Behavioral Pharmacology of Inhalants

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Categories of Inhalants

Inhalants comprise a disparate group of mind-altering substances that are classified by their intake route. Despite more than 7 million users, there is a paucity of medical and neuropsychological literature. Only rarely are investigations into inhalant abuse reported in prominent substance abuse journals. This is probably due to multiple factors including (1) difficulty in studying or neglect of the population in which inhalant use is most prevalent, namely, individuals in lower socioeconomic strata; (2) the finding that use rarely occurs in isolation from other polydrug use or exposure to other inhalants; and (3) the fact that these substances are not generally classified as abused drugs because they are commonly available legal products.

Inhalants are grouped into four categories:

1. **Volatile gases and solvents** (e.g., lighter fluid, barbecue grill gas, spray paint, paint thinner, glue): The active compound is generally acetone, ethyl acetate, n-hexane, propane, n-butane, toluene, methylbutylketone.

2. **Propellant aerosols** (e.g., hair spray, deodorants, other pressurized products): The intoxicating inhalant is the propellant rather than the active ingredient. Active compounds include methylene chloride, isobutane, propane, bromochlorodifluoromethane, and various halons including trichlorofluoromethane, dichlorodifluoromethane, chlorodifluoromethane, and dichlorotetrafluorothane.

3. **Nitrites**: These compounds include butyl nitrites and amyl nitrites. The former are often known by the trade names Rush, Locker Room, Bolt, and Climax. They are intended for use as room deodorizers. The latter are packaged in capsules known as “poppers” or “snappers.” They were originally administered to treat angina.

4. **Anesthetic gases**: These compounds, most notably nitrous oxide (“laughing gas”), are typically contained in small cylinders called whippets, which propel whipped cream in commercial machines. Other common compounds include halothane and enflurane.

Neurobehavioral Effects

Typically the acute effects are manifest as disinhibited behavior and euphoria. High doses are capable of precipitating an acute psychosis and seizures (Brust, 1993). Chronic

Overdose of inhalants is far more likely to produce lethal consequences than are noninjectable drugs. General causes of fatalities include respiratory arrest, vomiting with aspiration, or suffocation from plastic bag inhalation (Brust, 1993). The specific cause of death may depend upon the substance. For example, death from anoxia has been reported from nitrous oxide, whereas fatal cardiac arrhythmia and cerebral edema have resulted from exposure to fluoroalkane propellants, particularly trichlorofluoromethane (Freon 11), dichlorodifluoromethane (Freon 12), and solvents found in typewriter correction fluid (Aviado, 1977; King, Smialek, & Troutman, 1985). Cardiac arrhythmia may occur over a period of several hours after initial use (Brust, 1993). In one cohort of fatalities the most prevalent manner of death was suicide (28 percent), leading Garrett (1992) to suggest the possibility of an acute drug-induced disinhibitory effect on a population known to have elevated rates of clinical depression.

**Alkyl Halides**

Exposure to this diverse group of predominantly chlorinated and fluorinated hydrocarbons (methyl chloride, freons) typically produces nausea, vomiting, fatigue, and muscle pain. Hepatitis, acute renal tubular necrosis, or both may follow exposure in 1 to 3 days (Linden, 1990). One college-age experimenter with freon reported his reactions in an Internet discussion forum. He tried inhaling Freon after hearing that it “gave a similar buzz to nitrous (oxide).” Initially, “for about 2 seconds, I had a small buzz. Then my vision went wild. A wall about 5 feet from me seemed like it moved from two inches . . . to 100 miles . . . every second. Then my heart started beating like wild. As I collapsed to the floor, I managed to gasp out ‘get an ambulance!’” The individual was extremely frightened by the experience and concluded that he “would sooner drink gasoline than [inhale freon] again” (Dillinger, 1993). Such an individual might be considered lucky to recount his experiences, since cardiac arrhythmia and death have been known to follow exposure to fluoroalkane propellants (Aviado, 1977; Brust, 1993). Carbon monoxide poisoning is an additional complication of exposure to methylene chloride. Severe or fatal methemoglobinemia may be produced.

**Amyl and Isobutyl Nitrite**

Nitrite inhalants were originally used to reduce pain from angina and as an antidote to cyanide poisoning (Wood, 1988). Blood flow increase throughout the brain has been reported after administration of amyl nitrite (Mathew, Wilson, & Tant, 1989). Consequent symptoms of dizziness and headache are attributed to dilation of arteries in the brain. Test subjects administered amyl nitrite report increased feelings of anger, depression, and fatigue. Acute toxicity from anoxia-methemoglobinemia is a well-known side effect, and vital organs may become starved of oxygen because of “profound peripheral vasodilation, pooling of blood in the extremities and impaired vascular return” (Wood, 1988, p. 30). Nitrite drugs are immunotoxic and render AIDS patients more susceptible to Kaposi’s sarcoma.

Isobutyl nitrite, an inhalant similar to amyl nitrite, continues to be sold legally as a locker room deodorizer. Inhalation is similar to amyl nitrate. Symptoms include severe headache, blurred vision, and eye pressure (Wood, 1988). Inhalation produces a “high” lasting from a few seconds to several minutes. Intoxication is accompanied by a decrease in blood pressure followed by an increase in heart rate, flushed face and neck, dizziness, and headache. Toxicity from methemoglobin can be a cause of death (Wood, 1988).

**Butane**

Butane inhalation can result in burns in the case of cigarette lighter fluid abuse, or sudden death from cardiac dysrhythmia (Siegel & Wason, 1990). Tohhara, Tani, Nakajima, and