| DM | |
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| Type 1 | -pt DNA must have an affinity for Ai first<br>-we think that the trigger for Ai is viral inf or toxins<br>-β cells destruction could take years to finish<br>-symptoms appear once enough β cells have been destroyed |
| Type 2 | -due to over production of insulin, like any other receptor, the body develops a resistance against the stimulus<br>-resistance development could take years to progress as well<br>-insulin resistance is a physic response in aging people, or it could be caused by a DNA mutation, or even the potato life!<br>-once the body have developed very strong resistance against β cells, they stop secreting insulin (hyperinsilinemia assists too)<br>-relativly less eventual KB compared to type 1 |
| Hb A1C | -it's the result of non-enzymatic binding of glc to Hb (due to hyperglycemia)<br>-used to diagnose DM in labs (mostly DM2) (>6.5 %)<br>-can be used to predetect retinopathies and prevent them<br>-very helpful to spot uncontrolled DM (for 2 months)<br>-using Hb A1C & FPG: will spot on best diagnosis of DM |
| Physio | **once the body reaches the point of aninsulinemia:**<br>**hepatic glucogenesis**<br>-by glycogenolysis<br>-it's a natural response: the dec in insulin always triggers glucagon release. Glucagon action is causing hyperglycemia by glycogenolysis & glucogenesis<br>**minute glc uptake by tissues**<br>especially muscles & fat<br>**Lipolysis**<br>KB genesis<br>**muscles Pr catabolism**<br>-insulin triggers glc & AA intake, so when its def muscles catabolize their own Pr to have more AA.<br>Result: excessive non-uptook AA in blood & excessive AA in muscles |

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| Compli. | **microvascular**<br>(retnopathy & nephropathy) “blindness & renal failure”<br>-its highly related to the duration of the disease<br>After 20y (DM1 = 100% / DM2 = 50 – 80%)<br>-microalbuminuria (little albumin in urine <300) is an indicator of nephropathy (if urine albumin >300 → proteinuria) |
| | -Advanced glycation end AGE of Pr (Pr bind to glc over time), which results in Pr not being able to enter cells, thus cells dysfun<br>-inc IC sorbitol over time, which pulls water in (osmolarity) cells then swell & lessen their fun & compress microvessels<br>-lipolysis uses ROS →oxidative stress → cells & vessels damage<br>**macrovascular**<br>(cardiac thrombus)<br>**Neuropathy**<br>-Loss of both myelinated and unmyelinated nerves<br>-its highly related to the duration of the disease & the control of it |
| AGE | -is non-enzymatic slow binding of Pr to glc (only in hyperglycemia)<br>-the Pr that is now bound to glc is called AGE<br>-AGE can interfere with vascular collagen, thus microvascular pathies<br>-AGE have a receptor called “RAGE” that when triggered produces ROS, thus infl & damage |
| Sorbitol | -glc is IClly metabolized to Sorbitol by aldose reductase<br>(Polyol pathway)<br>-we think that sorbitol production uses NADPH, thus ROS production<br>-we think that it affects PKC, thus affect Vascular endothelial growth factor, resulting in changing vascular permeability<br>-we know that sorbitol drags ECF in (osmolarity) |