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Exposure to THI during gestation and lactation impairs the striatum, hippocampus and cognition in rat offspring (G1/G2): The role of apricot kernel extract

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Thiacloprid (THI) is a widely used neonicotinoid suspected to exert neurotoxic effects in mammals, while bitter apricot kernels are traditionally used in folk medicine for the treatment of various diseases. In the context of limited data on thiacloprid-induced multigenerational neurotoxicity, this study aimed to evaluate the neurotoxic effects of THI exposure in utero and during lactation in rat offspring, as well as to investigate the neuroprotective potential of bitter apricot kernel extract as a natural protective strategy. Female rats were gavaged with 0.02 mg/kg THI or/and 50 mg/kg bitter apricot kernel extract from day1 of gestation to day21 of lactation, neurobehavioral tests were performed to assess the cognitive function of the rats' offspring. In addition, bioanalytical methods were performed to determine swelling and mitochondrial permeability, AchE and cytochrome-c activities, and redox status in striatal and hippocampal mitochondria of rat generations (G1, G2) at 100 days of age. The results showed that thiacloprid exposure impaired neurological behavior and cognitive performance, including memory and learning, in both the G1 and G2 offspring. It also induced mitochondrial edema and disrupted the cellular redox status. Conversely, bitter apricot kernel extract exhibited a significant cytoprotective effect against this neurotoxicity. Finally, thiacloprid demonstrates multigenerational neurotoxicity, leading to neuronal impairments, which can be mitigated by apricot kernel extract.

Keywords: Thiacloprid, brain mitochondria, oxidative stress, cognition, multigenerational toxicity