeding (**mesocortical dopaminergic** pathways)

MECHANISM OF APPETITE REGULATION

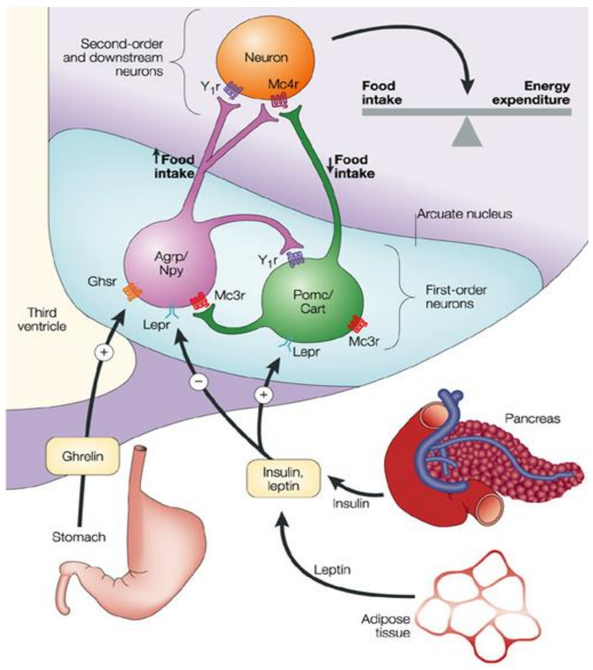


Fig 1. Hypothalamic mechanism of appetite regulation[1]

SEROTONIN

Inhibition of orexigenic NPY and AgRP neurons (5-HT_{1B} receptors)

Activation of anorexigenic POMC neurons (5-HT_{2C} receptors)

Decrease of food intake

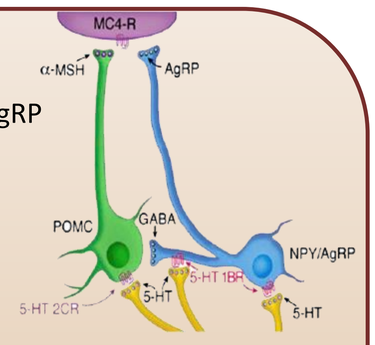


Fig 2. Serotonin role in food intake[2]

PERIPHERAL REGULATION

ANOREXIGENIC HORMONES
- Leptin
- Insulin
- Cortisol

OREXIGENIC HORMONE
- Ghrelin

STRESS

Stress exposure induces changes in brain development and behavioral outcomes affecting **eating patterns**, as stress and feeding systems share the same neuroanatomy.

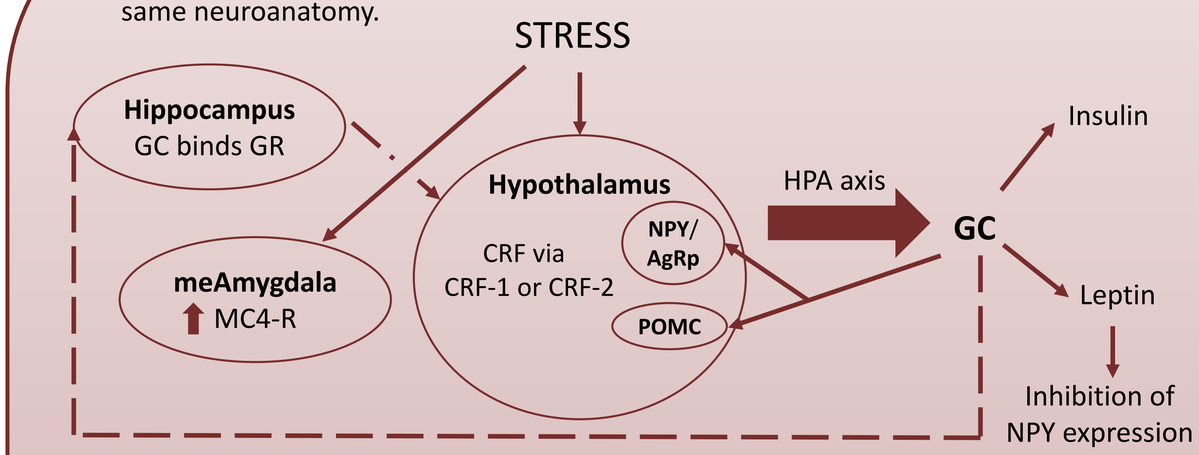
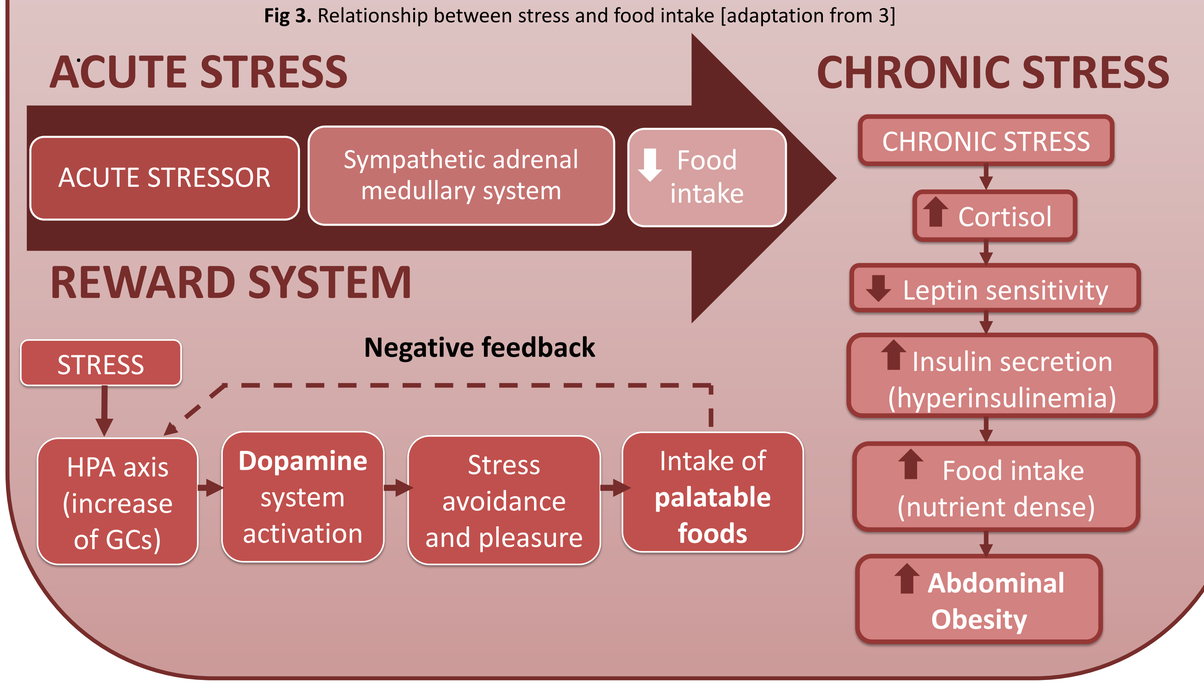


