ECS	
Organs	-Pituitary thyroid parathyroid adrenal pineal thymus -(endo&exo): panc gonads -(neural&endo): hypothalamus -(other hormones producers): fat cells SI walls stomach Kidneys heart
Types	-endocrine: released by glands to affect target cells(TC) -paracrine: released into ECF(interstitial) to affect variety of TC -autocrine: the same secretor is the TC -NT: usually have a local effect -neuroendocrine: released into circulation to affect TC -cytokines: Pr released into ECF to fun as (auto, para, endo) 　　　　　(e.g.: interleukin, lymphokine, adipokine) (auto & para are not really hormones, cuz hormones must have a distant effect not local)

Hormones		
transport	-hydrophilic: dissolve in plasma (Pr & CAT) -lipophilic: bound to plasma Pr - inc hl & work as reservoir 　　　　　(steroids & thyroid hormones)	
Types	Pr	-**parathyroid**, **PG**, **panc**(insulin & glucagon) -stored until needed -synthesized as prehormones then posttranslation into prohormones, then into hormones
	steroids	-**adrenal cortex**(cortisol & aldosterone) **ovaries & placenta**(progesterone & estrogen) **testes**(testosterone)
	AA	(only aminoacids, not full Pr peptide chains) -**thyroid**(thyroxin & triiodothyronine) **Adrenal medulla**(E & NE)
MOA	Hormone binds to receptor - enz activation - release of 2ndM - effect	
Effect on	-ion channels 　　-G Pr receptor 　　-enzymatic receptors -IC receptors (genetic activation - thyroids & steroids) (receptors are highly specific to one hormone)	
Receptors site	-surface (Pr, CAT) -cytoplasm (steroids) -N (thyroid)	

MOA of 2ndM	(cAMP) Hormone binds to receptor, then whole binds to G Pr - G Pr displaces GDP for GTP to get activated - active G Pr activates adenylate cyclase enz(ACE) - active ACE transforms ATP into cAMP - cAMP activates Pr kinase to cause cellular effect (mem phospholipid) Hormone binds to receptor - activation of G Pr - active G Pr binds and activates phospholipase enz(PLE) - PLE splits phospholipid PIP into DAG & IP3, both act as 2ndM - DAG activates Pr kinase while IP3 triggers the release of stored Ca - Ca(3rdM) causes effect (tyrosin kinase) -surface receptor -used by insulin & GH -formed of 2 unites that bind to form a dimer once insulin binds -active TK phosphorylates signaling subs thus inducing growth
Steroids & thyroids	diffuse into ICF - activates IC receptor - ReceptorHormonComplex travel to N to bind to DNA receptor - produces mRNA - produces Pr that causes cellular effect
Target cells	-activation depends on: Blood level of hormone # of receptors The affinity of receptors to hormones -Up/Down regulation is the inc or dec of # of receptors due to specific conditions related to the hormone
Conc in circulation	-forms of hormones in blood: free or bound -indicates: release rate, and degradation rate -measurement: RIA(RadioImunoAssay) ELISA(Enz-Linked ImmunoSorbent Assay)
Removal from circulation	-degradation enzs -kidnyes -liver (free hormones are easier to clear than Pr-bound ones)

Types of interactions	-permissiveness: the need of 2+ hormones to cause an effect (thyroids-GH, def. in infants causes dwarfism) -synergism: the effect of 2+ hormones together is much greater than each alone summed up (blood glucose levels **with** glucagon, cortisol & E) -antagonism: hormones oppose the action of one another (glucagon antagonizes insulin, calcitonin antagonize parathyroid)
Regulation	-by positive & negative feedback mechanism -hormones are synthesized/released in response to: humoral, neural or hormonal stimuli
Regulation MOA	-negative feedback: Most common (LH from PG stimulates testosterone release, which will inh LH further secretion) -positive feedback: rare (LH stimulates estrogen release, which will greatly stimulates further LH release)