Insulin	
Structure	Pr (two amino acid chains linked by disulfide bonds)
Synth as	Proinsulin
Active frm	C- peptide (51 AA)
Hl	6 minutes
MOA	Hyperglycemia → glc enters beta-cells → glc is metabolized making more and more ATP → high IC ATP conc triggers the closure of K channels → no K leaves the cells → high IC K triggers depol. → opening of Ca channels allowing Ca in → Ca binds to insulin granules → release of insulin
Secretors	-high blood: glc, AA, KB -eating a meal (para symp activation + GIT hormones) -hormones: GIP "gastroinh Pr", glucagon, gastrin, CCK, secretin, VIP, E "on Beta-receptor"
Inh	-low blood: glc, AA, KB -symp -hormones: somatostatin & E "on alpha-receptor"
Receptor	-tyrosine kinase family -transmembranal (two alpha subunits and two beta subunits) **Types:** GLUT1 (on erythrocytes, brain) GLUT2 (liver, panc, SI) GLUT3 (brain) GLUT4 (insulin sensitive - on muscle, adipose tissue)
Action time	**Rapid** S Liver, Muscles & Adipose uptake of: glc, AA & K "potassium" **Intermediate** M -Pr anabolism & lessens its catabolism -glycogen synth -glycolytic (NOT glycogenolytic) -inh glc phosphorylase (it breaks glc to give energy) **Delayed** H Lipid synth

Fun	**liver** -anabolise: Pr, lipid, glycogen(by GLUT2 - glucokinase system) -inh: ketogenesis, gluconeogenesis, glycogenolysis -stimulate: glycolysis **Muscles** -stimulate: (glc, AA & K uptake), glycogenesis, ribosomes Pr synth -inh: proteolysis **Adipose tissue** -stimulate: (glc & K uptake) & lipogenesis -inh: lipolysis

Glucagon		
causes	Hyperglycemia	
DNA	coded in chromosome 2	
synth steps	Preproglucagon → proglucagon → glucagon	
regulation	**Secretors** -hypoglycemia -inc blood AA -symp -stress -exercise	**Inhibitors** -hyperglycemia -somatostatin -insulin
TC	Liver	
Effects	-Glycogenolysis -Gluconeogenesis -Lipid oxidation (to produce KB)	

Adipose tissue	
FFA synth stimulators	-E (inc sensitivity to other hormones) -GH -cortisol -T3

DM			
Affects	-all body cells -Carbs, lipid, & Pr metabolisms		
Symptoms	Polyuria (excessive urination) Polydypsia (excessive thirst) Polyphagia (excessive hunger). retinopathy (vision loss) -due to smallen vessels- neuropathy (causes amputation) -due to smallen vessels- nephropathy (chronic renal failure) -due to smallen vessels-		
GTT	-Glucose Tolerance Test: is a test that can be used to help diagnose diabetes or pre-diabetes. -patient must fast 8-12h prior to the test -done by giving pt highly glc conc solution then testing his plasma 2h afterwards (normally after 2h glc level should be normal) -FPG <100 mg/dl -normal postprandial glucose < 140 mg/dL -prediabetic postprandial glucose 140 - 199 mg/dL -diabetic postprandial glucose ≥200 mg/dL -urine glc measurement indicates renal threshold for glucose		
Types	1, 2 & 3		
DM 1			
	DM1	**DM2**	**DM3**
Epedim	Children	adults	-
Etiology	Insulin def (Ai) (insulin dependent)	insulin resistance (insulin independnt)	gestational diabetes (pregnancy)
Lab	-high blood glucagon resulting in further hyperglycemia & KB (ketoacidosis)	can develop into Type 1 diabetes if uncontrolled.	-Resembles DM2 -if she didn't control it while she is preg. Leads to: **macrosomia** (high birth weight)
Treatment	-insulin supp (we dream of treating it via oral meds or Beta cells transp)	diet & life style change	improve following delivery.