

# No laughing matter: a comprehensive overview of nitrous oxide abuse

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## No Laughing Matter: A Comprehensive Overview of Nitrous Oxide Abuse

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2013 SUBSTANCE OVERVIEW The issue of inhalant abuse is a multifaceted problem, stretching across many communities and locales in the entirety of the United States. Inhalants as a class of drug vary widely, ranging from volatile solvents and aerosol propellants, to more broadly, any drug delivered by inhalation. Nitrous oxide straddles these categories, as it finds use not only a common dental analgesic, but also as a propellant, in such uses as whipping cream, automotive racing, and rocketry.

As a result, nitrous oxide is found in many forms: medical-grade, food-grade, and industrial-grade, with various purities (Erowid). Commonly known as “laughing gas,” nitrous oxide wears many hats and goes by many names. For example, those who use nitrous oxide in automotive racing or rocketry applications know it as NOS (Alai). In this usage, nitrous oxide is applied as a powerful oxidizer, allowing a vehicle’s engine to combust gasoline more rapidly, with an associated increase in horsepower (Winter). This form of nitrous oxide is packaged with many additives to not only improve its performance but also to deter its abuse (Erowid).

When abused recreationally however, nitrous oxide finds many more colorful nicknames, such as “hippie crack” and “nangs” (Erowid). It is a true gas, existing as a gas under atmospheric pressure at room temperature, although it assumes a liquid state when compressed in a gas cylinder (Alai). Nitrous oxide is a strong oxidizing agent with a density of 1.9 kg/m<sup>3</sup>, and it has a linear molecular form consisting of two atoms of nitrogen and one atom of

oxygen (Winter). Nitrous oxide has no color and has a sweet odor, familiar to anybody who has tasted whipped cream (Erowid).

Nitrous oxide is commercially manufactured by heating ammonium nitrate to 240C. Impurities such as ammonia and nitric acid in addition to excess water vapor are removed through an extensive gas scrubbing process. Food-grade nitrous oxide is often stored in white, 8-gram cylinders with a sealed metal puncture cap and packaged in boxes of 100 cylinders. Medical-grade nitrous oxide is stored in French-blue cylinders and is pressurized to ~4400 kPa at room temperature. The Pin Index Safety System configuration for nitrous oxide cylinders is 3-5 (Banks and Hardman). HISTORY OF NITROUS OXIDE ABUSE

British chemist and Presbyterian minister Joseph Humphrey first synthesized nitrous oxide gas in England in 1772. Priestly later published his work in a 1776 journal, “ Experiments and Observations on Different Kinds of Air” (Priestly). This publication proved extremely intriguing to the scientific community, leading to additional research and Humphry Davy’s influential 1800 book, “ Researches, Chemical and Philosophical: Chiefly Concerning Nitrous Oxide. ” Throughout the remainder of the nineteenth century, nitrous oxide’s usage as a recreational drug became increasingly popular.

Traveling medical shows and carnivals featured affectionately named “ Nitrous Oxide Capers,” in which attendees paid a small fee to inhale a minute’s worth of gas (Brecher). In 1844, Dr. Horace Wells, a British dentist, first demonstrated the use of nitrous oxide as a dental anesthetic. His initial testing on a patient at Harvard Medical School was unsuccessful, resulting in

the public dismissal of nitrous oxide as a legitimate anesthetic. Nevertheless, nitrous oxide had a sudden resurgence in the dental community in the early 1860s, thanks to a series of dental institutions opened by lecturer and showman Gardener Quincy Colton.

These practices utilized nitrous oxide as their primary form of dental anesthesia, henceforth standardizing its usage in the United States (Erowid).

**METHODS OF ADMINISTRATION** Nitrous oxide is administered via inhalation of compressed gas. Users most frequently obtain nitrous oxide from small, 8-gram canisters used to charge whipped cream dispensers. These canisters are readily available for purchase in most restaurant supply stores, on the Internet, and in head shops across the nation (Narconon).

Once obtained, the contents of the canisters are released into empty whipped cream dispensers or into a balloon. A pressure release valve, or “cracker”, must be used in order to safely siphon the gas into a balloon (Erowid). These valves are also available for purchase online or in head shops (Narconon). Users with access to larger cylinders of nitrous oxide sometimes inhale the gas through a nasal hood or anesthesia mask, as used in dental surgery. However, this method is extremely dangerous without proper medical supervision, as users can inhale larger amounts of gas without access to fresh oxygen.