Fig 3. Relationship between stress and food intake [adaptation from 3]



DEPRESSION

Depression is characterized by fast fatigue, loss of interest for normal activities, altered HPA axis activity, chronic stress and monoamine deficiency.

HYPERACTIVATED HPA AXIS

DEPRESSION → Hyperactivated HPA axis → ↑ Cortisol

High cortisol levels can produce:

- Damage to hippocampal neurons
- Hippocampal atrophy which disturb cortisol negative feedback to hypothalamus and HPA axis (**hypercortisolemic state)**
- Leptin resistance and increase of NPY secretion
- **Increase of** food **intake** and **abdominal obesity**

SAME RESULTS AS IN CHRONIC STRESS

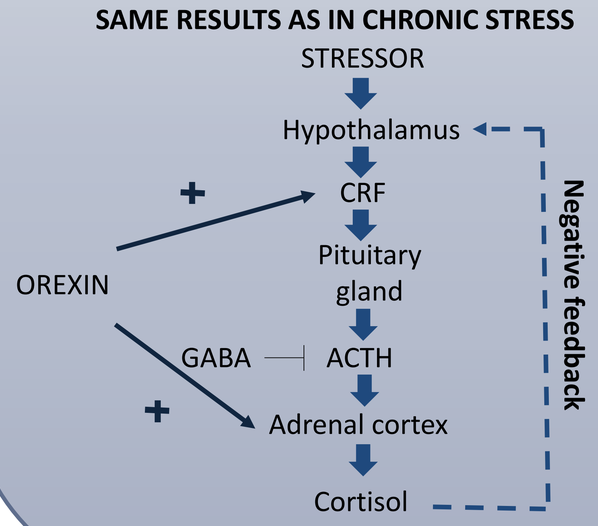


Fig 4. HPA axis normal activity which is altered in depression

SEROTONIN

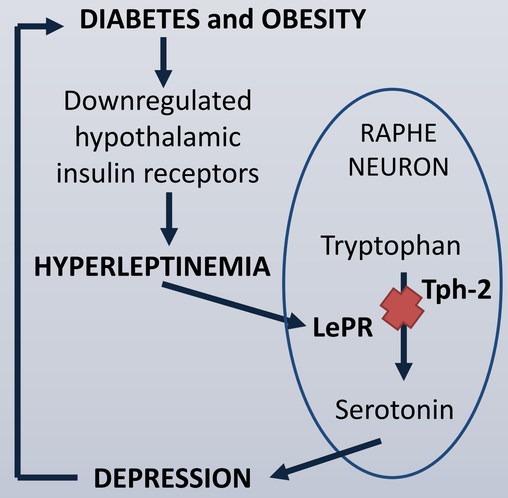


Fig 5. Disregulated serotonin system induced by leptin in depression [adaptation from 4]

Low serotonin levels:

- Reduce the activation of 5-HT_{2C} POMC neurons receptor
- Decreases an inhibitory drive onto AgRP neurons by activation of 5-HT_{1B} receptors
- Increase of NPY secretion **increase of food intake**

OBESITY and **DIABETES**

CONCLUSIONS

APPETITE REGULATION

- Hypothalamic control of appetite involves the modulation of **orexigenic** and **anorexigenic** pathways that determine the positive or negative balance between food intake and energy expenditure.
- Peripheral components are in a bidirectional communication with the brain through the autonomic nervous system and hormones.
- **Insulin** and **leptin** inhibit the orexigenic NPY/AgRP neurons and activate **anorexigenic** POMC neurons resulting in a decrease of food intake.
- **Ghrelin** activates AgRP/NPY neurons and stimulates food intake.
- **Serotonin** acts on POMC neurons to induce melanocortins secretion and thus satiety.

INFLUENCE OF EMOTIONS ON APPETITE REGULATION

- Individuals regulate their emotions and mood by changing both food choices and quantities.
- **Acute stress** activates the sympathetic adrenal medullary system and is associated with a **decrease** of **food intake**.
- **Chronic stress** produces a hyperactivation of the HPA axis, increases cortisol levels and comfort food intake, which leads to **abdominal obesity**.
- **Depression** is associated with chronic stress, altered HPA axis activity and elevated cortisol levels which lead to **abdominal obesity**.
- Depressed patients suffer a **dysregulation** of the **serotoninergic system** resulting in a reduction of serotonin levels and an increase of food intake.

REFERENCES

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