Health Consultation

CARBON MONOXIDE POISONING AT AN INDOOR MOTOCROSS FACILITY
ST. CLAIR COUNTY, MICHIGAN

Prepared by the
Michigan Department of Community Health

JUNE 11, 2009

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

You May Contact ATSDR Toll Free at
1-800-CDC-INFO
or
Visit our Home Page at: http://www.atsdr.cdc.gov

HEALTH CONSULTATION

CARBON MONOXIDE POISONING AT AN INDOOR MOTOCROSS FACILITY
ST. CLAIR COUNTY, MICHIGAN

Prepared By:

Michigan Department of Community Health
Under a Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

Table of Contents

Table of Contents i
List of Tables i
List of Appendices i
Acronyms and Abbreviations ii
Summary 3
Purpose and Health Issues 3
Background 3
Discussion 4
Comparison Levels for Carbon Monoxide 4
Carbon Monoxide Levels Measured at Facility 6
Estimated Carbon Monoxide Levels 7
Toxicological Evaluation 8
Children's Health Considerations 9
Other Populations of Concern 10
Community Health Concerns 10
Conclusions 10
Recommendations 11
Public Health Action Plan 11
Preparers of Report 12
References 13
Certification 15

List of Tables

Table 1. Comparison values for carbon monoxide in indoor air 6

Table 2. Maximum carbon monoxide levels recorded on January 9, 2009 at an indoor motocross facility in St. Clair County, Michigan. (Values in parts per million [ppm].)……..7

List of Appendices

Appendix A. Mathematical Model for Estimating Carboxyhemoglobin and Exposure Concentrations A-1

Appendix B. ATSDR Public Health Hazard Categories……...…….B-1

Acronyms and Abbreviations

$\Delta COHb_t$	change in ml of CO per ml of blood at time t
Δt	exposure duration
θ	time between end of exposure and blood sampling (theta)
ACGIH	American Conference of Governmental Industrial Hygienists
AEGL	Acute Exposure Guideline Level
AQD	Air Quality Division
ATSDR	Agency for Toxic Substances and Disease Registry
B	activity-level factor
C	percent COHb in blood
CO	carbon monoxide
COHb	carboxyhemoglobin
$COHb_{t-1}$	ml of CO per ml of blood at the previous measurement
D	background COHb
D_L	diffusivity of the lung for CO
EPA	U.S. Environmental Protection Agency
f	respiration rate
Hg	mercury
K	activity-level factor
kg	kilograms
M	ratio of affinity of blood for CO to that for oxygen
MDCH	Michigan Department of Community Health
MDEQ	Michigan Department of Environmental Quality
MIOSHA	Michigan Occupational Safety and Health Administration
min	minutes
ml	milliliters
mm	millimeters
NAAQS	National Ambient Air Quality Standard
OHb_{max}	ml of oxygen per ml bood under normal conditions
OSHA	Occupational Safety and Health Administration
P_{CO}	partial pressure of CO in inhaled air
PEL	Permissible Exposure Limit
P_L	barometric pressure minus the vapor pressure of water at body temperature
P_{O2}	average partial pressure of oxygen in the lung capillaries
ppm	parts per million
t	exposure duration
T	recovery time
TLV	Threshold Limit Value
TWA	time-weighted average
V_A	alveolar ventilation rate
V_b	blood volume
V_{CO}	rate of endogenous CO production
V_D	anatomic dead space
V_E	total rate of ventilation

Summary

Three boys, ages seven to 10, were diagnosed with nonfatal carbon monoxide (CO) poisoning following seven hours of participating in indoor motocross events. Indoor air testing, done after the incident, suggests that CO levels inside the facility could have reached potentially harmful levels, especially to susceptible populations. As shown by health outcome data, conditions at the facility posed an urgent public health hazard to the affected children. Due to mounting controversy, the owner has closed the facility.

Owners and operators of indoor motorized sport arenas should evaluate adequacy of ventilation inside their structures, establish public-health-protective action levels for CO, and inform workers at, and users of, the facility about the hazards of exposure to CO.

