homeostasis

- **describe homestasis and explain its significance**
  - ability to maintain relatively stable internal conditions even though the outside world changes continuously
  - literal translation is “unchanging”
  - it indicates dynamic state of equilibrium- internal conditions constantly vary, but always relatively the same.
  - Homeostasis happens when organism fed and functioning smoothly
  - adequate blood levels, monitored heart activity, waste is expelled. Chemical, neural, thermal factors.
  - Bases off of communication, neural electrical impulses or blood-borne hormones.
  - Three components : Receptor -> control centre -> effector
  - receptor- sensor that that responds to stimuli, sending info to control centre via afferent pathways (sensory neurons)
  - Control centre – determines appropriate response, then sends to effector via efferent pathways
  - Effector- efferent, motor neurons. Feedback.
- **describe how negative and positive feedback maintain body homeostasis**
  - most homeostasis control mechanisms are NEGATIVE
  - negative - makes a change opposite to one that naturally occurred or was triggered to occur
  - for example, the environment is hot so your body tries to get cooler (sweating is the mechanism)
  - reflexes (pulling away from hot stove)
  - humoral example is when insulin is released (that’s the mechanism) to control glucose level
  - the result of the POSITIVE mechanism increases the effect of stimulus.
  - Referred to as cascades
  - normally happens in infrequent events
  - for example, oxytocin intensifies labour contractions during baby birth. Makes contractions more frequent and more powerful.
  - Likely to go out of control, so less common. Safer not to have them.
  - Can be regional.
  - Platelets clot blood. Cut happens in vessel, then platelets fill in gap, and release of chemicals to attract more platelets.
  - **describe the relationship between homeostatic imbalance and disease.**
  - Bodies get less stable/efficient as we get older and we become more prone to disease
  - when negative feedbacks are overwhelmed and positive ones get out of control.
  - EXPLAIN MORE INTO DETAIL
  - too active a thyroid is an immune system disorder, can lead to things like graves disease
- **Nervous system:**
  - **Compare somatic and autonomic nervous systems**
    - they differ in their effectors, their efferent pathway and ganglia, and their target organ responses to their neurotransmitters.
  - *ANS*

        - referred to as involuntary or subconscious. General visceral motor system.
        - Effectors: Smooth and cardiac muscles and glands.
        - Efferent pathways and ganglia: thicker, heavily myelinated. two neuron chain transmission-preganglionic neuron is brain or spinal, but then the postganglionic neuron is the second motor neuron, outside CNS (muscle)
        - because its a chain its slower than SNS
        - neurotransmitter effects: two. NE(norepinephrine) sympathetic, ACh by parasympathetic. Can be exictatory or inhibitory.
    - *SNS*
        - effectors: skeletal muscle
        - efferent pathways: SNS's motor nerves are through the CNS and lead to cranial and spinal nerves. Motor system has NO GANGLIA.
        - Neurotransmitter effects: all somatic motor neurons release ACh(acetylcholine) to skeletal. Effect is excitatory.
- **Compare the functional differences between the sympathetic and parasympathetic divisions of the ANS**
    - chapter 15/16 notes pdf
    - anatomy of ANS in pages 527-538
    - both serve the same organs, just in opposite ways
- **parasympathetic**
    - maintenance and energy conservation-rest and digest.
    - Sympathetic can turn off parasympathetic
- **sympathetic**
    - excitatory system -fight or flight
    - ity during activity – promotes constriction of some blood vessels (visceral and sometimes sutaneous) blood to shunt blood. Dilates bronchioles so beath deeper for more O2. Causes liver to release more glucose to keep up increased energy.
    - Temporarily shuts off ANS so can focus energy.
- **Endocrine system:**
- endocrine use hormones to communicate instead of electrochemical nerve pulses
- hormones are secreted by way of extracellular fluids.
- Last longer than electrical impulses, happen slower.
- **Control**
    - reproduction,
    - growth and development
    - electrolyte, water, nutrient levels in blood
    - metabolism and energy
    - mobilization of body defences
- endocrinology is the study of endocrine glands and hormones.
- Endocrine organs (glands) in the body are small, spread out in body.
- **Distinguish between exocrine and endocrine glands, and localize the major endocrine glands**
    - EXOCRINE glands release non-formal substances onto membrane surfaces through ducts. (tears, sweat, saliva)
    - ENDOCRINE (ductless) glands, release hormones into surrounding tissue, then hormones are received due to vascular and lymphatic drainage.

