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## 2-Hydroxyethyl Methacrylate (HEMA) treatment modulates the autophagic process in stem cell from human dental pulp through ERK/pERK signalling

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Autophagy is an intracellular process that degrades organelles or cellular components in order to ensure the maintenance of cell homeostasis. It can be considered a genetically programmed, adaptive response to stress. In restorative dentistry practice, free resin monomers can be released. The aim of this study was to investigate the effect of HEMA on proliferation and autophagy in human dental pulp stem cells (hDPSCs). Human DPSCs were treated with different concentrations of HEMA (3 and 5 mmol L<sup>-1</sup>). To evaluate the proliferation rate, MTT and trypan blue assays were used. Autophagic markers such as microtubule-associated protein 1 light chain 3 (LC3-I/II) and ubiquitin-binding protein (p62) were analyzed through immunofluorescence observations. Beclin1, LC3-I/II, and p62 were evaluated by means of Western blotting detection. Considering that activity of extracellular signal-regulated kinase (ERK) and its phosphorylated form (pERK) mediates several cellular processes, such as apoptosis, autophagy, and senescence, the involvement of ERK/pERK signaling was also evaluated. Our results showed a decreased cell proliferation associated with morphological changes in HEMA-treated cells. The Western blot results showed that the expression levels of Beclin1, LC3-I/II, and ERK were significantly elevated in HEMA-treated cells and in cells co-treated with rapamycin, an autophagic promoter. The expression levels of p62 were significantly reduced compared to the untreated samples. Protein levels to the autophagic process, observed at confocal microscopy

confirmed the data obtained from the Western blot. The up-regulation of ERK and pERK levels, associated with nuclear translocation, revealed that ERK pathway signaling could act as a promoter of autophagy in dental pulp stem cells treated with HEMA. Then in response to HEMA injury, dental pulp stem cells activate autophagy as a pro-survival cytoprotective mechanism. Further studies are necessary to consider the strategic and therapeutic applications of this research in tissue repair and regeneration.

### Speaker Biography

Guya Diletta Marconi is a Researcher in Biomedical field at Department of Medical, Oral and Biotechnological Sciences, University of Chieti, Italy. She spent one year as a postdoctoral researcher at Torrey Pines Institute for Molecular Studies, in San Diego. She received her Ph.D. in April 2016 in Drug Sciences at University of Chieti, she spent half of her PhD abroad as visiting PhD student at the Sanford Burnham Prebys Medical Discovery Institute in San Diego, where she extended her skills and experience with a focus on the identification and validation of peptides inhibitors of target proteins involved in cancer progression. She works with multidisciplinary group that carry highly translational basic science research. The laboratory of Prof. O. Trubiani, where she works as a researcher, used mesenchymal oral stem cells, aimed to validate novel bioactive scaffolds as promising new approaches for tissue regeneration and repair. The current research is focused on mechanisms involved in stem cell-regulated tissue homeostasis and repair, particularly on the identification of molecules and phenotypic changes responsible for the regulation of the stem cell regenerative potential.

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