OFFICIAL ABSTRACT and CERTIFICATION

Xenoestrogen Bisphenol-A's Neurotoxicity via Estrogenic Activity and Resulting Alzheimer's Disease Pathogenesis

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Alzheimer's Disease (AD) affects millions of people in the U.S. alone, yet there is currently no effective cure on the market. Exact causes of AD are elusive, but many studies have seen a correlation between environmental pollution and AD incidence rates. This project evaluated how neurotoxicity of Bisphenol-A (BPA), a ubiquitous pollutant and xenoestrogen, is linked to AD via estrogen receptor signaling.

My results have shown that BPA at low concentrations significantly reduced neuronal cell survival in a dose-dependent manner, demonstrating the xenoestrogen's neurodegenerative properties, and significantly exacerbated amyloid-beta neurotoxicity in a synergistic manner. Through ELISA, I observed that BPA induced abnormally elevated levels of Amyloid Precursor Protein (APP), which demonstrates BPA's potential to indirectly promote amyloid plaque depositions—formations largely responsible for nervous system damage and AD symptoms (i.e. memory loss). Further experimentation with immune cells also revealed that BPA activated immune response via promoting the expression of pro-inflammatory cytokines, such as interleukin-1beta (IL-1beta) and tumor necrosis factor-alpha (TNF-alpha), and therefore can induce neuroinflammation. Furthermore, Tamoxifen, an antagonist for estrogen receptor-alpha (ER-alpha), produces deleterious, synergistic effects when co-incubated with BPA and 17beta-estradiol (E2) in immune cells, suggesting that BPA ' s toxicity to immune cells is possibly exerted through interactions with ER-alpha.

In sum, this study discovered a novel link between BPA's toxicity and Alzheimer's pathophysiological formation; urgent investigation on animal and human models is needed for quick action to ban or heavily reduce plastic disposal in the environment, preventing leachates like BPA from negatively impacting the human body and the ecosystem.

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