- MAIN ENDOCRINE glands
    - pituitary-in the brain, controlled by and below hypothalamus
    - thyroid-butterfly gland in the throat
    - parathyroid- dorsal to thyroid
    - adrenal-top of the kidneys
    - pineal-behind the brain stem
    - hypothalamus-NEURALENDOCRINE-produces and releases hormones
- other organs release hormones, but they just have endocrine cells or something

- **Describe the different structural classes of hormones and their mechanisms of action**
    - **<u>CLASSES</u>**
    - HORMONES long distance chemical signals that travel through blood and lymph
    - autocrines are a no – they chemically exert their effects on the same cells that secrete. Local is not hormone.
    - Paracrines also local, but affect more than just their own secretion cells.
    - Nearly all are either AMINO ACID BASED /or/ STEROIDS
    - AMINO ACID BASED
        - includes most hormones.
        - Molecules can be amino acid derivatives (simple), peptides (short chains), or proteins (long polymer chains)
    - STEROIDS
        - made of cholesterol
        - only includes gonadal and adrenocortical (of major glands)
    - EICOSANOIDS
        - third class only some researchers include because mostly paracrines or autocrines (highly localized aka within the same tissue)
        - includes leotrienes and prostaglandins (lipids)
        - leotrienes:mediate inflammation and allergies
        - prostaglandins: have more extreme varied effects like blood pressure and birth
    - **<u>MECHANISMS</u>**
    - pages 593-598
    - hormone's response in target cell depends on cell type (muscle, or other)
    - cells alter one of the following in a target cell:
        - membrane potential and/or membrane permeability (ion channels)
        - stimulates enzyme or other protein production
        - activates or deactivates enzymes
        - initiates cell secretion
        - initiates mitosis
    - All either
        - water soluble(all amino acid based except thyroid)-act on membrane receptors using G proteins that act on second messengers.
        - lipid soluble(steroid and thyroid)-act inside cell
    - *Plasma membrane receptors and second messenger systems*
    - amino acid based hormones (excluding thyroid) use second messengers to emit their effects intracellularly
    - second messengers are activated when a hormone binds to a membrane receptor.

- Examples: cyclic AMP (cAMP) which is used by neurotransmitters and olfactory
- **cyclic AMP signalling system**
    - uses hormone receptor, G protein, effector enzyme(adenylate cyclase). Level of cyclic AMP internally depends on balance between cyclic nucleotide phosphodiesterase (PDE) and Adenylate cyclase
    1. hormone binds receptor (in plasma membrane)
    2. receptor activates G protein(intracellular)
        - It's off when GDP(guanosine diphosphate) is bound to it
        - it's on when GTP (guanosine triphosphate) is bound to it
    3. G protein then activates adynal cyclase (Gs stimulates, Gi inhibit)
    4. adynal cyclase produces cAMP using ATP
    5. cAMP then binds to target protein kinases which starts cascade (sequence of chemical reactions). Protein kinases then phosphorolate, until kinase is degraded by the enzyme phosphodiesterase.
- **PiP2- signalling system**
- membrane phospholipid
- intracellular calcium ions act as a second messenger.
- Uses G protein, membrane bound effector, phospholipase C
- Phospholipase C splits PIP2(**p**hosphatidyl **i**nositol bis**p**hosphate) into two second messengers : diacyllglycerol (DAG) and inositol triphosphate (IP3).
- DAG, like cAMP, activates kinase. IP3 releases calcium from storage intracellularly.
- Calcium then either does the wished response, or sends to calmodulin to do.
- **other signalling mechanisms**
- cyclic guanosine monophosphate (cGMP) second messenger for some hormones.
- For insulin its tyrosine kinase that is activated by autophosphorylation when insulin binds.
- Insulin receptors and has docking sites for relay proteins that trigger phosphoralation.
- **Intracellular receptors and direct gene activation**
- steroids diffuse into their target cells since they're lipid soluble
- then they bind to and activate receptors
- the complex then makes it way to chromatin to specific DNA
- when receptor hormone binds to DNA, it turns on a gene -prompts transcription to produce mRNA for that gene
- then the DNA replication process happens

