STEM CELL BIOLOGY LABORATORY

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Head of Laboratory
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Major position/appointments and professional training

- Principal Investigator, Scientific Researcher grade I
- Member of the Scientific Council of ICBP-NS
- PhD Coordinator
- Supervision of Graduate Students and Postdoctoral Fellows
- Expert Evaluator for National and International Grants
- Invited Peer Reviewer for International Scientific Journals

MAJOR RESEARCH INTERESTS

- **Regenerative therapies for injured heart**
- **Mesenchymal stem cell-based approaches for type 1 diabetes**

SPECIAL TECHNICAL EXPERTISE

- Flow-cytometry analysis and cell sorting
- Confocal microscopy
- Animal models of myocardial infarction and hind limb ischemia
- Ex vivo platforms mimicking cell-cell functional interactions in cardiac tissue

PUBLICATIONS

32 scientific articles, with more than 1000 citations (Google Scholar).

SELECTED NEW FINDINGS OF THE LABORATORY

• Concurrent transplantation of Endothelial Colony Forming Cells and factors released by cultured Circulating Angiogenic Cells enhanced the outcome of angiogenic therapy, compared to any single element.

• Mesenchymal Stem Cells and Endothelial Progenitor Cells have several distinct, yet complementary, paracrine effects, so that the combination of these two cell populations produced many benefits after transplantation.

• Subcutaneous transplantation of Mesenchymal Stem Cells could be considered as a safe and non-invasive procedure to induce protection and repair of the ischemic heart.

• Short treatment of Mesenchymal Stem Cells with 5-azacytidine resulted in a restricted differentiation potential with concomitant increased chondrogenic commitment.

• An insult may cause apoptosis or necrosis in endothelial cells, as a function of the intensity rather than its nature.

• Cardiomyocyte apoptosis was not induced by ischemia per se, but rather by the oxidants from the surrounding environment at the time of reperfusion.

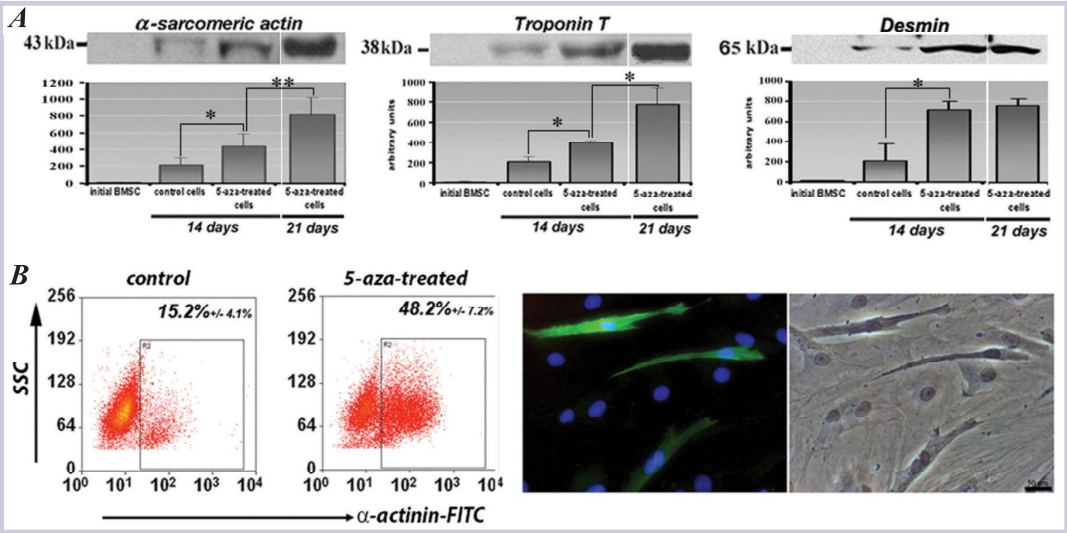
PREVIOUS PROJECTS /PUBLICATIONS /

1. THE SIGNALING PATHWAYS INVOLVED IN THE OXIDATIVE STRESS-INDUCED APOPTOSIS OF ENDOTHELIAL CELLS

The role of reactive oxygen species (ROS) in the pathogenesis of vascular diseases was well established, but few data existed on the mechanisms by which ROS induced endothelial cells (EC) death. Our results showed that (i) oxidative stress induced EC death either by apoptosis or necrosis and (ii) the mechanisms of EC death differed as a function of the oxidative stress intensity. Thus, the same insult can cause apoptosis and/or necrosis, as a function of the intensity rather than the nature of the offense (Burlacu A et al., Severity of oxidative stress generates different mechanisms of endothelial cell death. Cell and Tissue Research. 2001; 306: 409-416).

2. IN VITRO DIFFERENTIATION OF ADULT BONE MARROW STROMAL CELLS INTO CARDIO-MYOCYTES

In the attempt to increase the myogenic commitment of bone marrow stromal cells (BMSC), we investigated the extent of conversion induced by the demethylation agent 5-azacytidine. Our results demonstrated a promoting effect of 5-azacytidine on the expression of muscle-specific proteins and genes in BMSC in culture.



Priming of BMSC to cardiomyogenic differentiation with 5-azacytidine. (A) Western blot analysis of cardiac-specific proteins in the initial BM aspirate, untreated and 5-azacytidine-treated BMSC. (B) Flow-cytometry analysis (left) and ICC staining (right) of α-actinin staining in 5-azacytidine-treated cells after 14 days.

Priming of BMSC to cardiomyogenic differentiation may have significant applications in cellular approaches to ameliorate muscle loss after myocardial ischemia (Rosca AM, Burlacu A. Effect of 5-azacytidine: evidence for alteration of the multipotent ability of mesenchymal stem cells. Stem Cells Dev. 2011; 20:1213-1221, Burlacu A et al., Promoting effect of 5-azacytidine on the myogenic differentiation of bone marrow stromal cells. Eur J Cell Biol. 2008; 87: 173-184).