

Air Quality and Noise During Indoor Motorsports Events

Indoor events featuring motorized vehicles can generate substantial amounts of combustion byproducts. Many buildings used for such events were not originally designed with them in mind (e.g., ice skating rinks, basketball arenas, etc.). If ventilation and air handling in the building is inadequate, levels of these byproduct gases can increase to the point they pose a health hazard. Indoor motorsports events can also be very loud, which can result in permanent hearing damage. In February 2018, Environmental Sanitation Program staff requested the assistance of the Environmental Epidemiology Program, Utah Department of Health, in identifying appropriate action levels for local health departments to protect the health of spectators. The primary pollutants of concern were carbon monoxide (CO) and nitrogen dioxide (NO₂), and the average length of an event was stated to be 90 to 120 minutes.

Carbon Monoxide

Carbon monoxide is a colorless, odorless, tasteless, non-irritating gas found in both indoor and outdoor air. It is formed when a carbon-based fuel, such as gasoline or diesel fuel, is not burned completely (ATSDR, 2012). Carbon monoxide can come from both human-made as well as natural sources. In outdoor air, the most important source of CO is automobile exhaust, and it remains for an average of two months before being converted to carbon dioxide. Natural sources of CO include volcanic activity, wildfires, and releases from vegetation as a metabolic byproduct. Indoor sources of CO are typically man-made and include improperly installed or filtered kerosene and gas space heaters, furnaces, wood stoves, and generators. Exposure to CO may also occur from cigarette smoke, both in smokers and those exposed to second-hand smoke. High levels of CO can result from using gasoline- or propane-powered equipment or vehicles in poorly ventilated indoor spaces (ATSDR, 2012; NAS, 2010).

Exposure to high levels of CO can be life threatening, and CO poisoning is one of the leading causes of death due to poisoning in the U.S. (ATSDR, 2012). Exposure to CO causes adverse health effects by preferentially binding to hemoglobin, the primary oxygen-carrying molecule in the blood. This binding forms a compound called carboxyhemoglobin (COHb). Normal metabolism results in background COHb levels of 0.5-0.8%; at around 4% COHb, people with heart diseases may experience worsened symptoms (NAS, 2010). Increased COHb reduces the ability of blood to carry oxygen and results in insufficient oxygen reaching the tissues, a condition known as hypoxia. Organs with a high oxygen requirement, such as the heart and brain, are especially sensitive to hypoxic conditions. Symptoms of CO exposure include headache, nausea, vomiting, dizziness, blurred vision, confusion, chest pain, weakness, heart failure, difficulty breathing, seizures, and coma. People with heart or lung diseases are more vulnerable to the toxic effects of CO. Most lethal effects are likely due to severe heart rhythm abnormalities and low blood pressure. During pregnancy, breathing high levels of CO can cause miscarriage and other adverse birth outcomes, and breathing lower levels may harm the mental development of the child (ATSDR, 2012; EPA, 2013a; NAS, 2010)

Nitrogen Oxides

Nitrogen oxides are a mixture of gases composed of nitrogen and oxygen and include nitrogen dioxide and nitric oxide. Nitrogen dioxide, a nonflammable, reddish-brown gas with a strong, harsh odor, is often used as an indicator for the larger group of NO_x gases during measurement and assessment (ATSDR, 2002; NAS, 2012). Gases in the NO_x family are major components of

air pollution and contribute to the formation of ozone and particulate matter (EPA, 2013b). Nitrogen oxides are released from motor vehicle exhaust; burning coal, oil, and natural gas; arc welding; electroplating; engraving; and dynamite blasting. Households that burn wood or use kerosene heaters and gas stoves may have higher levels of NO_x indoors. Nitrogen oxides are also found in tobacco smoke, so smokers and people who breathe in second-hand smoke may also be exposed to high levels (ATSDR, 2002; EPA, 2013b).

Nitrogen oxides are irritants to the airways in the respiratory system. Breathing low levels can irritate the eyes, nose, throat, and lungs, leading to coughing, shortness of breath, tiredness, and nausea. Exposure to low levels can also result in fluid build-up in the lungs several days after exposure. Breathing high levels of nitrogen oxides can cause rapid burning, spasms, and swelling of tissues in the throat and upper respiratory tract, reduced oxygenation of body tissues, a build-up of fluid in your lungs, and death. Longer exposures to elevated concentrations of NO_x may contribute to the development of asthma and potentially increase susceptibility to respiratory infections. People with asthma, as well as children and the elderly, are generally at greater risk (ATSDR, 2002; EPA, 2013b; NAS, 2012).