- **Target cell specificity**
    - in order for a cell to respond to a hormone, it must have specific receptors for that hormone or its messengers.
    - Thyroxine found in most cells for metabolism, but ACTH (adrenocorticotropic hormone) are only found in specific cells in the brain.
    - Whether or not hormone has affect depends on:
        1. blood levels of hormones
        2. number of receptors for hormone in the target cell.
        3. Strength of the binding between hormone and receptor
    - up regulation- lower levels of a hormone cause target cells to produce extra receptors
    - down regulation- prolonged exposure to high hormone concentration.
- **Control of hormone release**

    - negative feedback mechanism-regulates levels of hormones, stops or inhibits secretion.
    - Nervous system can turn on/off any of these (sympathetic controls parasympathetic)
    - Endocrine gland stimuli : humoral, neural, hormonal stimuli
    1. humoral - release hormones in direct response to changing hormone blood level
    2. neural – nerve fibres stimulate hormone release (stress response, sympathetic)
    3. hormonal – response to other hormones' release. Promote rhythmic release.
- half life, onset and duration of cell activity
- interaction of hormones at target cells.
- **Half-Life and duration**
- hormones can be free or bound to carrier. Mostly lipid-soluble (steroids and thyroid).
- Concentration reflects (1) rate of release (2) speed of activation and degradation
- the length of time for a hormone's blood level to decrease by half.
- **Hormonal interaction**
- permissiveness: situation in which a hormone can't be fully productive without another hormone present.
- Synergism: more than one hormone produces the same affect in a target cell.
- Antagonism: hormone is released to produce opposite effect of another hormone.
- **Describe the functional organization of the hypothalamic-pituitary axis**
    - affects your stress (fight or flight) response
    - companion to the sympathetic nervous system
    - its an example of a cascade
- **Parathyroid gland**
- parathyroid hormone(PTH)- most important hormone controlling calcium in blood.
- Stimulates osesteoclasts(bone -reabsorbing cells) to digest some bone and release calcium into blood
- enhances reabsorption of calcium by kidneys
- promotes activation of calcium, which in turn increases calcium absorption.
- **The pineal gland**
- diencephelon
- contains salt
- major secretion of melatonin-controls antioxidant and detoxification levels in blood.
- Receives input from visual pathways (retina -> sprachiasmatic nucleus of hypothalamus -> superior cervical ganglion -> pineal gland)
- melatonin can affect growth and sexual maturity.
- Superchiamatic nucleus – biological clock
- **Other endocrine glands and tissues**
- pancreas forms hormones using islets
    - alpha cells – glucagon synthesizing. Hyperglycemic.
    - beta – insulin synthesizing. Hypoglycemic.
    - Somatostatin, pancreatic polypeptide (PP) also other peptides
- Glucagon
    - makes glucose from lactic acid(glucogenolysis) and noncarbohydrate molecules (gluconeogenesis)
    - release of glucose to the blood by liver cells.
    - Also lower blood levels of amino acids.

    - Falling blood levels prompt alpha cells to secret flucagon, or sympathetic NS.
    - Glucagon is suppressed by rising levels of glucose, insulin, and somatostatin.
- Insulin
    - part of proinsulin peptide chain. Removed from chain right before beta cells release.
    - Lower blood glucose levels, normally right after eating.
    - Enhances membrane transport of glucose.
    - Inhibits breakdown of glycogen to glucose.
    - Inhibits amino acids or fats from becoming glucose to monitor metabolism.
    - Participated in neuronal development, important for behaviour, and memory.
    - Receptor for phosphorylates
    - insulin release triggers enzymes to:
        1. oxidize glucose for ATP production
        2. join glucose to form glycogen
        3. convert glucose to fat (especially for adipose tissue)
    - excess glucose becomes fat, insulin promotes protein synthesis and fat storage.
    - *Factors that influence insulin release*
    - high blood glucose, amino acid, or fatty acid
    - other hormones, ay hyperglycemic hormone (glucagon, epinephrine, GH, thyroxine, glucocorticoids).
    - Too much insulin results in hypoglycemia, or low blood glucose levels.
    - Thirst, lots of urination, weight loss, all signs of diabetes and bad homeostasis
    - pages 598-604, 611-612, 617-620