UNIVERSITA'
DI PAVIA

DOTTORATO DI RICERCA IN BIOINGEGNERIA
E BIOINFORMATICA
DIPARTIMENTO DI INFORMATICA E SISTEMISTICA

AVVISO DI SEMINARIO

Cardiomyocyte Proliferation: A Platform for Myocardial Repair?

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Abstract - The human heart does not regenerate. Instead, following injury, human hearts scar. The loss of contractile tissue contributes significantly to morbidity and mortality. In contrast to humans, zebrafish and newts faithfully regenerate their hearts. Interestingly, regeneration is in both cases based on cardiomyocyte proliferation. In addition, mammalian cardiomyocytes proliferate during fetal development. Their proliferation reaches its maximum around chamber formation, stops shortly after birth, and subsequent heart growth is mostly achieved by an increase in cardiomyocyte size (hypertrophy). The underlying mechanisms that regulate cell cycle arrest and the switch from proliferation to hypertrophy are unclear. We demonstrated, developing a novel cell free system, that DNA of adult cardiomyocytes can be replicated. We identified p38 MAP kinase as a key negative regulator of cardiomyocyte proliferation and subsequent studies revealed that p38 MAP kinase plays also an important role in the regulation of mitosis and cytokinesis. Moreover, we showed that inhibition of p38 MAP kinase together with FGF1 stimulation can induce adult cardiomyocyte proliferation in vitro. Finally, we could translate this knowledge into a therapy for acute heart failure in an animal model. Currently, we are employing bioinformatic strategies to elucidate regulatory mechanisms that control cardiomyocyte proliferation during development. Taken together, our data suggest that functional regeneration of the heart based on cardiomyocyte proliferation is possible.

Aula Seminari
Dipartimento di Informatica e Sistemistica, piano D
Martedì 11 dicembre 2007, ore 16,00

I dottorandi e gli interessati sono cordialmente invitati.

L'organizzatore — Prof. Riccardo Bellazzi

Il coordinatore del dottorato — Prof. A. Buizza