Acute Exposure Guideline Levels

Acute exposure guideline levels (AEGLs) are used to describe human health effects from rare exposures to airborne hazardous substances among the general public. They are developed through a collaborative effort between public and private sectors worldwide, including the Environmental Protection Agency (EPA), the Agency for Toxic Substances and Disease Registry (ATSDR), the National Academies (formerly the National Academy of Science), and numerous universities. Primarily intended as guidance for emergency responders when dealing with chemical spills and other catastrophic events, they can also be applied to other rare airborne exposures. They are designed to protect the elderly, children, and other individuals who may be susceptible (EPA, 2017).

Rather than repeated exposures, AEGLs are calculated for five relatively short periods: 10 minutes, 30 minutes, one hour, four hours, and eight hours. Levels of AEGLs are determined by the possible severity of the toxic effects of exposure, with level 1 being the least and level 3 being the most severe (EPA, 2017):

- Below Level 1: Mild and progressively increasing but transient and non-disabling odor, taste, and sensory irritation or certain asymptomatic, non-sensory effects.
- AEGL Level 1: Notable discomfort, irritation, or certain asymptomatic non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.
- AEGL Level 2: Irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.
- AEGL Level 3: Life-threatening health effects or death.

The AEGLs for carbon monoxide and nitrogen dioxide (NO₂, a proxy for the group of NO_x gases) are shown in **Table 1**, along with the health end points from which they were derived (NAS, 2010; NAS, 2012). For CO, AEGL 1 values were not derived because susceptible persons may experience more serious effects (roughly equivalent to AEGL 2) at concentrations that do

not yet cause AEGL 1 effects in the general population. Children and persons with coronary artery disease were determined to be the populations most susceptible to the effects of CO exposure (NAS, 2010). For NO_x, asthmatics were determined to likely be the most susceptible population, although their response to NO_x exposure is variable (NAS, 2012).

For comparison purposes, the EPA's national 1-hour standards for outdoor air are 35 parts per million (ppm) for CO and 0.1 ppm for NO₂ (EPA, 2016c). Utah's outdoor air is consistently well below those levels (UDOH, 2017a; UDOH, 2017b).

Table 1: AEGLs for carbon monoxide and nitrogen dioxide. Levels are in parts per million (ppm).

		10 min	30 min	60 min	4 hours	8 hours	Health End Point
Carbon Monoxide	AEGL 1	NR	NR	NR	NR	NR	-----
	AEGL 2	420	150	83	33	27	Cardiac effects in persons with coronary artery disease. AEGL 2 level/time combinations would result in 4-12% of hemoglobin bound to CO (COHb).
	AEGL 3	1,700	600	330	150	130	Most lethal poisonings are associated with ≥40% COHb. AEGL 3 level/time combinations would result in 14-23% COHb.
Nitrogen Dioxide	AEGL 1	0.5	0.5	0.5	0.5	0.5	Slight burning of the eyes, headache, chest tightness, labored breathing in some asthmatics.
	AEGL 2	20	15	12	8.2	6.7	Burning of the nose and chest, cough, and labored breathing in healthy adults.
	AEGL 3	34	25	20	14	11	Marked respiratory irritation, pathologic changes to the lungs, fibrosis and swelling of the heart, liver necrosis, but no deaths in monkeys.

NR: Not recommended. Susceptible persons may experience more serious effects (roughly equivalent to AEGL 2) at concentrations that do not yet cause AEGL 1 effects in the general population.

AEGL 1: Notable discomfort, irritation, or certain asymptomatic non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.

AEGL 2: Irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

AEGL 3: Life-threatening health effects or death.

