Mitochondrial Dysfunction & Contraction

The impact of Mitochondrial Impairment on CoriX® IPS Cardiomyocyte Beating

Antagonists and treatment behavior causes an immediate inhibition of mitochondrial function while TET treatment causes an immediate uncoupling of (Fig. 4, right panel) (Fig. 4, right panel). TET-induced cardiomyocyte beating is maintained (Fig. 4). Analysis of O2 consumption (using MitochondriaTest® and IBA (using pHiTest®) suggests that ATP generation is associated with mitochondrial oxygen consumption. Analysis of O2 consumption in the presence of the DPI I inhibitor rotenone also demonstrates that, under stressed conditions, TET (TET) treatment increases O2 activity is driven by phosphorylation and regulation (Fig. 10).

Impact of Coupling of Contractility and Metabolism

Impact of altered Beat Rate on Metabolism

Treatment with the β-adrenergic antagonist isoproterenol can lead to a significant increase in beating rate (Fig. 4). An O2 consumption increase can be observed by MitoXpress while ATP supplementation and partial pressure causes the impact of treatment on metabolism (Fig. 4).

Rising O2 consumption is measured in beating cardiomyocytes (Fig. 4). The O2 consumption increase is achieved by isoproterenol (Fig. 4).

Impact of Mitochondrial Substrate and Increased Beat Rate on Cellular Oxygenation

In vivo, the most important regulatory factors for ATP production are fatty acid and carbohydrates. However, cardiomyocyte metabolism is particularly adaptable to various substrates such as amino acids, ketone bodies and lactate. Isoproterenol is one of the most important ligands for the activation of metabolic processes in hypoxic conditions (Hynes et al., 2013).

The MitoXpress® assay highlights the importance of substrate preference and sequencing when designing and interpreting in vitro cellular analysis.

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Measurement Principles

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