pituitary gland - P. lobe	
is	down growth of HT
Connected to HT by	Hypothalamic hypophyseal tract HHT, which is actually the axons of the HT N
Hormones	**ADH Oxytocin** (these are directly released into the P. lobe, unlike A. lobe where HHPS takes the R/I hormones and deliver it to A. lobe) (it doesn't synthesize hormones! It stores the one HT have made)
Histo	-herring bodies: a storage place that holds ADH or OX inside, but not both -pituicytes: the barrier between axons and circulation, have a great effect on negative FB

ADH	
Aka	Vasopressin
Made by	Supraoptic N
Stages	Pre-pro-hormone → nonapeptide (9AA) → ADH
Receptors	**-V1a**: VC & liver glycogenolysis **-V1b**: A. lobe of PG (inc Adrenocorticotropics release) **-V2**: in main cells of <u>distal convoluted tubules</u> & <u>collecting ducts</u>
MOA (V2)	binds to its receptor in kidney → triggers cAMP system to synthesize <u>aquaporin Pr</u> → the Pr is released into lumen & causes massive change in cells permeability to H2O, allowing huge amount of H2O into the circulation, resulting in conc. Urin **(inc urine osmolality)**
Regulation	**osmotic pressure:-** by osmoreceptors in HT (if the plasma have lots of Na then its highly osmotic "more conc." so HT will secrete more ADH and stimulate thirst, vise-versa) **Blood volume:-** By <u>baroreceptors in ICA/aortic arch</u> & <u>stretch receptors in left atrium - (inc BP = less ADH, vise-versa)</u> In a nutshell: inc blood osmolality(conc.) or dec its volume, stimulates ADH release

Factors R/I	**Stimulants:-** **(VIP)** -inc serum conc. -pain -nausea (very effective) -hypoglycemia -nicotine -opiates (pain killers) - stress -anti-neoplastic meds -solutes that have no or slow penetration of a semiper. Barrier, cause ADH release (like Na) **Inhibitors:-** -less serum conc. -ethanol -alpha agonists -atrial natriutic peptides ANP -subs that very rapidly enter the barrier, don't change osmotic pressure, thus doesn't stimulate release of ADH
Path	**SIADH** Syndrome of Inappropriate ADH -is hyponatremia & plasma hypo-osmolality (less conc) -caused by inappropriate cont secretion of ADH, or of its action **Diabetes Insipidus** -neurogenic: failure of hypophysis to make ADH -nephrogenic: failure of kidneys to response to ADH

Oxytocin	
Made in	Paraventricular N (stored along ADH in P. lobe of PG)
Fun	-very strong uterine contraction "myometrium contr" (delivery) -triggers milk ejection "triggers letdown reflex" (contr of myoepithelial cells of milk alveoli)
Regulation	-mechanism: Positive FB -S: -**Love hormone** (hugging, touching, orgasms "in both sexes", ejaculation in men "contr of vas deferens") -Stress -I: OH
Autism	JSE Autistic people have significant low plasma conc of Oxytocin. Its found quite elevated in those whom are socially successful

SUMMARY

	Osmoreceptors	Baroreceptors
Site	HT	ICA & aortic arch
Senses	Plasma osmolality	BP
Signals once…	Receptor activated	Receptor suppressed
Stimulated once…	1% above 280 mosm	15% drop
ADH released	Small amount	Huge amount (causing VC)

	Pars distalis	Pars nervousa
Embryo origin	Oral cavity	Down growth of HT
Hormones	GH, TSH, FSH, LH, ACTH, PRL	ADH, Oxytocin
Hormones origin	A. lobe	HT
Blood supply	S. hypophyseal artery	I. hypophyseal artery
HT control	By releasing R/I hormones into HHPS that delievers it to A. lobe	Direct neural signals to P. lobe