The cardiac potassium (K₊) channels summary

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Cardiac K⁺ channels are membrane-spanning proteins that allow the passive movement of K⁺ ions across the cell membrane along its electrochemical gradient. They regulate the resting membrane potential, the frequency of pacemaker cells and the shape and duration of the cardiac action potential. Normal K⁺ channel function is essential to maintain electrical stability in the heart. Gene mutations that alter the assembly, trafficking, turnover or gating of cardiac K⁺ channels can cause LQTS, SQTS, J-wave syndromes and AF.

1. Delayed Rectifier Potassium (K⁺) Currents/Channels

- a) The rapidly activating component of the delayed rectifier K⁺ current, *I*_{Kr}, rapid-rates of activation onset. Name: Kv11.1 (HERG), Gene:KCNH2, Human Chromosomal location:7q35–36
- b) The slowly activating component of the delayed rectifier K⁺ current, I_{Ks} , slow-rates of activation onset: Name: Kv7.1 (KVLQT1), Gene: KCNQ1. Human Chromosomal location 11p15.5
- The ultrarapid (I_{Kur}) ultra-rapid rates of activation onset. Name: KCNA5, Gene:12p13.3, Human Chromosomal location: 12p13.3

2. Inward rectifying K⁺ channels

- *I*_{K1}, "The transient outward current". Name: Kir2.1 (IRK1), Gene: KCNJ2, Human Chromosomal location: 17q23.1–24.2
- *I*_{KATP} ATP-sensitive K⁺ channels, K_{ATP} Name: Kir6.2 (BIR), Gene:KCNJ11, Human Chromosomal location:11p15.1

• I_{KAch} The acetylcholine-activated K⁺ current, I_{KAch} Name: Kir3.1 (GIRK1), Gene: KCNJ3, Human Chromosomal location: 2q24.1 11p15.1

3. Transient outward currents

- I_{tof} I_{to1} 4-aminopyridine (4-AP)-sensitive, calcium-independent K⁺ current (I_{to1}) is rapidly activated and inactivated in response to depolarization
- I_{to2} , I_{to2} 4-AP-insensitive, Ca²⁺-activated Cl⁻ or K⁺ current (I_{to2})

4. Intracellular cation activated currents

- ➤ IKNa,
- > IKCa) and at least one
- "background leak" current (IKleak)