SCIENTIFIC PROGRAM

January 27th-29th 2021

.online

XX Congress of the Brazilian Society for Cell Biology

SBBC
Sociedade Brasileira de Biologia Celular

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Stress-inducible Protein 1 (STI1) is a essential cochaperone for HSP70-HSP90 complex formation, playing a crucial role in proteostasis. Complete depletion of STI1 in mouse is lethal throughout unkown molecular mechanisms, demonstrating a key unexplored function of this protein in initial stages of mammalian development. The proteostasis machinery is enhanced in pluripotent stem cells, that can be used as a in vitro system to study development. Thus, mouse embryonic stem cells (mESCs) wild-type, downregulated (STI1-Δ) and overexpressing (STI1-OE) STI1 were used as a model to investigate the role of this cochaperone in pluripotency maintenance. STI1-Δ mESCs showed lower expression of pluripotency markers, such as alkaline phosphatase and the core transcription factors OCT4, SOX2 and NANOG, concomitant with decreased proliferation and increased levels of DNA-damage and apoptosis markers. On the other hand, mESCs STI1-OE showed enhanced expression of pluripotency factors and a substantial increase in proliferation rates, when compared to both wild-type and STI1-Δ. Additionally, a protective effect is observed in STI1-OE cells, based on the reduced expression. of apoptosis and DNA-damage markers. Furthermore, our data also demonstrate that STI1 may have an impact on the differentiation capacity of mESCs, since STI1-Δ embryoid bodies have reduced diameter and volume. Together, our results suggest that STI1, a component of proteostasis network, plays a fundamental role in pluripotency maintenance in mESCs. This work contributes to the still recent understanding of posttranslational control of pluripotency, helping to clarify possible central players, such as STI1 and its partners, as masters post-genomic controllers of the pluripotent phenotype.

SC007

A COMPARISON BETWEEN TWO POPULATIONS OF MESENCHYMAL STEM CELLS THAT CAN IMPROVE THE OSTEOGENESIS IMPERFECTA MOUSE PHENOTYPE

Topic: **Stem cells**

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Osteogenesis imperfecta (OI) is an inherited disease characterized by fragility, deformity and low bone density, besides other clinical manifestations. Type-I OI is the mildest and most common form of the disease, caused by a mutation in the COL1A1 gene and resulting in half normal-collagen production. Our purpose was to compare mesenchymal stem cells from bone marrow (BM-MSCs) with those from adipose tissue (AD-MSCs), both used for improving heterozygous oim mice phenotype, similar to human type-I OI. Mice were irradiated and cells injected into femoral condyles, bone mineral density (BMD) and femoral length were measured by X-ray, at the beginning and end of the assay. Femurs and quadriceps allowed bone fragility evaluation by biomechanical test and collagen Col1a1 and Col1a2 quantification via ELISA. BMD showed no significant difference between the groups, while femur length variation was higher in the BM-MSCs group, compared with the control (P=0.0301). Fragility was improved with AD-MSCs when flexion extension to fracture (P=0.0028) and time to fracture (P=0.0032) were evaluated. There was no significant difference in the maximum load supported by femurs until fracture. An increase in Col1a1 concentration with BM-MSCs in comparison with AD-MSCs (P=0.0281) was obtained. Although no significant difference in Col1a2 concentration was observed between the groups, a higher expression level was obtained with AD-MSCs. We can thus conclude that AD-MSCs were more efficient than BM-MSCs for improving bone quality in type-I OI. This project was supported by CNPq and FAPESP: the animal experimentation was approved by CEUA/IPEN and registered as 206/18.

SC008

INFLUENCE OF MATERNAL OBESITY ON RESIDENT CARDIAC STEM CELLS IN OFFSPRING AND ITS RELATIONSHIP WITH THE DEVELOPMENT OF CARDIOVASCULAR DISEASE.

Topic: **Stem cells**

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