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COMMENTARY

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A Brief Reference to Liver Damage Caused by Chemicals

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Commentary

The liver is the main organ responsible for the metabolism of drugs and toxic chemicals, and so is the primary target organ for many organic solvents. Work activities with hepatotoxins exposures are numerous and, moreover, organic solvents are used in various industrial processes. Organic solvents used in different industrial processes may be associated with hepatotoxicity. Several factors contribute to liver toxicity; among these are: species differences, nutritional condition, genetic factors, interaction with medications in use, alcohol abuse and interaction, and age. This review addresses the mechanisms of hepatotoxicity. The main pathogenic mechanisms responsible for functional and organic damage caused by solvents are: inflammation, dysfunction of cytochrome P450, mitochondrial dysfunction and oxidative stress. The health impact of exposure to solvents in the workplace remains an interesting and worrying question for professional health work. Some studies have suggested that exposure to organic solvents may induce liver toxicity because most chemicals are metabolized in the liver and toxic metabolites generated through the metabolism are the main cause of liver damage.

Organic solvents are used in various industrial processes such as spray painting, paint manufacturing, degreasing, metal processing, aeronautical and auto manufacturing maintenance and manufacturing, as well as various chemical storage facilities. Exposure to hepatotoxins can occur through intentional or accidental ingestion in food or absorption of toxic contaminants through the skin. Contamination includes the ingestion of water, skin absorption via water baths, and volatilization of solvents, and heated bathrooms with a shower of water. Although a number of industrial chemicals are known to be hepatotoxins, liver disease from occupational exposure is rarely suspected or diagnosed.

Three conditions must be fulfilled for the diagnosis of professional toxic hepatitis: Liver damage should take place after occupational exposure to a substance; patient occupational history and the workplace in question is necessary; Liver enzymes must increase to at least double the upper limit of normal levels; and Tertiary conditions, such as other causes of liver disease, must be excluded. The most important factors contributing to toxicity liver are protein binding, species differences, points of binding inside the liver intracellular, nutritional condition, genetic factors, interaction with medications in use, alcohol abuse and interaction, and age. For the age factor, it has been shown that age susceptibility clearly plays a role. For instance, neonatal rats are less susceptible to carbon tetrachloride and bromobenzene toxicity as compared to adult animals. The hepatotoxic effects of some of the solvents were recognized as early as 1887. Very little is known about the frequency of occupational liver injury by solvents. It is still difficult to assess the damage from exposure due to difficult controls in the workplace. Clinical presentation of occupational liver disease may be acute/subacute or chronic, but is often insidious. Liver damage is likely to be more severe in the hepatocellular type than in the cholestatic or mixed type; a patient with elevated bilirubin levels in hepatocellular liver injury indicates serious liver disease. Patients with the cholestatic or mixed type are likely to develop chronic disease more frequently than those with the hepatocellular type. The solvents suspected to be responsible for liver occupational disease are: dimethylformamide (DMF), dimethylacetamide (DMA), trichloroethylene (TCE), tetrachloroethylene, carbon tetrachloride, xylene, toluene, and chloroform.

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