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The Impact of Polybrominated Diphenyl Ether (BDE-47) Administration in Mice and its Implications in Non-alcoholic Fatty Liver Disease

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The Impact of Polybrominated Diphenyl Ether (BDE-47) Administration in Mice and its Implications in Non-alcoholic Fatty Liver Disease

BDE-47 is a brominated flame-retardant widely found in the environment. It can be found in the air, soil, water, and many household products, such as cars, computers, and furniture. Previous studies have shown that BDE-47 can accumulate in body tissues after human exposure to the compound, including in the human liver. This accumulation over time may contribute to a higher risk of Non-alcoholic fatty liver disease (NAFLD), which is characterized by the accumulation of fat in liver cells that is not due to alcohol consumption. Over time, this can lead to hepatic steatosis, liver cirrhosis, and eventually liver cancer or failure.

Preliminary studies in human hepatocytes (HepG2 cells) have displayed an increase in lipids, triglycerides, and modulated lipogenic gene expression after being treated with BDE-47, which suggests that BDE-47 has the ability to induce lipid accumulation in the liver. BDE-47 was then tested further in an animal study, in which it was hypothesized that administration of BDE-47 in mice fed either a normal or high fat diet would increase liver fat content. Adult male mice were fed BDE-47 through their diets (0.0003% in 10% kcal or 45% kcal high fat diets) over a period of 8 weeks. Food consumption and body weights were measured every 2-3 days. After 8 weeks, a necropsy was performed, in which mice livers were collected for further analysis. Liver lipid, triglyceride, and cholesterol contents were then quantified using UV spectrophotometry.

Average mouse body weights were graphed, showing a steady increase in the body weights of each treatment group over the time period of 8 weeks. Percent body weight gain was also graphed, and showed a significant increase in high-fat diet over the low-fat diet control. The treatment group fed both high-fat diet and BDE-47 showed significant percent weight change compared to both the low- and high-fat diet controls. After liver fat content was analyzed and graphed, the mice treated with BDE-47 showed slightly lower hepatic lipid, triglyceride, and cholesterol content than the low- and high-fat-treated control mice. It was therefore concluded that the results of the animal study were not consistent with the data from the preliminary studies in HepG2 cells, in which there was an increase in total lipids and triglycerides in the BDE-treated cells. While the cell model supported the hepatic steatosis and NAFLD fat accumulation hypotheses in human hepatocytes, the animal model data does not support this; further research should be done to determine its implications in NAFLD. The next steps include analyzing gene expression using protein and RNA isolation samples, to conclude whether the lipid data observed can be supported by changes in gene expression.