IN THIS ISSUE

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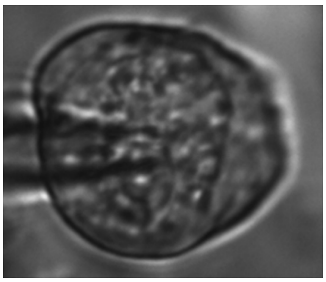
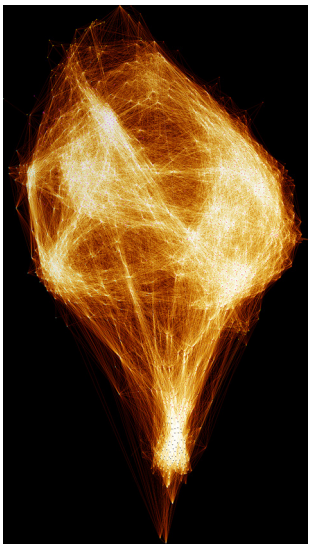


Open Software to Quantify Cardiac Contraction (p e5)

Sala et al create MUSCLEMOTION, an open-source software for quantifying contractions in both heart muscle cells and tissues.

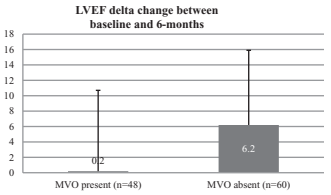
Measurement and analysis of cardiac contractility are of high importance in studying muscle function, assessing disease phenotypes, and evaluating drug toxicity. Unlike electrophysiological measurements, for which there are well-established and standardized techniques applicable to a range of samples, the techniques for measuring myocardial contractions tend to be sample-specific, not easily comparable, and require specialized expertise. Sala and colleagues have now developed a novel software that can quantify contractions in movies of specimens as diverse as single heart cells, cultured cardiac myocytes, 3D cardiac organoids, and even living Zebrafish hearts. They tested the ability of their software, called MUSCLEMOTION, to detect drug-induced and disease-related contraction differences in a variety of samples and showed that the performance of the software was comparable to various gold-standard techniques tailored to those specific samples. By making MUSCLEMOTION user-friendly and freely accessible, the authors hope to facilitate comparisons of contraction data, not just between samples, but between laboratories as well, thereby facilitating data replication and reproducibility.



64 Novel Genetic Loci for Coronary Artery Disease (p 433)

Van der Harst and Verweij provide evidence for an omnigenic model of coronary artery disease.

Coronary artery disease (CAD) is a multifactorial condition with contributions from both genetic and environmental factors. It has been associated with a number of genetic loci, yet linking these to potential pathological pathways remains difficult. Van der Harst and Verweij have now carried out the largest GWAS (genome-wide association study) to date. Examining nearly 8 million single nucleotide polymorphisms (SNPs) in a total of 122733 CAD patient samples, they identified 64 novel CAD-associated loci. These candidate genes, however, are involved in such broad biological functions and expressed in such diverse tissues that the contribution of genetic factors appears to be even more complicated than first thought, as many loci have no obvious link to CAD. Based on these findings, the authors suggest that the model of CAD as a polygenic disease—one caused by multiple genes affecting a handful of key pathways—may be incorrect. Instead, they surmise that the results point to an omnigenic model—whereby the link between many SNPs and CAD may be one of interconnectedness of gene-regulatory mechanisms. If so, determining cell-specific networks of gene expression may be the best route to understanding CAD pathology.



Long-Term MRI Follow-Up of MVO After Cell Therapy (p 479)

Traverse et al report the final 2-year results of the stem cell TIME trial.

Stem cell therapies are under investigation for their potential to promote cardiac tissue repair after myocardial infarction (MI). Because changes to the myocardium following reperfusion could influence cell engraftment and survival, the timing of cell delivery was examined in the TIME trial (Timing In Myocardial Infarction Evaluation), which compared delivery of cells on day 3 with day 7 following MI in 120 patients. At 6 months, the results indicated that cells delivered at either time point offered no benefits to patients. And 2 years later, these results remain the same. However, the study has also provided one of the largest MRI datasets of patients following MI. By analyzing these data, Traverse and colleagues show that patients who had microvascular obstructions at baseline have poorer outcomes—larger infarct size, more adverse left ventricle remodeling, reduced recovery of ventricle function, and greater likelihood of requiring an implantation device. From these findings, the team suggests that the presence of microvascular obstruction may be a powerful predictor of subsequent left ventricle failure, and that microvascular dysfunction identifies the most at-risk patients.

Written by Ruth Williams

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