Title: *Bisphenol A differentially impacts neurodevelopment in Drosophila melanogaster from distinct genetic backgrounds*

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Abstract: Bisphenol A (BPA)—an environmentally prevalent chemical used to manufacture plastics—may be a risk factor for neurodevelopmental disorders (NDDs). Yet, how BPA impacts neurodevelopment at the cellular level is largely unknown. Another outstanding question is if BPA exposure more severely impairs neurodevelopment in individuals with genetic risk factors for NDDs. Drosophila melanogaster are an ideal model for testing the developmental neurotoxicity of chemicals due to their short generation time, simple and well-characterized brain structure, easily quantifiable behaviors, and genetic tractability. We investigated how BPA affected neurodevelopment in two distinct genetic lines of fruit flies: w1118 (control) and the Fragile X Syndrome (FXS) model. FXS flies have a mutation in the gene Drosophila fragile X mental retardation 1 (dFmr1), which causes neurodevelopmental phenotypes that parallel phenotypes observed in Fmr1-mutant mammals. Fmr1-associated phenotypes include impaired neural stem cell (NSC) development and reduced courtship activity. Using fluorescent labelling and microscopy to image NSCs in the larval Drosophila brain, we found BPA exposure in control flies had no impact on the number of NSCs but reduced the NSC proliferation rate. In FXS flies, BPA exposure reduced the number of NSCs but had no impact on their proliferation rate. Using the courtship assay, we found that BPA exposure reduced courtship activity of control flies. FXS flies already have impaired courtship activity and exposure to BPA neither rescued nor enhanced this phenotype. This study demonstrated that the neurodevelopmental phenotypes caused by BPA can vary drastically in response to different genetic backgrounds.

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