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ABSTRACT BOOK

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ENDOCRINE DISRUPTORS AS THREAT TO ECOSYSTEMS FUNCTION: A GENOTOXICOLOGICAL APPROACH

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The Endocrine Disruptor Chemicals (EDCs) are compounds of not steroid origin which mimic estrogens and bind to estrogen receptors, interfering with the endocrine system. Many commercial chemical agents, including pesticides, like polychlorinated biphenyls and alkylphenols, or personal care products, such as shampoos, soaps, toothpastes, sunscreens and lotions, showed estrogenic effects on animals. Most of these compounds pass into the environment through industrial discharges, and, consequently, can be found in the atmosphere, in the soil, in the water cycle and can potentially enter the food chain. Exposure occurs through drinking contaminated water, breathing contaminated air, ingesting food, or contacting contaminated soil and people who work with pesticides, fungicides, and industrial chemicals are at particularly high risk for developing reproductive or endocrine abnormalities.

In the last decade, the number of researches about the dangerous effects of these compounds has increased. However, most of these studies showed an approach, both *in vitro* and *in vivo*, based on toxicologically relevant concentrations, whereas few data were reported about the effects of small doses of these compounds. *Vice versa*, in the present paper, we show results obtained by our group for some ECDs (Glyphosate, Chlorothalonil, BP-A and BP-3) tested at low concentrations, starting from the Acceptable/Tolerable Daily Intake (ADI/TDI) values adopted by Council of Europe, US EPA and FAO (0.50 µg/mL, 0.02 µg/mL, 0.05 µg/mL and 0.10 µg/mL for Glyphosate, Cholothalonil, BP-A and BP-3, respectively).

We evaluated the genotoxicity of these compounds by Chromosomal Aberrations (CAs) and Micronuclei (MNi) assays that allow the evaluation of the clastogenic and/or aneugenic properties of single compounds or mixtures of them. In particular, the CAs assay allows the detection of cells carrying unstable aberrations (chromosome and chromatid breaks, deletions, fragments and rings) whereas MNi represent acentric chromosomal fragments or whole chromosomes left behind during mitotic cell division and that appear in the cytoplasm of interphase cells as small additional nuclei.

As general result, we observed a significant increase of CAs and MNi frequencies at all tested concentrations, including the ADI/TDI concentration values and, in some cases, also their submultiples, indicating possible clastogenic and aneugenic effects of these compounds also at low concentrations. Although the limitations typical of *in vitro* studies, it is our opinion that the increased cytogenetic damage observed by our group at ECDs concentrations equal and lower than the established ADI/TDI values requires further investigations in order to establish the effective genotoxicity threshold of these extensively used compounds.