Increased Myocardial Infarction After Age Nihility: Outcomes in 16,213 non-Japanese

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Background: Most studies of acute myocardial infarction (MI) focus on patients aged up to 85. However, the aging of the population has resulted in increased numbers of non-Japanese MI. The purpose of this study was to examine the incidence of non-Japanese MI and its outcomes in older patients aged 85 or more in the United States.

Methods: Using the statewide myocardial infarction database, we identified all non-Japanese MI in the state of California. We then compared the incidence, mortality, and outcomes of non-Japanese MI aged 85 or more to those aged 65-84.

Results: The annual number of non-Japanese MI deaths increased from 80 women and 120 men in 1985 to 1986 to 1500 women and 2500 men in 2002. The incidence of non-Japanese MI was 7.6 per 100,000 for women and 12.2 per 100,000 for men.

Increased Myocardial Infarction Prevalence of C-Reactive Protein in Human Coronary Heart Disease: Relation to Endothelial Dysfunction and Microvascular Reactivity

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Background: Increased plasma C-reactive protein (CRP) is a hallmark of chronic heart disease (CHD) but cellular sources during disease progression and potential roles as a predictor of the disease process are not well defined. A known source of CRP in CHD is the endothelium. Also, CRP is an indicator of systemic inflammation and a predictor of acute coronary events. We have recently shown that CRP and endothelin-1 (ET) levels were increased in CHD patients and that CRP levels were increased in patients with CHD and those changes were related to local endothelial stress and/or microvascular reactivity.

Methods: Here, we hypothesize that ET-1 production is increased in patients with CHD, and that CRP levels are increased in patients with CHD, and that those changes are related to local endothelial stress and/or microvascular reactivity. Patients with CHD showed significant increases in ET-1 and CRP levels compared to those without CHD. Furthermore, patients with CHD showed significant increases in ET-1 and CRP levels compared to those without CHD.

Conclusions: These findings provide evidence for the production of CRP in CHD and for the potential of CRP as a marker of endothelial dysfunction and microvascular reactivity. These observations may also help explain the increased risk of increased CRP in CHD patients.

TNF-α Enhances the Engraftment of Mesenchymal Stem Cells into Infarcted Myocardium Using BM-PC

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Background: Mesenchymal stem cells (MSCs) have therapeutic potential after myocardial infarction. We hypothesized that TNF-α may enhance MSCs' engraftment in the heart by modulating the inflammatory and reparative microenvironment.

Methods: TNF-α was isolated from the infarcted myocardial niche and used to stimulate MSCs. The effects of TNF-α on MSC engraftment and differentiation were assessed using a co-culture model.

Results: TNF-α significantly increased MSC engraftment and differentiation in the infarcted myocardium. Furthermore, TNF-α induced the expression of pro-inflammatory cytokines, which may contribute to the enhanced MSC engraftment.

Conclusion: These findings suggest that TNF-α enhances MSC engraftment in the infarcted myocardium, which may have therapeutic implications for myocardial repair.

Increased Glucose Uptake by Antimycin-S3 Reduces Hypoxia-Induced Apoptosis in Cultured Neoplastic Rat Cardio endothelial Cells

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Background: Antimycin has been shown to inhibit glucose uptake in rodent cells. We hypothesized that antimycin-S3 would reduce glucose uptake in human cells and that this would reduce hypoxia-induced apoptosis.

Methods: Antimycin-S3 was applied to cultured human endothelial cells. The effects on glucose uptake and cell viability were assessed.

Results: Antimycin-S3 significantly reduced glucose uptake and decreased apoptosis.

Conclusion: Antimycin-S3 may be a potential therapeutic agent to reduce hypoxia-induced apoptosis in human cells.

Mitochondrial Haplogroup NB5 is Protective Against Myocardial Infarction in Japanese Males

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Background: Several studies have shown that mitochondrial haplogroups are associated with myocardial infarction in Japanese males.

Methods: Mitochondrial DNA was sequenced in 1000 Japanese males and the association with myocardial infarction was assessed.

Results: Haplogroup NB5 was significantly associated with myocardial infarction in Japanese males.

Conclusion: Haplogroup NB5 may be protective against myocardial infarction in Japanese males.