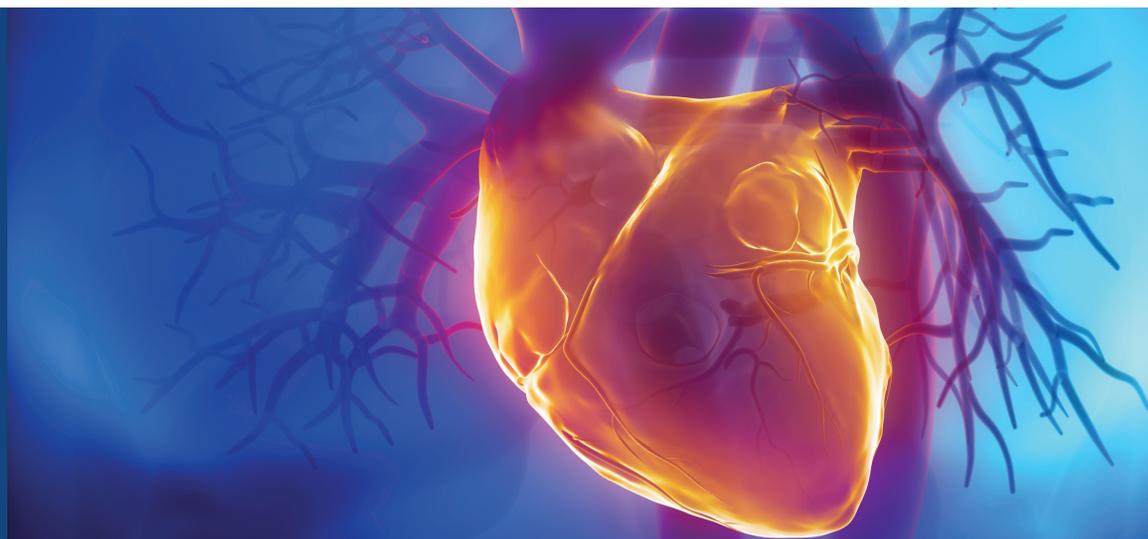


Summary

The Charles River ion channel portfolio includes over 120 targets which have been organized into Channel Panels[®] based on current scientific findings, proving a useful tool in guiding early screening and selectivity profiling.



DISCOVERY

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Ion Channel Families:

- Calcium, voltage-gated (Cav1.2/ $\beta_2/\alpha_2\delta_1$ and Cav3.2)
- Hyperpolarization-gated (HCN2 and HCN4)
- Potassium, inward rectifier (Kir2.1, Kir3.1/3.4 and Kir6.2/SUR2A)
- Potassium, voltage-gated (Kv1.5, hERG (Kv11.1), Kv4.3 and KvLQT1/mink (Kv7.1))
- Sodium, voltage-gated (Nav1.5)

Need a custom version of this assay?

Visit [criver.com/pi-ds-ion-channel-profiling-assay](https://www.criver.com/pi-ds-ion-channel-profiling-assay)

Ion Channel Selectivity Profiling: Cardiac

Our Cardiac Channel Panel[®] provides off-target profiling of the major ion channels that influence ventricular action potential duration and regulate heart rate.

Selectivity Profiling

Identification of a compound's target specificity and potential for off-target effects is a critical step in the drug discovery process and often includes assessments against specific target class families, critical safety targets or by therapeutic area. In addition to our [therapeutic area-specific Channel Panels[®]](#), we offer screening on a number of [electrophysiology platforms](#). When required, our scientists can design customized panels to meet a client's needs. As pioneers in the field of [ion channels](#), we are able to provide expert consultation to facilitate interpretation of results.

Cardiac Channel Panel[®]

The voltage-gated cardiac potassium channel, [hERG](#), is responsible for the rapid delayed rectifier current (IKr) that regulates repolarization of the action potential. Inhibition of IKr is the most common cause of delayed repolarization and QT prolongation by non-cardiac drugs, and is associated with a potentially fatal ventricular arrhythmia, Torsade de Pointes. However, the influence of hERG inhibition on the QT interval can be blunted by compensatory inhibition of [ion channels](#) that carry depolarizing currents (e.g., Nav1.5 and Cav1.2) that regulate the plateau phase of the action potential. Moreover, delayed repolarization via inhibition of other sources of potassium current (e.g., Kv4.3, KvLQT1/minK and Kir2.1) may augment or supplant hERG inhibition as a cause of [QT prolongation](#). Drug-induced increase in Nav1.5 current can also delay repolarization and prolong QT. Our Cardiac Channel Panel[®] provides off-target profiling of the major channels that influence ventricular action potential duration. Additionally, the panel provides profiling of channels that regulate heart rate (e.g., HCN2, HCN4, Kir3.1/Kir3.4 and Cav3.2), atrial repolarization (Kv1.5), and response to metabolic stress (Kir6.2/SUR2A).

EVERY STEP OF THE WAY