As a result, the vast majority of nitrous oxide users prefer to use the aforementioned whipped cream dispenser or balloon methods of inhalation (Wagner, Clark, Wesche, Doedens, and Lloyd). Whipped cream canisters and standard latex party balloons have a maximum capacity of approximately

three 8-gram cylinders. Depending on personal preference, users can fill their vessel of choice with one, two, or three cylinders at a time (Creamright). Once the whipped cream dispenser or balloon is filled to the user's liking, the gas inside is rapidly inhaled into the user's lungs.

The gas is often held in the lungs for twenty to sixty seconds to induce hypoxia and enhance the euphoric effects. The gas is then exhaled normally, and the user resumes regular respiration of room air (Erowid). **IMMEDIATE EFFECTS** Once inhaled, nitrous oxide is immediately absorbed through the lungs, dissolving directly into blood plasma. Within ten to fifteen seconds, nitrous oxide molecules replace oxygen molecules in the user's lungs, thereby stimulating a euphoric sensation. Users then experience a brief "high" usually lasting between thirty seconds and two minutes with a standard dose (Erowid).

Symptoms of nitrous oxide abuse include slurred speech, impaired coordination and balance, difficulty thinking clearly and processing information, unresponsiveness to verbal and painful stimuli, and occasionally loss of consciousness. Assuming the user returns to regularly breathing room air after the initial gas inhalation, these symptoms should subside within two minutes. The onset and peak effect timing of nitrous oxide is determined by the quantity consumed (Narconon). Nitrous oxide inhalation impacts the function of numerous physiologic systems.

First, nitrous oxide decreases tidal volume and increases respiratory rate following activation of the central nervous system. Myocardial depression and an increase in central sympathetic outflow also occur following nitrous

oxide inhalation. Inhaled nitrous oxide may produce the second gas effect, as nitrous oxide has a more rapid rate of diffusion across alveolar basement membranes than nitrogen gas. This rapid exit of nitrous oxide from the alveoli initiates a concentration of other alveolar gases, thereby accelerating the uptake of nitrous oxide into the bloodstream (Banks and Hardman).

The primary method of nitrous oxide elimination is via the exhalation from the lungs. Nitrous oxide exits the body entirely unchanged in chemical formula and structure. Small amounts of nitrous oxide diffuse through the skin and the renal system, and anaerobic bacteria in the GI tract reduce any outstanding nitrous oxide into nitrogen gas (Erowid). **ACTION ON THE BRAIN** Nitrous oxide causes vasodilatation, resulting in an increase in cerebral blood flow and causing a corresponding increase in intracranial pressure (Erowid). Unlike most inhalants, nitrous oxide does not augment the effects of non-depolarizing neuromuscular blockers.

Accordingly, nitrous oxide does not produce the same neuromuscular depression experienced with other inhalant abuse. Nitrous oxide activates opioid receptors in the periaqueductal grey of the midbrain, stimulating a release of norepinephrine and activation of 2-adrenoceptors in the dorsal horn of the spinal cord (Banks and Hardman). **LONG TERM EFFECTS** Risks involved in nitrous oxide inhalation revolve primarily around displacing oxygen. Although nitrous oxide does not bind with hemoglobin and instead dissolves into the blood, continued inhalation of pure nitrous oxide without supplemental oxygen can lead to hypoxia.

Nitrous oxide-induced hypoxia is especially dangerous because users may not realize that they are asphyxiating themselves – the impulse to breathe is triggered by a build-up of carbon dioxide, rather than a lack of oxygen (Banks and Hardman). Aside from hypoxia, there are relatively few physiologic dangers associated with nitrous oxide abuse. The most significant of these effects is Vitamin B depletion and deficiency. This can potentially lead to acute or chronic paresthesia, the sensation of “pins and needles,” and can inhibit the activity of methionine synthetase, thereby interfering with DNA synthesis in leukocytes and erythrocytes.

