POSTER SESSION

1135 Experimental and Clinical Heart Transplantation
Monday, March 18, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 3:00 p.m.-4:00 p.m.

1135-141 Long-Term Beneficial Effects of Neonatal Cardiomyocytes on Left Ventricular Ejection Fraction, Regional Wall Motion, and Scar Thickness After Transplantation Into Infarcted Rat Hearts
Jochen Muller-Ennse, Kirk L. Peterson, Larry Kedes, Peter Whittaker, Joan S. Dow, Robert A. Kloner, Heart Institute Good Samaritan Hospital, Los Angeles, California.

The long-term effects of cardiac cell transplantation on myocardial dysfunction are unknown. We tested, therefore, the impact of neonatal cardiomyocytes on cardiac contractility, left ventricular (LV) volumes and LV geometry 6 months after transplantation into infarcted hearts. Cardiomyocytes from male neonatal Fisher 344 rats (12-d, 3-5 x 10^5-50 50 μl) or medium were injected into the infarcts of adult syngeneic female animals 1 week after left coronary artery ligation. LV angiography 6 months after cell transplantation revealed a greater ejection fraction in treated animals than control (0.36±0.03 vs. 0.25±0.02, both, n=9, P<0.01; 55±17% of the myocardiadic infarct zone was dyskinesic in control rats vs. 29±5±3% in cell treated rats (P<0.05). Average chordal shortening within the myocardial infarct zone was negative in controls but positive in treated rats (P<0.02). LV hemodynamics (n=9 each) showed similar enddiastolic pressures, maximal systolic pressures and maximal rates of rise and fall of ventricular pressure (+dP/dt, -dP/dt). Post mortem, hearts were fixed in distension (10 mmHg intracavity pressure), and LV geometry data were obtained. LV volumes and infarct sizes were similar in the treated group as in control (0.42±0.02 vs. 0.45±0.03 ml and 31.5±1.7% vs. 34.2±2.8%), but scar thickness was greater in treated animals (99±97 μm vs. 61±41 μm, P<0.02). Previously, we have shown that more than half of neonatal cardiomyocytes survive for at least 6 months after transplantation into infarcted hearts. Transplanted neonatal cells reduced regional dyskinesia and had a small beneficial effect on systolic contraction. For the first time, it has been shown that cell transplantation therapy improves long-term in vivo global and regional left ventricular function after myocardial infarction.

1135-142 Identification of Genes Involved in Acute Rejection Following Heart Transplantation in a Murine Model: Use of cDNA Arrays
Anja L. S. Roussoulieres, Oksana Raskin, Lara Chalabreysse, George Dureau, Pascale Boissonnet, Jean-Francois Obedia, Jean-Francois Obedia, Bijal Patel, Maria L. Espesj, Eric Sue, Sheyrlene Go, Jon A. Kobashigawa, University of California, Los Angeles, California.

Background: More than half of neonatal cardiomyocytes survive for at least 6 months after transplantation into infarcted hearts. Transplanted neonatal cells reduced regional dyskinesia and had a small beneficial effect on systolic contraction. For the first time, it has been shown that cell transplantation therapy improves long-term in vivo global and regional left ventricular function after myocardial infarction.

1135-143 Mesenchymal Stem Cell Therapy Prevents Deterioration of Left Ventricular Function in a Porcine Myocardial Infarction Model
Mohammed S. Gayan, Kaname Takizawa, Malika Frantzen, Robb Macdillian, Michael Lit, Michael C. Fishbein, Takashi Miyamoto, Raj Makkar. Cedars-Sinai Medical Center, Los Angeles, California.

Objectives: To test this hypothesis, we developed a porcine model of LV dysfunction. An apical myocardial infarction was induced by cell embolization of the distal LAD. Biplane ventriculography was used to measure ejection fraction (EF) pre and post-MI. At one month post-MI, animals underwent thoracotomy and epicardial injection into the apical scar (27 g needle, 3 ml final volume) of either 150 million MSC (cell group) or growth medium alone (placebo group). Prior to injection, MSC, MIP-1 and underexpression of VE-Cadherin in the acute rejection group (group A).

Conclusions: Our results suggest that the production of MIP-1 beta and the suppression of the VE-Cadherin production by the endothelial cells may participate in the early phase of an acute rejection. Indeed, these proteins may be implicated in the regulation of leukocyte recruitment and migration into the myocardial tissue. cDNA arrays offer a powerful tool to study transcription profile patterns in acute allograft rejection. Such transcription profiles may be of great help in identifying new diagnostic and/or therapeutic tools in acute rejection.