DKA		
Epedim	Mostly with DM1	
KB	Synth in	Liver - mitochondria (ketogenesis)
	Used by	peripheral tissues (Ketolysis)
	BBB	can penetrate BBB, it's the 2ndry energy source for CNS (glc can penetrate it too & it's the primary)
DM	-only in uncontrolled DM, peripheral fat will undergo lipolysis & FFA will be mobilized to the liver to make KB there -ketogenesis > ketolysis (hyperKBemia & KBuria)	
ketogenesis	-hepatocytes oxidize FFA to acetyl CoA, which is used to make KB, using HMG CoA synthase **(VVIP: acetyl CoA is primarly used for ketogenesis)**	
KB possible forms	KB is made initially as acetoacetate can be reduced to β-Hydroxybutyrate or decarboxylated to acetone -β-Hydroxybutyrate can be oxidized back to acetoacetate (by dehydrogenase) -Acetoacetate can be converted to acetoacetyl CoA (by thiophorase) - Acetoacetyl CoA can be converted to a normal acetyl CoA	
Acetyl CoA	-can also bind to oxaloacetate in Krebs cycle, to provide energy -↑ Acetyl CoA production activates pyruvate carboxylase	
Pyruvate carboxylase	converts pyruvic acid into oxaloacetate	
oxaloacetate	Used mainly for glucogenesis rather than krebs cycle	
Ketolysis	-requires thiophorase -in extrahepatic tissues only, cuz the liver doesn't have the enz (in mitochondria only, so doesn't happen in RBCs - they don't have mitochondria) "RBC & liver don't use ketolysis"	
Symptoms	-acetone breath smell -Acidosis (due to the acidic KB in the blood) -Dehydration (due to glucosuria)	
Worseners	-Severe illness (MI) -Trauma -meds (steroids) -inappropriate insulin treatment -inf	

Hyperosmolar hyperglycaemic state	
AKA	Hypperosmolar non-ketosis
Epedim	Elders DM2 - much more lethal than DKA
KBemia	Little KB in the blood
Glcemia	Severally high!
Plasma osmolarity	Severally high!
Pathogen.	Blood insulin is insuff to allow body to use blood glc, But suff enough to prevent lipolysis & ketogenesis
Symptoms	CNS manif.

Hypoglycemia	
Epedim	Mostly with DM1
State	emergency case
Symptoms	-CNS disturbance (coma, confulsions, confusion…) -might evolve into complete cerebral dysfunction or necrosis
Symptoms presentation	**if blood glc <60** -sudden fall -sympathetic-hyperstimulation like symptoms -anxiety, tremors, sweating & palpitation **if blood glc <40** -gradual fall -neuroglycopenia symptoms (neuro-glyco-penia: CNS - glc - less) -headache, confusion, loss of consciousness & <u>seizures (if glc<30)</u>
Treatment	-glc intake (symptoms disappear within minutes
DM	<u>Glucagon & E</u> would no longer resolve hypoglycemia in prolonged DM
Body correction of hypoglc.	-dec: insulin -inc: E, glucagon, GH & Cortisol