BOOK OF ABSTRACTS



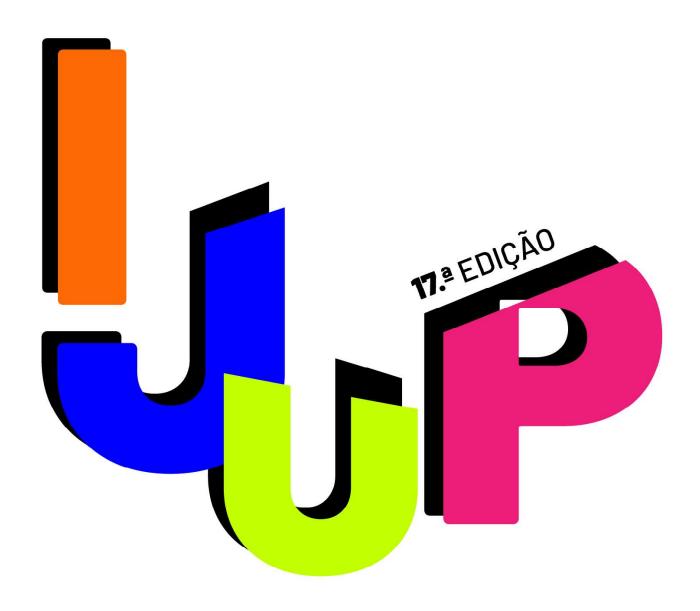
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21703 | Unveiling uterine senescence and its potential role in age-related female fertility decline

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Background & Aim: Nowadays, infertility is a major topic present in public health discussions due to its increased prevalence worldwide. With time, female infertility rates have increased, mainly, because women are delaying childbearing to their late 30s/early 40s, leading to fertility decline, complications during gestation, and a higher demand for assisted reproductive techniques. Uterine alterations have been pointed out as an important contributor, as uterine function is linked with the ability to conceive and carry a healthy pregnancy. Previous data reported uterine alterations in reproductively aged mice (1). Also, using human uterine samples, it was shown an age-related increase in albumin carbonylation (2) and variations in the oxidative status of extracellular matrix proteins. We believe that cellular senescence contributes to uterine alterations, impacting tissue microenvironment and impairing its function. In this project, we aim to evaluate the appearance of senescence-related uterine alterations with increasing age. Methods: Uterine samples from term-pregnant women with ages between 20 and 41 were homogenized, protein lysates were submitted to western blot analysis for relative quantification of senescence-associated proteins, including markers of nuclear damage and Senescenceassociated secretory phenotype (SASP) constituents. Results: Currently, our results show a strong and significant positive correlation between nuclear proteins, such as HP1, Lamin B1, and PH2AX, and age. Regarding pro-inflammatory proteins, MCP1 tended to increase with age while IL6 and IL1 β did not change. Also, SASP proteins like MMP3 and PAI1 presented a weak negative correlation with age. Conclusions: In the uterus, reproductive ageing courses with increased senescence-associated nuclear alterations reflecting the existence of DNA damage and organelle destabilization. Further analysis is needed to confirm the absence of significant variations in SASP proteins.

Keywords: Uterine Senescence, Female Fertility, Reproductive Ageing.

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