The effects of plasticizer Dibutyl Phthalate on mammalian spinal locomotor activity

Francisco Diaz, Class of 2026

Currently, we live in a world where plastics are all around us. As a result, we continue to become more exposed to harmful chemicals emanating from the degradation of these plastics. A typical example of exposure is through our diet. Chemicals in plastic containers can latch onto our food. Additionally, these compounds reach bodies of water that contain fish, which we consume.

Of these environmental contaminants is a family of chemicals called phthalates. They are used as industrial additives to enhance the flexibility of plastics. Dibutyl phthalate, or DBP, is one of these chemicals. DBP is found in various daily products, such as children's toys, medical supplies, and beauty products. Research has linked DBP to cell death, disruption in reproductive mechanisms, and neurotoxicity. Previous studies suggest DBP induces oxidative stress, an accumulation of harmful and unstable molecules that harm surrounding molecules by stealing electrons. Our work aimed to evaluate the neurotoxic effects of DBP on the central pattern generator network (CPG), a circuitry of neurons capable of generating rhythmic activity, in the spinal cord of neonatal mice.

Spinal cords were isolated from neonatal mice, ages 0 - 5 days, via a ventral laminectomy after euthanization. To acquire fictive locomotion, an active and bursting spinal cord at this stage, we perfused the spinal cords with Ringer solution and neurotransmitters NMDA at 6 μM and serotonin at 9-12 μM. Sucking electrodes were positioned at flexor (L2) and extensor (L5) ventral roots to register their action potentials or bursting. Once the burst activity was determined to be phase-locked, consistent bursting, the control began for 35 minutes. After the control, DBP at either 10 μM, 50 μM, or 100 μM concentrations was introduced for 75 minutes. Finally, the spinal cord was washed for 45 minutes.

The parameters of this experiment were Burst Amplitude, the height of a burst, Burst Duration, the length between the start and end of a burst, and Cycle Period, the length between the start of a burst and the start of the next occurring burst. Burst Amplitude offered a look into the recruitment of motoneurons, while the other two parameters suggested the state of the CPG network’s excitability. Because we were expecting DBP to induce neurotoxicity in the spinal cord, we anticipated a decrease in amplitude as a result of a reduction of motoneuron recruitment and an increase in Burst Duration and Cycle Period as a result of an affected CPG network, which would make bursts longer and slower as opposed to fast and robust.

Faculty Member: Manuel Díaz-Ríos

Funded by the Kufe Family Research Fellowship

Acevedo, J., Santana-Almansa, A., Matos-Vergara, N., Marrero-Cordero, L. R., Cabezas-Bou, E., & Díaz-Ríos, M. (2016). Caffeine stimulates locomotor activity in the mammalian spinal cord via adenosine A1 receptor-dopamine D1 receptor interaction and PKA-dependent mechanisms. *Neuropharmacology*, *101*, 490–505. https://doi.org/10.1016/j.neuropharm.2015.10.020