



TREATMENT OPTIONS FOR HYPERGLYCEMIA

CLASS ^{2*}	PRIMARY PHYSIOLOGICAL ACTION(S) ²	CORE DEFECTS ¹							
Biguanides	• ↓ Hepatic glucose production	KIDNEY	PANCREATIC ALPHA CELL	LIVER	MUSCLE	PANCREATIC BETA CELL	G.I. TRACT	ADIPOSE TISSUE	BRAIN
				Or				190	
Sulfonylureas	• ↑ Insulin secretion	(1)		0	0		0	110	
Thiazolidinediones (TZDs)	• ↑ Insulin sensitivity			(A)					
DPP-4 Inhibitors (Dipeptidyl Peptidase-4 Inhibitors)	 ↑ Insulin secretion (glucose-dependent) ↓ Glucagon secretion (glucose-dependent) 						J		
SGLT2 Inhibitors (Sodium-Glucose Cotransporter-2 Inhibitors)	Blocks glucose reabsorption by the kidney, increasing glucosuria								
GLP-1 Receptor Agonists (Glucagon-Like Peptide-1 Receptor Agonists)	 ↑ Insulin secretion (glucose-dependent) ↓ Glucagon secretion (glucose-dependent) Slows gastric emptying ↑ Satiety 								
Insulins	 ↑ Glucose disposal ↓ Hepatic glucose production Other 			OV.					

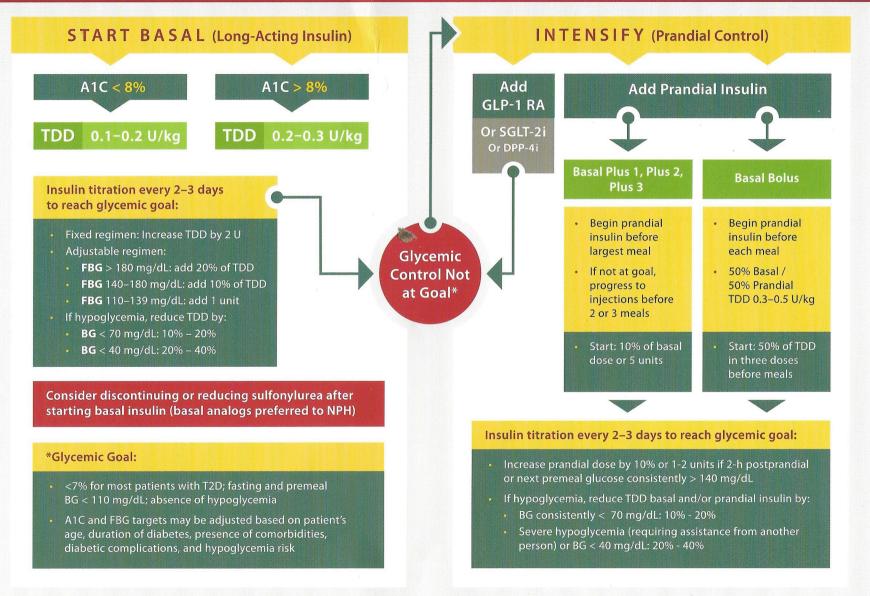
^{1.} DeFronzo, RA. From the triumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. Diabetes. 2009;58:773-795.



^{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.

ALGORITHM FOR ADDING/INTENSIFYING INSULIN



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GLYCEMIC CONTROL ALGORITHM