Noise

Noise-induced hearing loss is a permanent condition caused by damage to the nerve cells in the inner ear that worsens with increasing exposure. Both the Occupational Safety and Health

Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH) have set regulations and recommendations for occupational noise exposure, which is measured in decibels (dB). OSHA sets legally enforceable limits in the workplace, called permissible exposure limits (PELs). By contrast, NIOSH sets recommended levels based on the best available science, called recommended exposure limits (RELs). The OSHA PEL and the NIOSH REL specify maximum allowable daily noise doses. The noise dose is based on both the sound level and the duration, so for each increase of 3 dB (NIOSH) or 5 dB (OSHA) in noise levels, the duration of the exposure should be cut in half. Exposures at or above these levels for the specified amount of time are considered hazardous. These levels are shown in **Table 2** (NIOSH, 2016).

Table 2: Occupational noise-related regulations and guidance. Note that NIOSH uses a 3 dB time/intensity exchange rate, while OSHA uses 5 dB rate. When the noise level increases by 3 dB (or 5 dB), the allowed exposure time is cut in half.

Time to reach 100% daily noise dose	Exposure level per NIOSH REL	Exposure level per OSHA PEL
8 hours	85 dBA	90 dBA
4 hours	88 dBA	95 dBA
2 hours	91 dBA	100 dBA
1 hour	94 dBA	105 dBA
30 minutes	97 dBA	110 dBA
15 minutes	100 dBA	115 dBA

dBA: Noise level in decibels weighting the sound frequencies using the ‘A’ standard, since the human ear does not respond to all frequencies equally. More information on this weighting can be found at OSHA, 2008.

For comparison purposes, a normal conversation is approximately 60 dB, hair dryers and lawn mowers are about 90 dB, a bulldozer is about 105 dB, a chainsaw is about 110 dB, and a jet engine at takeoff is about 140 dB (NIOSH, 2018).

Studies of Air Quality at Motorsports Events

Studies have examined the issue of potentially hazardous air quality during indoor motorsports events, although much of the data is several decades old. Lévesque and colleagues monitored levels of CO and NO₂ in a 14,000 seat indoor ice skating rink during three monster truck and car demolition exhibitions. Both time-weighted averages and maximum peak levels of CO and NO₂ were measured. While NO₂ was generally below the limit of detection of 0.5 parts per million (ppm), time-weighted average concentrations of CO ranged between 33 ppm and 100 ppm. Peak levels exceeded 200 ppm between one and ten times per event, and maximum peak levels exceeded 1,200 ppm three times. Potential symptoms of exposure to CO and NO₂ were not monitored, unfortunately, though no linked cases were identified among area medical clinics (Lévesque et al., 2000).

The same research group conducted a similar study during indoor karting races. Levels of CO were measured in the air as well as in the lungs of 10 race participants during a 45 minute race. The average concentration of CO in the air was 41 ppm, while the increase in lung CO averaged 16.2 ppm, which is equivalent to a 3% increase in COHb (Lévesque et al., 2005).

Investigators from NIOSH conducted a health hazard evaluation at monster truck and motocross shows held at a 12,000 seat arena in Cincinnati, Ohio. Personal and area air monitoring were conducted during the roughly 2 hour shows to evaluate exposures to CO and volatile organic compounds. Noise exposures were also assessed. However, personal monitoring was limited to four workers at the event. The monster trucks burned methanol and did not have exhaust suppression systems, while the motocross motorcycles burned a mix of high octane gasoline and racing engine oil and were equipped with exhaust and noise suppression systems. Time-weighted average spectator exposures ranged from 42 ppm to 80 ppm of CO, averaged over one hour. The peak level was 246 ppm. Crowd noise exposures were averaged from 30 minutes before the show until the end of the show. Average exposures ranged from 97 – 100 dB using the NIOSH system and 92 – 95 dB in the OSHA system (NIOSH, 1998).

The Cincinnati Health Department evaluated CO levels during monster truck jumps, tractor pulls, and mud races held at an indoor arena seating 16,000 people. The vehicles involved in the shows used a variety of fuels and had no emission control systems. Most events lasted approximately two to three hours, and the arena's ventilation system was operated maximally and a commercial truck entrance was kept open. Average CO levels ranged from 79 – 140 ppm, with peak levels ranging from 140 – 283 ppm. As the ventilation system was clearly inadequate, the time between each run during the events was increased. However, this was ineffective in lowering CO levels (MMWR, 1994).

Report prepared by:

Nathan LaCross, PhD, MPH
Epidemiologist
Environmental Epidemiology Program
Bureau of Epidemiology
Utah Department of Health
nlacross@utah.gov

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