POSTER

NEURODEGENERATION AND DNA DAMAGE FOLLOWING CO-EXPOSURE TO POLYSTYRENE MICROPLASTICS AND BISPHENOL A IN ZEBRAFISH EMBRYOS

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The increasing co-occurrence of multiple environmental pollutants represents an emerging concern for the health of living organisms, as interactions can alter their bioavailability, toxicity, and overall biological impact. Contaminants such as microplastics can interact with other environmental pollutants through their hydrophobic surfaces, adsorbing and transporting them. These interactions are still under investigation, as they depend on several factors, including binding forces, pH, and temperature [1]. In this study, we investigated the co-exposure effects of polystyrene microplastics (PS-MPs) and bisphenol A (BPA) in the first 72 h of zebrafish development. Five experimental groups were established: a control group, a group exposed to PS-MPs (1 mgL-1), a group exposed to BPA (25 µM), a co-exposure group (PS-MPs + BPA), and a group treated with 0.01% of dimethyl sulfoxide (DMSO) to exclude the solvent toxicity. The results showed that both PS-MPs and BPA impaired the expression and activity of poly(ADP-ribose) polymerase (PARP), as well as the expression of glial fibrillary acidic protein (GFAP). BPA also decreased acetylcholinesterase activity (AChE), unlike PS-MPs alone. Furthermore, gene expression analysis of neurodevelopmental markers (nkx2.2a, elavl3, nes, syne1a, reln, gabrr2, gria3a, parp1, gfap) revealed significant dysregulation in the groups exposed to PS-MPs or BPA individually. Notably, the co-exposure group exhibited reduced biological impact. Analysis of PARP activity revealed a return to normal conditions in the co-exposure, while in the GFAP expression and AChE activity minor damage in the co-exposure group was observed. These data were consistent with gene expression profiles in which, in some cases, the damage was completely reversed, while in others it was mitigated. These results add information to our previous study [2], supporting the hypothesis that PS-MPs may sequester BPA, thereby reducing the concentration of free BPA and its subsequent bioavailability.

References

1. Zhu Y Y et al. J.Hazard Mater Adv; 2022;5:100046.
2. La Pietra A et al Environ Toxicol Pharmacol; 2025; 121:104919.