Insulin secretagogues

Sulfonylureas & meglitinides	DPP-4 inhibitors
MOA: block ATP-de-pendent K+ channels	Sitagliptin "januvia"
SU	MOA: inhibit DPP4 enzyme (cytoplasmic recept-or)--> no incretin (GLP1) degredation--> Increase in GLP1 stimulates insulin secretion.

1 gen: Tolbutamide-chlorpropamide.
long acting SU so long hypoglycemic episodes.
Only eliminated renallly; risk to renally compromised patients.

2 gen: Gliclazide-glyburide "glibenclamide"
Shorter acting SU with pendant lipohilic gp larger or aromatic.
Undergo enterhepatic circulation and eliminated in urine & bile.

3 gen: Glimepiride
Completely metabolized by oxidation of pendant methyl substituent into methoxy metabolites (mostly in feces) & COOH metabolites (mostly in urine).

Meglitinides/glitinides

D-phenylalanine-Repaglinide-Nateglinide

Insulin sensitizers

Biguanides	Thiazolidinediones (TZD)
Metformin	Rosiglitazone-Pioglitazone
MOA: reduces liver glucose release and increases glucose uptake into tissue (decreases BG level).	MOA: activate the nuclear receptors (PPAR-y) which causes transcription of genes stimulating lipid uptake & adipogenesis.

Best described as anti-hyperglycemic agent because it doesn't cause hypoglycemia.

1st line for T2D with normal kidney function (**Ineffective without insulin**).

Alpha-glucosidase inhibitors

Voglibose

MOA: Delay digestion and carb absorption.

SE: flatulence, bloating, abdominal cramping

Adjuvant therapy.