

GASTROINTESTINAL TRACT

Core Defect

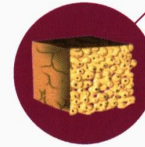
Decreased incretin effect



ADIPOSE TISSUE

Core Defect

Increased lipolysis



PANCREATIC ALPHA CELL

Core Defect

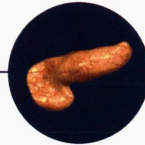
Increased glucagon secretion



PANCREATIC BETA CELL

Core Defect

Decreased insulin secretion



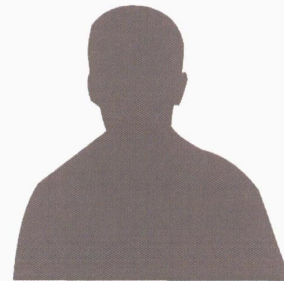
KIDNEY

Core Defect

Increased glucose reabsorption



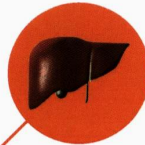
CORE DEFECTS IN T2 DIABETES¹



LIVER

Core Defect

Increased hepatic glucose production



MUSCLE

Core Defect

Decreased glucose uptake



























































BRAIN

Core Defect

Neurotransmitter dysfunction



TREATMENT OPTIONS FOR HYPERGLYCEMIA

CLASS ^{2*}	PRIMARY PHYSIOLOGICAL ACTION(S) ²	CORE DEFECTS ¹							
		KIDNEY	PANCREATIC ALPHA CELL	LIVER	MUSCLE	PANCREATIC BETA CELL	G.I. TRACT	ADIPOSE TISSUE	BRAIN
Biguanides	<ul style="list-style-type: none"> • ↓ Hepatic glucose production 								
Sulfonylureas	<ul style="list-style-type: none"> • ↑ Insulin secretion 								
Thiazolidinediones (TZDs)	<ul style="list-style-type: none"> • ↑ Insulin sensitivity 								
DPP-4 Inhibitors (Dipeptidyl Peptidase-4 Inhibitors)	<ul style="list-style-type: none"> • ↑ Insulin secretion (glucose-dependent) • ↓ Glucagon secretion (glucose-dependent) 								
SGLT2 Inhibitors (Sodium-Glucose Cotransporter-2 Inhibitors)	<ul style="list-style-type: none"> • Blocks glucose reabsorption by the kidney, increasing glucosuria 								
GLP-1 Receptor Agonists (Glucagon-Like Peptide-1 Receptor Agonists)	<ul style="list-style-type: none"> • ↑ Insulin secretion (glucose-dependent) • ↓ Glucagon secretion (glucose-dependent) • Slows gastric emptying • ↑ Satiety 								
Insulins	<ul style="list-style-type: none"> • ↑ Glucose disposal • ↓ Hepatic glucose production • Other 								

1. DeFronzo. RA. From the triumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. *Diabetes*. 2009;58:773-795.

2. Inzucchi SE, Bergenstal RM, Buse -JB, et al. Management of hyperglycemia in type 2 diabetes, 2015: a patient-centered approach. *Diabetes Care*. 2015;38:140-149.

* This tool is intended to provide an overview of T2DM drugs and is not specific to only one product within the class. It is not intended to make any express or implied comparison among products. Classes shown are from the ADA Guidelines Chart and are not all T2DM classes available to treat hyperglycemia.

HISTORY & PROGRESSION OF TYPE 2 DIABETES

