IJAE
Vol. 123, n. 1 (Supplement): 160, 2018
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

# The endocrine disruptor Bisphenol A affects cell proliferating ability in PHA-stimulated peripheral lymphocytes exerting a biphasic effect

Stefania Lucia Nori[1], Raffaella D'Auria[1], Mario Capunzo[1], Andrea Viggiano[1], Antonietta Santoro[1] and Vanessa Nicolin[2]

[1] Università di Salerno, Dipartimento di Medicina, Chirurgia e Odontoiatria "Scuola Medica Salernitana", Baronissi
[2] Università degli Studi di Trieste, Dipartimento CSID, Trieste

In the last two decades, a growing number of studies highlighted the importance of the interaction between the estrogen-receptor pathway modulation and immune cell activity in human metabolic regulation [1]. It is recognized that steroid pathways can regulate immune cell metabolism and thus the intacrine-paracrine effects of steroids could be implicated in promoting immune-mediated steroid-dependent cancers and autoimmune diseases. Moreover, it has been shown that both monocytes and T-lymphocytes express estrogen receptors (ER$\alpha$ and ER$\beta$) [2], therefore it is plausible that environmental pollutants interfering with steroid pathways, such as Bisphenol A (BPA) -a widespread contaminant of plastic, epoxy resins, toys and electronics with structural and functional similarities to steroids - could influence immune cell activity leading to cancer, autoimmune disease and neurological disorders. To gain insights into this issue, we studied the effects of BPA in Phytoemagglutinin (PHA)-stimulated Peripheral Blood Mononuclear Cells (PBMCs) from healthy donors examining cell survival by MTT assay, cell proliferation by BrdU assay, and cell cycle progression by cytofluorimetric analysis at different time points (24-72 hs) and concentrations (ranging from 5 nM to 200 $\mu$M).

Results show that BPA does not induce apoptosis or necrosis, at all time and doses tested, instead promotes cell proliferation at lowest concentrations. Cytofluorimetric analysis by Propidium Iodide staining also indicates that BPA is able to emphasize PHA-induced effects in enhancing cell proliferation at concentrations ranging from 25 to 100 nM. However, the compound markedly inhibits cell cycle progression at concentrations greater than 25 $\mu$M causing a G1 cell cycle arrest starting from 24 hs of treatment. On the other hand, BPA was unable to induce any effect in resting PBMCs, suggesting that BPA -induced biphasic effect in proliferating cells could be the result of mechanisms other than its estrogen-like behavior.

## References

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Key words

Bisphenol A, PBMCs, cell cycle progression.