Cardiac safety is one of the leading causes of late stage compound attrition in the pharmaceutical industry and accounts for 7-10% of all drug failures.  Early biomarkers of cardiac safety are widely focused on repurposing existing cardiac endpoints. However, different types of cardiotoxic events are present in the heart and participate in the overall cardiac function. These assays should provide a comprehensive evaluation of a compound's potential for both structural and functional cardiotoxicity.

Cardiotoxicity Potential can be Identified by Changes in Metabolism

Metabolite ratios detect cardiotoxicity potential independent of changes in cell viability. Developed multiple models using combinations of HRMS methods, including C8 and HRMS methods, including C8 and LC-MS. The models analyzed were validated using a blinded test set of 24 compounds (12 positive, 12 negative).

Cardiac events that occur in the heart can be identified by changes in metabolism. The overall cardiac function is maintained by the electrical activity of cardiomyocytes, which is generated and maintained by the coordinated activity of ion channels. Over 70 different types of ion channels are expressed in cardiomyocytes, and these ion channels participate in the overall cardiac function. Many of these ion channels are implicated in the etiology of arrhythmias.

Cardiac safety is a critical factor in the development of new drugs. The heart is responsible for pumping blood throughout the body, and its function is essential for maintaining life. Cardiac safety is a major concern in drug development because cardiac toxicity can lead to serious adverse events, including arrhythmias, heart failure, and even death. As a result, cardiac safety is a critical factor in the development of new drugs, and various assays are used to assess cardiac safety.

In this study, we developed a biomarker-based assay for evaluating the cardiotoxicity potential of compounds based on changes in metabolism observed through the metabolome of induced pluripotent stem cell (iPSC)-derived cardiomyocytes.

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