Purpose and Health Issues

The purpose of this health consultation is to discuss public health issues that may result from being a participant in, or spectator of, indoor motorized sporting events and to make health recommendations to protect persons involved in these activities. The specific case illustrating this discussion concerns the non-fatal poisoning of several children by carbon monoxide (CO). The children had been participating in motocross events at an indoor track that was formerly a golf dome. The local health department and a state legislator asked the Michigan Department of Community Health (MDCH) for a public health opinion regarding the case.

MDCH conducted this health consultation for the federal Agency for Toxic Substances and Disease Registry (ATSDR) under a cooperative agreement. ATSDR conducts public health activities (assessments/consultations, advisories, education) at sites of environmental contamination and concern. ATSDR is primarily an advisory agency. Therefore, its reports usually identify what actions are appropriate to be undertaken by the regulatory agency overseeing the site, other responsible parties, or the research or education divisions of ATSDR. As such, ATSDR recommendations may not encompass all types of federal and state requirements from a regulatory perspective. Thus, the purpose of a health consultation is not to evaluate or confirm regulatory compliance but to determine if any potentially harmful exposures are occurring or may occur in the future.

Background

On December 30, 2008, the Michigan Department of Environmental Quality (MDEQ) Air Quality Division (AQD) Toxics Unit referred the St. Clair County Health Department to MDCH regarding a CO poisoning event. Three boys, ranging in age from seven to 10 years, had participated in indoor motocross events several days previously. The boys had been in the sports facility for about seven continuous hours. At least two of the boys had been riding motorcycles on the indoor track; the third may have only been an observer. During the car ride home from the facility, the boys complained of light-headedness and nausea. One of them vomited (R. Elmouchi, MDEQ AQD, personal communication, 2009). The adult driver brought the boys to the emergency room, where their carboxyhemoglobin (COHb) was checked. COHb levels ranged from 9.4 to 13.6% (St. Clair County Health Department case file, unpublished data,

2009). (Baseline COHb levels in non-smokers are less than or equal to 0.8% [EPA 2005].) The boys received supplemental oxygen, as well as ibuprofen for their headaches. Only one boy's blood was reportedly redrawn, six hours later. The COHb level had fallen to 1.2%. The boys had arrived at the emergency room at 10 PM and were discharged by 4 AM the following morning (St. Clair County Health Department case file, unpublished data, 2009). No further health symptoms have been reported to MDCH.

The local health department staff person indicated that he had spoken with AQD staff from the MDEQ district office. MDEQ regulates ambient (outdoor) air in Michigan but has no authority regarding indoor air (except in cases of vapor intrusion from the soil). The Michigan Occupational Safety and Health Administration (MIOSHA) regulates indoor air in occupational settings. There are no indoor air standards in Michigan for non-occupational settings, such as private homes or recreational facilities. MDCH contacted MDEQ and learned that the state senator representing the St. Clair County area had contacted MDEQ (and subsequently contacted MDCH) (R. Elmouchi, MDEQ AQD, personal communication, 2009).

MDCH contacted the building supervisor for the township where the facility is located, seeking additional information. According to the building supervisor, the owner of the facility, upon hearing about the children's poisoning, hired a consultant and asked the MIOSHA Consultation, Education, and Training Division to assist in determining how best to correct conditions at the track (MDCH case file, 2009). MIOSHA has industrial hygienists on staff and could assess the situation from the standpoint of safety to the track employees. Because the MIOSHA investigation was not initiated by a complaint but rather by a request, the data collected by that agency was considered Confidential Business Information and could not be shared without the facility owner's consent (C. Passamani, MIOSHA, personal communication, 2009). However, the owner did release to the township the data gathered by his private consultant. The township shared these data with MDCH.

Discussion

Comparison Levels for Carbon Monoxide

Currently, there are no non-occupational indoor air standards for CO in Michigan. MIOSHA relies on the 1989 Permissible Exposure Limit (PEL) set by the Occupational Safety and Health Administration (OSHA) which, for CO, is 35 parts per million (ppm; MIOSHA 2002; R. Dayringer, MIOSHA, personal communication, 2009). In 1989, OSHA reduced the CO PEL from 50 ppm to 35 ppm, but the new value was vacated due to legalities and issues with economic and technical feasibility. OSHA feels that the 50-ppm value is not sufficiently protective of workers (OSHA 1999). Although the federal agency can no longer use the 1989 value, MIOSHA was not part of the lawsuit and currently uses 35 ppm as a time-weighted-average (TWA) exposure limit (R. Dayringer, MIOSHA, personal communication, 2009).

