IN THIS ISSUE

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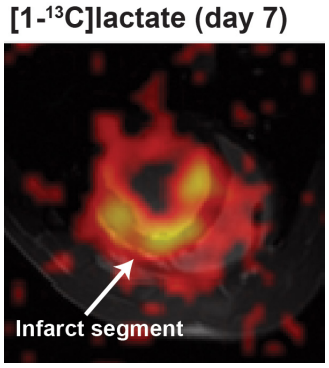
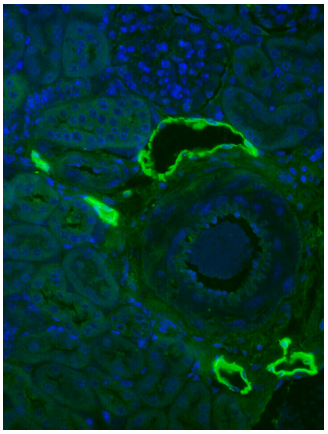


Hyperpolarized MRI of Cardiac Inflammation (p 1084)

Hyperpolarized MRI offers a noninvasive view of heart inflammation after myocardial infarction, say Lewis et al.

The inflammatory response to myocardial infarction (MI) influences functional recovery and patient outcome. To gain a better understanding of such post-MI inflammation, Lewis and colleagues employed hyperpolarized magnetic resonance imaging—a newly developed imaging modality. In this form of MRI, hyperpolarized carbon-13 (^{13}C)–containing substrates, such as pyruvate, are used as a contrast agent to observe metabolic activity—for example, conversion of the pyruvate into lactate. Because inflammation is associated with a metabolic shift to glycolysis, and thus with lactate production, the team reasoned that post-MI inflammation would be readily detectable by hyperpolarized MRI. Indeed, the team found that in rats, a more intense ^{13}C-lactate signal was observed in the hearts after MI than before. In contrast, in animals with depleted immune cells, or given an anti-inflammatory drug, the ^{13}C-latacte signals of post-MI and noninfarcted hearts were similar. Strong post-MI ^{13}C-lactate signals were also observed in the much larger hearts of pigs, suggesting this noninvasive approach might be applicable to humans.



Renal Lymphatics and Hypertension (p 1094)

Boosting renal lymphangiogenesis prevents hypertension, report Gelston et al.

Hypertension affects almost half of the adult population of the United States and is a robust risk factor for coronary artery disease, stroke, and other cardiovascular disorders. Kidney inflammation is thought to contribute to the development of hypertension. Indeed, in animal models, immunosuppression has been found to prevent the disorder. However, treating millions of hypertensive patients with immune-suppressing drugs would leave them vulnerable to infections. It has been observed that certain hypertensive rats exhibit increased growth of renal lymph vessels, which in theory should promote the resolution of inflammation by facilitating exit of the immune cells. But whether modulating lymphangiogensis could affect hypertension was unknown. Now Gelston and colleagues have shown that hypertension in mice is also associated with increased lymphangiogenesis and that renal-specific overexpression of a lymphangiogenesis promoting factor—VEGF-D—could reduce immune cell accumulation in the kidneys and, more importantly, prevent hypertension. These results suggest that the renal lymphatic system is a key player in regulating hypertension and could be a novel target for future therapies.

NAC and Cardiac Hypertrophy and Fibrosis in HCM (p 1109)

Antioxidant treatment does not improve symptoms of hypertrophic cardiomyopathy, report Marian et al.

Hypertrophic cardiomyopathy (HCM), which can lead to arrhythmia, heart failure, and sudden cardiac death, is generally caused by mutations in sarcomere proteins. At the tissue level, the condition is characterized by left ventricle hypertrophy, fibrosis, and oxidative stress. Currently, there is no clinical means to reverse tissue pathology associated with HCM, but in animal models, the myocardium has been shown to improve after treatment with the antioxidant N-acetylcysteine (NAC). Marian and colleagues now report the findings of the HALT-HCM trial (Hypertrophy Regression With N-AcetyLcysTeine in HCM)—a double-blind, randomized, placebo-controlled study of 42 HCM patients. In this study, 29 patients received NAC as a high-dose capsule taken orally twice a day, while 13 patients received placebo capsules. After 12 months of treatment, physical fitness, electrocardiography, and heart structure (by MRI) of the patients were compared. However, there were no significant differences in these measures between the 2 groups. While the trial had only a small number of participants, which may have obscured a mild effect, the results suggest that, contrary to the preclinical animal models, NAC may not be an effective treatment for humans with HCM.

Written by Ruth Williams
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