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Review

Impact of endocrine-disrupting chemicals on neural development and the onset of neurological disorders

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Abstract:
Even though high doses of organic pollutants are toxic, relatively low concentrations have been reported to cause long-term alterations in functioning of individual organisms, populations and even next generations. Among these pollutants are dioxins, polychlorinated biphenyls, pesticides, brominated flame retardants, plasticizers (bisphenol A, nonylphenol, and phthalates) as well as personal care products and drugs. In addition to toxic effects, they are able to interfere with hormone receptors, hormone synthesis or hormone conversion. Because these chemicals alter hormone-dependent processes and disrupt functioning of the endocrine glands, they have been classified as endocrine-disrupting chemicals (EDCs). Because certain EDCs are able to alter neural transmission and the formation of neural networks, the term neural-disrupting chemicals has been introduced, thus implicating EDCs in the etiology of neurological disorders. Recently, public concern has been focused on the effects of EDCs on brain function, concomitantly with an increase in neuropsychiatric disorders, including autism, attention deficit and hyperactivity disorder as well as learning disabilities and aggressiveness. Several lines of evidence suggest that exposure to EDCs is associated with depression and could result in neural degeneration. EDCs act *via* several classes of receptors with the best documented mechanisms being reported for nuclear steroid and xenobiotic receptors. Low doses of EDCs have been postulated to cause incomplete methylation of specific gene regions in the young brain and to impair neural development and brain functions across generations. Efforts are needed to develop systematic epidemiological studies and to investigate the mechanisms of action of EDCs in order to fully understand their effects on wildlife and humans.

Key words:
estrogen receptors, aryl hydrocarbon receptors, PPARγ, RXR, fetal basis of adult-onset disease, nervous system