glc	
Regulators	-the liver uses panc in response to brain (GIT-ECS-CNS)
Sources	**external** -diet -carbs are digested into monosacc. -frct & galactose are digested into glc -starch provides direct glc **Internal** -gluconeogenesis -building blocks: glycerol, lactate, pyruvates & glucogenic AA
Hyperglycemia	In its severe cases, glc binds to Pr (Pr-glycation)

Glc homeostasis stages					
Stages	**1**	**2**	**3**	**4**	**5**
Starter	Meal intake	Early fasting (when intook diet glc is exhausted)	Late fasting (when made glycogen is exhausted)	Fasting for days	Prolon-ged fasting
Tissues using glc	All the body (70% - liver)	Liver, muscles & fat use glc in low rates		CNS, RBC, renal medulla & little for muscles	Little CNS, RBC, renal medulla
Liver fun	glycogenesis (70% of all intook glc)	Glycogenolysis to provide glc	Glucogenesis out of non-glycogen sources	Glucogenesis out of non-glycogen sources **-fat burn (liver)**	**-fat burn (liver)** & little Gluco-genesis
Renal fun	-	-	-		
Liver fuel source	Diet **glc**	Synthesized **glc** (out of glycogen)	Synthesized **glc** (out of non-glygoen subs)	-	-
CNS fuel source				-Synthesized **glc** (out of non-glygoen subs) -**KB**	-**KB** -little **glc**

stages	Features
Note	-they overlap -intake of a meal restarts to stage 1
Stage 1	-glycogenesis -excessive glc intake is converted into FFA & triglyceride in liver, then transported via VLDL into adipose tissue -glucogenesis is inh (inh of: cori & alanine cycles)
Stage 2	-glycogenolysis -glycogen can be exhausted within a day, if exercised
Stage 3	-glucogenesis (out of: glycerol, lactate, pyruvates & glucogenic AA) -depends on: feeding status (how much did he eat before) hepatic stored glycogen physical activity
Stage 4	-dec glucogenesis & FFA oxidation (causes KB inc) -KB can be used as energy source
Stage 5	-almost no glucogenesis & FFA oxidation (causes KB inc) -main source is now KB -hyperKBemia inh muscles proteolytic (conserve muscles)
End-fate	-when body finally exhausts of the last particle of KB, it starts muscles proteolysis

Hormones regulating glc	
Dec blood glc	Insulin
Inc blood glc	Glucagon, somatostatin, cortiso, GH & E (insulin AntaGonizors)
Insulin MOA	-insulin receptor is of 2 parts: (a- EC) & (b- IC) -binding of insulin to (a) phosphorylates (b), thus activates the receptor -active receptor causes IC response to activate the hexose transporters (GLUT4) -active GLUT4 intakes blood glc **-CNS & liver have insulin independent glc transporters** (they don't need insulin to trigger glc influx)
Hyper-insulinemia	-caused by insulin resistance: DM & metabolic syndrome

Hyperglycemic agents					
	Glucagon	**Somatostatin**	**Cortisol**	**GH**	**E**
Strctr	Peptides	Peptides	Steroids	Normal Pr	CAT
By	Panc a-cells	Panc D-cells Stomach & SI	AG	A. lobe of PG	AG
Fun	glycogenolysis & liver glucogenesis	Inh glucagon & insulin secretion (indirect hyperglyce.)	-imp in fasting -lipolysis -liver glucogenesis (using AA)	-Inh insulin secretion -liver glucogenesis	-lipolysis -muscles glycol genolysis