GC (cortisol)	
Origin	Sugar derivative
Producers	Fasciculate & reticularis
Fun	-Causes hyperglycemia (from its name man!...) -liver glucogenesis -AA mobilization (moving from muscles to other organ…) -essential for stress response (once sudden stress, it catabolizes fat & secretes glucose, providing energy for stress-imp-organs)
TC	All body, almost…
Regulation	-ACTH (regulated by CRH mainly & ADH) -highest peak in morning & lowest in late night
Effects	**Metabolisms** -Ca: anti-Vit D (causes osteoporosis) -Carbs: glucogenesis from AA of muscles (muscles atrophy) Enhance liver glycogen formation Inh glucose usage by the body -fat: mobilization by activation of HSL & using Beta-oxidation (end result is ketone bodies) -Pr: mobilization of AA from muscles (Pr catabolism in all body & anabolism in the liver only) HyperPremia (as they trans. To the liver) **Anti-infl** -inh infl mediators: prostaglandins & leukotrines … (via inh phospholipase A2) -greatly reduces swelling (less capil. Perm.) -dec histamine effects -stablizing lysosomes membranes -dec production of eosino. & lympho. (thus less Ig) **CVS** -maintains blood volume (less capil. Perm.) -cortisol plasma levels vary with water intake, so it does have an action similer to aldosterone, but not remotely as potent -sensitizes arterioles to E (permissive effect) **Renal** -maintain normal renal fun & enhances its excretions **CNS** -emotions perception alter

	GIT -inc HCL secretion -dec SI absorption **Development** -permessive regulation of normal maturation -inh linear growth (affects bones & CT) (if absent → gigantism) **Resp** -surfactant synth (phospholipid maintaning alveolar surface tens)

Path	
Hyper-cortisol-emia	**Causes** -primary: AGC pathies (dec: CRH & ACTH. Inc: cortisol) -secondary: PG pathies (dec: CRH. Inc: ACTH & cortisol) HT pathies (inc: everything)
Cushing	-is chronic high GC levels in blood -can be caused by an ectopic ACTH (lungs) -signs: Abnormal fat distribution (central obesity, buffalo hump & moon) Stria DM -treatment: treating the underlying cause
AGC insuff	**Cause** -primary: <u>Addison</u>, Ai, Tumors, inf, hemorrhage, metabolic failures & ketoconazole (med) -secondary: PG & exogenous suppression **Symptoms** -fatigue -nausa -weight loss -skin hyperpigmentation -hypotension -women loss of axial & pubic hair -hypoglycemia -stress intolerance **Adrenal crisis** -asthenia (fatigue) -severe abdominal pain -vascular collapse **Treatment** GC & MC replacement therapy

Androgen (men's hormone)		
derivatives	-Cholesterol: progestin -progestin: GC, MC & androgens -androgens from AG have 20% less activity	
Manner	Paracrine	
Fun	-exert muscarinic effects (man-ification) -anabolisms & growth (inh fat deposition & inc muscles mass) -sperms production "spermatogenesis" (by sertoli cells) -forming penis & scrotum (ballsack…) -related to feeling anger! -lipido control (sexual desires)	
Forms	-testosterone (the most active form)	
Class	-androgens are the family that include: DHEA & androstenedione	
Zona reticularis	-produces: androgens & DHEA (can be converted into estrogen) -controlled by: ACTH -TC: all body	
Sex hormones	-in both sexes AGC produces both <u>Androgens</u> & <u>Estrogens</u> (the hormone of the opposite sex is produced in a small amount) -other extra-AGC organs produce little sex hormones too. -little of testosterone in male can be converted into estrogen by <u>aromatase</u>, found in adipose tissue (female get estrogen this way! First they have lots of testesterone, then they convert almost all of it into estrogen, in males the enzyme is little active. This also explains the presence of opposite sex hormones)	
In females	Role	its produced as an intermediate step in the synthesis of estrogen however, a small portion escapes into the circulation
	fun	-axillary & pubic hair growth -pubertal spurt (the sudden marked growth in females after puberty) -developing & maintaining females drive (characteristics) -pre-delivery myometrium relaxation, to prevent premature uterine contraction (during delivery & preg.)

DHEA	-derivative of cholestrole from Androgens family والنعم... -synthesized in AGC -it's the primary precursor of estrogen -it's the most abundant androgen
Andro-stenedione	-family: androgens -structure: steroids -produced by: AGC, testes & ovaries -role: gets converted into testosterone then to estrogen if needed, in peripheral tissue especially fat. (highly active in women during postmenopausal, to replenish the lost estrogen) -used by athletics as a body-building supplement (cheating)