that improved understanding of HF in the community could lead to increased funding of both HF healthcare and of research into this major public health problem.

Carol Lovegrove

Original article Remme WJ *et al.* (2005) Public awareness of heart failure in Europe: first results from SHAPE. *Eur Heart J* [doi: 10.1093/eurheartj/ehi447]

Dual growth-factor treatment can regenerate infarcted myocardial tissue

Growth factors can activate cardiac stem cells in mice with infarcted hearts, leading to regeneration of the myocardium, US researchers have shown.

The human heart has been found to harbor cardiac stem cells and early committed cells (CSCs–ECCs). Researchers have, therefore, been trying to promote the survival and proliferation of these cells. Urbanek *et al*. investigated whether a combination of insulin-like growth factor 1 (IGF1) and hepatocyte growth factor (HGF) could stimulate migration and growth of cardiac stem cells.

The researchers identified replicating CSCs–ECCs in the atrioventricular groove of the mouse heart and labeled these cells using enhanced green fluorescence protein. Mice with infarcted myocardial tissue were then treated with IGF1 and HGF and the degree of death, survival and growth of CSCs–ECCs were evaluated by confocal microscopy at regular time points over 4 months.

Sixteen days after growth-factor treatment, new myocardium-containing arterioles, capillaries and contractile myocytes had developed at the site of infarction. Although the area of ischemic heart tissue was generally larger in growth-factor-treated mice than in controls, the former had a higher chance of survival as the newly formed myocardium led to improved ventricular function.

These findings show that CSCs–ECCs can respond to local administration of IGF1 and HGF by migrating to sites of ischemic injury and developing into functional myocardial tissue.

Claire Braybrook

Original article Urbanek K *et al.* (2005) Cardiac stem cells possess growth factor-receptor systems that after activation regenerate the infarcted myocardium, improving ventricular function and long-term survival. *Circ Res* **97:** 663–673

Anti-huHSP60 linked with adverse 1-year prognosis in patients with cardiac chest pain

Previous studies have shown that titers of antibodies to human heat shock protein 60 (anti-huHSP60) or mycobacterial heat shock protein 65 (anti-mHSP65) are linked with progression of carotid atherosclerosis and clinical outcomes. In what is claimed to be the first study to assess the prognostic implications of anti-huHSP60 titers in a population of patients with unstable angina, Birnie *et al.* have now investigated whether titers of anti-huHSP60 and anti-mHSP65 can predict adverse clinical outcome in patients with acute chest pain of suspected cardiac ischemic origin.

In this prospective study, venous blood samples were obtained from 588 participants, and their titers of anti-mHSP65 and anti-huHSP60 measured. Patients were followed up for a mean period of 304 days and the following endpoint outcomes were recorded: coronary heart disease death; nonfatal myocardial infarction; coronary artery bypass grafting; percutaneous transluminal coronary angioplasty; angiogram; and readmission with further cardiac chest pain.

The risk of an adverse 1-year prognosis increased by about 50% for patients with titers of anti-huHSP60 greater than 16 U/l compared with those with lower titers. This trend was still evident after adjustment for the established cardiovascular risk factors of age, hypertension, diabetes and smoking. Titers of anti-mHSP65, however, did not correlate significantly with 1-year clinical outcomes.

The authors conclude that elevated titers of anti-huHSP60 are associated with increased risk of an adverse 1-year prognosis in patients presenting with acute chest pain.

Claire Braybrook

Original article Birnie *et al.* (2005). Increased titres of anti-human heat shock protein 60 predict an adverse one year prognosis in patients with acute cardiac chest pain *Heart* **91:** 1148–1153

Early initiation of statins after ACS improves vascular endothelium function

Cholesterol lowering using statin regimens initiated soon after acute coronary syndromes (ACS) has been shown to improve endothelial