However, Vitamin B and Folate supplements can prevent these side effects (Banks and Hardman). Nitrous oxide can also induce potentially dangerous airspace expansion in the body, such as pneumothorax or bowel obstruction, due to its rapid diffusion properties. Other potential side effects include respiratory depression, apnea, hypotension, cardiac arrhythmias, dizziness, neuropathy, nausea, vomiting, ileus, bone marrow depression, and malignant hyperthermia. Pregnant women should not use nitrous oxide, as the gas diffuses into the placenta and can cause fetal depression (Erowid).

A few cases of frostbite on the vocal cords have been recorded following direct inhalation of nitrous oxide from a canister (Banks and Hardman). Also, nitrous oxide users are at risk of traumatic fall injuries such as broken extremities and concussions due to impaired balance and possible loss of consciousness (Narconon). STATISTICS Nitrous oxide abuse is most common among younger adolescents aged 16-17, although abuse does occur among older individuals. A 2011 study from the University of Michigan showed that

13% of 8th grade students reported abusing inhalants at least once (National Institute on Drug Abuse).

A similar study from the University of Virginia revealed that nitrous oxide was one of the top five substances abused by adolescent inhalant users (Narconon). According to the Substance Abuse and Mental Health Services Administration, the rates of inhalant abuse “ increased steadily from 3. 4 percent at age 12 to 5. 3 percent at age 14, then declined to 3. 9 percent by age 17 from 2002-2006. ” Data from this study also showed that “ adolescents age 12 to 17 represented 48 percent of all substance abuse treatment admissions reporting inhalants. Among these adolescent admissions reporting inhalant abuse, 45% had a concurrent psychiatric disorder (SAMHSA). Erowid. com, a popular website that provides information on psychoactive drug use, conducted a series of online surveys in September 2009 in a study examining the possible presence of contaminants in nitrous oxide chargers. One of the surveys asked regular nitrous oxide users their preferred method of inhalation; 46% preferred using a cracker with a balloon, 34. 4% preferred inhaling directly from a whipped cream dispenser, 11. % percent preferred a whipped cream dispenser with a balloon, 1. 7 % preferred a cracker with a bag, 0. 7% preferred a whipped cream dispenser with a bag, and 5. 0% preferred other methods (Erowid). Nitrous oxide is not physiologically addictive, although certain individuals can use it compulsively. While psychological addiction is possible, the only symptom of withdrawal is the desire to inhale more nitrous oxide (Dartmouth College). Furthermore, statistics on inhalant-related deaths are difficult to determine, as most deaths are severely under-reported.

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Most of the time, death is attributed to cardiac or respiratory failure subsequent to inhalant abuse. Studies from the Texas Commission on Alcohol and Drug Abuse between 1990 and 1993 revealed an average of 15 inhalant abuse-related deaths per year in the state of Texas. Of those deaths, 94% of victims were male, and 91% were Caucasian, on average (National Inhalant Prevention Coalition). Most recreational nitrous oxide-related deaths are caused by hypoxia when users affix masks without oxygen or place bags over their heads to concentrate the gas (Erowid).

#### EFFORTS TO ADDRESS ABUSE

The topic of inhalants has been addressed by multiple small grass-roots efforts in local communities, but has also seen national discussion. Although no national legislation is in place restricting the sale, consumption, and distribution of nitrous oxide, several states and municipalities have passed laws in an attempt to curb usage. Most of these laws specifically target underage consumption and sale of nitrous oxide to minors (Center for Cognitive Liberty & Ethics). For example, the Article 34 Title 7 § 3380. 5a-5b of the New York State Controlled Substances Act states: a) No person shall use nitrous oxide for purposes of causing intoxication, inebriation, excitement, stupefaction or the dulling of the brain or nervous system of himself or another. (b) No person shall sell any canister or other container of nitrous oxide unless granted an exemption pursuant to this subdivision. In no event shall any canister or other container of nitrous oxide be sold to a person under the age of twenty-one years. “ Any person who violates any provision of subdivision four or five of this section shall be guilty of class A misdemeanor” (New York State Legislature).

A number of non-governmental organizations such as the National Inhalant Prevention Coalition work to educate the public on the facts and dangers of inhalant abuse. These organizations frequently lobby on the state and federal level in attempt to pass more restrictive anti-inhalant abuse legislature. These preventative efforts have helped to several states integrate inhalant abuse education into primary and secondary school health education curricula (National Inhalant Prevention Coalition). Works Cited Alai, Nili N. " Nitrous Oxide Administration. " Ed. Rick Kulkarni.

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