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CARDIOVASCULAR BIOLOGY

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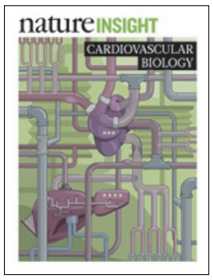

Cover illustration by Nik Spencer

The Macmillan Building
4 Crinan Street
London N1 9XW, UK
Tel: +44 (0) 20 7833 4000
e: nature@nature.com

nature publishing group

A healthy vasculature is crucial to our survival: blood vessels act as conduits to deliver oxygen and nutrients to every tissue in our body, remove waste and allow immune surveillance. Vascular endothelial cells also contribute to processes such as haematopoiesis and organ development during embryogenesis. Not surprisingly, vascular dysfunction is linked to diverse disorders, from cancer to eye diseases, and can also trigger heart failure and death. Understanding how vessels grow and function has huge potential for improving human health, and this exciting topic is developing at a rapid pace.

Anti-angiogenic agents that block vascular endothelial growth factor (VEGF) are being used in the clinic to treat patients with cancer and eye diseases, but efficacy has been limited. Peter Carmeliet and Rakesh Jain review the frontline research into the mechanisms regulating normal and pathophysiological angiogenic growth: they discuss the clinical experience with VEGF blockers and describe recently identified signalling pathways that contribute to vessel growth, maturation and quiescence, which may provide new avenues to improve anti-angiogenic therapy.

Mark Lindsay and Harry Dietz explore the pathogenesis of aortic aneurysm; an enlargement and weakening of the arteries, which can lead to fatal tearing. They show how research has implicated the cytokine transforming growth factor-β in the pathogenesis of this disorder, and how this has led to new therapeutic opportunities with losartan, an angiotensin II receptor antagonist.

Atherosclerosis is a disease of the arterial wall, in which lipid-filled plaques develop in the inner lining of the artery. Preclinical research has led to many hypotheses about the pathogenesis of plaque formation. Peter Libby, Paul Ridker and Göran Hansson discuss the limitations of animal models of atherosclerosis and the difficulties with extrapolating these findings to human disease, and suggest better ways to consolidate preclinical and clinical research.

A potentially fatal consequence of ruptured atherosclerotic plaques is myocardial infarction, which causes a massive loss of cardiomyocytes, leading to heart dysfunction or complete heart failure. In the last review of this series, Michael Laflamme and Charles Murry discuss the emerging prospects for regenerating the damaged myocardium, a dynamic area of research that draws from advances in stem-cell biology, developmental biology and tissue engineering.

Clare Thomas
Senior Editor

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REVIEWS