Effects of polychlorinated biphenyls on the nervous system.

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The neurological effects of polychlorinated biphenyls (PCBs) have been extensively investigated in humans and in animals. The main focus in human studies has been on the effects in neonates and young children, although studies of adults have also been conducted. A great deal of concern exists that even low levels of PCBs transferred to the fetus across the placenta may induce long-lasting neurological damage. Because PCBs are lipophilic substances, there is also concern that significant amounts might be transferred to nursing infants via breast milk. Studies in humans who consumed large amounts of Great Lakes fish contaminated with environmentally persistent chemicals, including PCBs, have provided evidence that PCBs are important contributors to subtle neurobehavioral alterations observed in newborn children and that some of these alterations persist during childhood. Some consistent observations at birth have been motor immaturity and hyporeflexia and lower psychomotor scores between 6 months and 2 years old. There is preliminary evidence that highly chlorinated PCB congeners, which accumulate in certain fish, are associated with neurobehavioral alterations seen in some newborn children. Subtle neurobehavioral alterations have also been observed in children born to mothers in the general population with the highest PCB body burdens. Because of the limitations of epidemiological studies, these effects cannot be attributed entirely to PCB exposure. In one general population study, there was strong evidence that dioxins, as well as PCBs, were contributors to the neurobehavioral effects seen in exposed children. Children born to women who accidentally consumed rice oil contaminated with relatively high amounts of PCBs and chlorinated dibenzofurans (CDFs) during pregnancy also had neurodevelopmental changes.

Studies in animals support the human data. Neurobehavioral alterations have also been observed in rats and monkeys following prenatal and/or postnatal exposure to commercial Aroclor mixtures, defined experimental congener mixtures, single PCB congeners, and Great Lakes contaminated fish. In addition, monkeys exposed postnatally to PCB mixtures of congenic composition and concentration similar to that found in human breast milk showed learning deficits long after exposure had ceased. A few other generalizations can be made from the data in animals. It appears that ortho-substituted PCB congeners are more active than coplanar PCBs in modifying cognitive processes. In addition, one effect observed in both rats and monkeys--deficits on delayed spatial alternation--has been known to be induced by exposure to ortho-substituted PCBs, defined experimental mixtures, and commercial Aroclors. Both dioxin-like and non-dioxin-like PCB congeners have been shown to induce neurobehavioral alterations in animals. Changes in levels of neurotransmitters in various brain areas have also been observed in monkeys, rats, and mice. Of all the observed changes, the most consistent has been a decrease in dopamine content in basal ganglia and prefrontal cortex, but further research is needed before specific neurobehavioral deficits can be correlated with PCB-induced changes in specific neurotransmitters in specific brain areas.

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