Current	Channel	Gating mechanism	Functional role
i_{K1}	K^+ channel (inward rectifier), K_{ir}	Voltage	Maintains high K^+ permeability during phase 4 Its decay contributes to diastolic depolarization Its suppression during phases 0-2 contributes to plateau
i_{Na}	Na^+ channel (fast) Nav 1.5	Voltage	Accounts for phase 0 of action potential Inactivation may contribute to phase 1 of action potential
i_{To}	K^+ channel (transient outward), K_{to}	Voltage	Contributes to phase 1 of action potential
i_{Ca}	Ca^{2+} channel (slow inward, L channels) Cav 1.2	Both	Contributes to phase 2 of action potential Inactivation may contribute to phase 3 of action potential Is enhanced by sympathetic stimulation and β-adrenergic agents
i_K	K^+ channels (delayed rectifier), K_r, K_s, K_{ur}	Voltage	Causes phase 3 of action potential Is enhanced by increased intracellular Ca^{2+}
i_{KATP}	K^+ channel (ATP-sensitive)	Ligand	Increases K^+ permeability when [ATP] is low
i_{KACh}	K^+ channel (acetylcholine-activated)	Ligand	Responsible for effects of vagal stimulation Decreases diastolic depolarization (and the heart rate) Hyperpolarizes resting membrane potential Shortens phase 2 of the action potential
i_f ("funny")	Na^+, Ca^{++}, K^+ (pacemaker current via HCN channel)	Both	Is activated by hyperpolarization and cyclic nucleotides and contributes to the diastolic depolarization Is enhanced by sympathetic stimulation and β-adrenergic agents Is suppressed by vagal stimulation