Thyroids	
transport	-T4: boundly (unusable until its free) -binds to: TBG (thyroxin-binding globulin), albumin, transthyretin
usage	tissues deiodinate T4 to T3, which is the active usable form.
rT3	-T4 can be converted to rT3 (reversed T3) which will <u>locally</u> deactivate T3 to prevent hyperdose toxicity
Fun	-maturation & metabolisms -thermogenesis
Regulation	(HT senses negative FB and releases TRH that stimulates PG to release TRH that stimulates thyroid to releases its hormones) -high thyroids in blood suppresses TSH & TRH
Tests	**TSH measurement** -test of choice -high amounts indicates hypotsm (after treatments initiation, takes 2 months to go back to normal) -sensitive **T4 measurement** -free&bound or free alone -assays thyroid fun -used to monitor <u>antithyroid treatments</u> or <u>thyroids supplements</u> -sensitive **T3 measurement** -rise of T3 is independent of T4 (cuz even if u have significant rise of T4, tissues will still convert only the needed amount to T3) -used to early diagnose T3 toxicity **Ig** Graves & hashimoto

Pathies: Goiter, hypotsm & hypertsm	
Goiter	
Is	Enlarged thyroid gland (the gland might be normal “euthyroid”)
Etiology	-iodine def -selenium def -hypertsm & hypotsm

Hypotsm	
In	**Infants** -irreversible CNS damage -diagnosed by serum TSH & treated by supplements -causes cretinism if untreated **Children** (delayed maturation, puberty & dwarfism)
serum	-hypercholesterolemia (due to dec LDL receptors on the liver & failure of GIT sterol excretion) -low TSH (indicates primary pathies of thyroid gland)
Class	**Primary** (mostly**,** is thyroid gland pathies) **Secondary** - HT-PG-thyroid axis pathies - in this case treat the primary disease and give supplements
Etiology	-hashimoto -meds -TSH def -congenetal -hypertsm treatment -iodine def -thyroids might be OK, but tissues are diseased and cant use them -conversion of most T4 to rT3
Treatment	**treatment is supplements for life…** -T4 supplement tablets -iodine supplement (**might** cause further sever hypotsm "wolff-effect", that's why monitoring is VIP)

Hypertsm	
Etiology	-PG hypersecretion of TRH (secondary cause) -graves -toxic multinodular goiter -thyroiditis -thyroid tumors (adenomas) -intake of iodine (diet or iodine-containing drugs) -intake of thyroids supplements
Damage by	Mainly thyrotoxicosis
Serum	-NO TSH & excessive thyroids (indicate primary hypertsm) -conginital TBG def can greatly alters the results -total T4 (free&bound) will inc in pregnancy due to inc TBG due to inc estrogen (normal T4 will be released from the gland as free, then immediately binds to TBG, this will happen over and over again, till there is no free, it will stimulate negative FB and the cycle cont)
Treatment	-antithyroid drugs -radioiodine -surgery

Thermogenesis	
Is	Heat production
Info	-we are homeothermic (keeping our body at a constant degree)
Types	**Obligatory** -is basic thermogenesis due to metabolisms (thermohemeostasis) -depends 30% on thyroids **Facultative** -extra thermogenesis on demand, in response to cold -catabolizing brown adipose tissue by sympathetic stimulus & thyroids (in hypotsm, its action is greatly reduced)
MOA	-subs oxidation produces ATP & a little of heat (thyroids inc heat production "reduces ATP synthesis efficiency") -some ATPs are burnt to provide heat -uncoupling of Pr UCP, is the spinning proton pump within the inner mem of brown adipose mitochondria (used to make ATP) (releases heat via exothermic protons movement down their gradient without ATP relation)

Organ	Stimulated by	Effect
HT-PG axis	-FB	-Alter thyroids release
Brown adipose tissue	-Sympathetic -T3	-lessens body's weight -produce heat
White adipose tissue	-Sympathetic -T3	-lessens body's weight -lipolysis
Liver	-Lipolysis	-alter cholesterol & lipid metabolisms -alter bile synth & release
Panc	-T4	-inc local T3 to affect beta cells
Muscles	-T4 -bile	-inc local T3 to inc energy