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a Threshold Limit Value (TLV) for CO of 25 ppm. This value is intended to maintain blood COHb levels below 3.5%, to minimize the potential for adverse neurobehavioral changes, and to maintain cardiovascular capacity for work and exercise (ACGIH 2001). TLVs are health-based values designed for use by industrial hygienists in making decisions regarding safe levels of

exposure to various chemicals found in the workplace. TLVs are not regulatory standards (ACGIH 2008).

The U.S. Environmental Protection Agency (EPA) Acute Exposure Guideline Levels (AEGLs) are developed by the Advisory Committee for Acute Exposure Guideline Levels of Hazardous Substances. The primary use of AEGLs is to assist organizations with emergency planning, response, and prevention programs. Although the AEGL values represent threshold levels for the general public (including susceptible subpopulations such as infants, children, the elderly, persons with asthma, and those with other illnesses), EPA recognizes that some individuals could experience the effects described at concentrations below the corresponding AEGL (EPA 2008a).

Normally, there are three levels of AEGLs (EPA 2008a):

- AEGL-1 is the concentration of a chemical in the air above which the general population, including susceptible individuals, could experience notable discomfort, irritation, or other disagreeable effects. The effects are not disabling and are transient and reversible upon cessation of exposure. Concentrations below the AEGL-1 may produce mild effects but, again, these would be temporary and cease after removal from exposure.
- AEGL-2 is the concentration of a chemical in the air above which the general population, including susceptible individuals, could experience irreversible or serious, long-lasting health effects or an impaired ability to escape.
- AEGL-3 is the concentration of a chemical in the air above which the general population, including susceptible individuals, could experience life-threatening health effects or death.

An AEGL may be averaged over 10, 30, or 60 minutes, or 4 or 8 hours. As defined by EPA's AEGL Program, "acute" exposure lasts no longer than 8 hours (EPA 2008a). Other agencies may define "acute" exposure differently, such as ATSDR defining "acute" exposure as lasting up to two weeks (ATSDR 2009). Within each AEGL level, the 8-hour value would be the lowest or most protective number.

There is no AEGL-1 level established for CO. This is because susceptible persons may experience effects more serious than described above for that level (EPA 2005). See further discussion under the *Toxicological Evaluation* section of this document.

The 8-hour value for the AEGL-2 for CO is 27 ppm and the 4-hour value is 33 ppm. These values should prevent COHb levels in patients with coronary artery disease from exceeding 4% and protect against the onset of angina (chest pain) during physical exertion (EPA 2005).

Table 1 displays the comparison values discussed here, starting with the most protective (lowest) value. The EPA National Ambient Air Quality Standard (NAAQS) for CO of 9 ppm was not included for consideration because, as indicated by its name, the standard addresses only ambient (outside) air.

Table 1. Comparison values for carbon monoxide in indoor air.

Agency	Comparison Value	Concentration (ppm)	Primary Use
ACGIH	TLV	25	Non-regulatory, worker level
EPA	AEGL-2, 8-hour	27	Non-regulatory, emergency planning
EPA	AEGL-2, 4-hour	33	Non-regulatory, emergency planning
MIOSHA	TWA Exposure Limit	35	Regulatory worker level in Michigan (1989 OSHA PEL)
OSHA	Current PEL	50	Current federal value but may not be protective

References: ACGIH 2001, EPA 2005, MIOSHA 2002, OSHA 1999

Acronyms:

ACGIH	American Conference of Governmental Industrial Hygienists
AEGL	Acute Exposure Guideline Level
EPA	U.S. Environmental Protection Agency
MIOSHA	Michigan Occupational Safety and Health Administration
OSHA	Occupational Safety and Health Administration
PEL	Permissible Exposure Limit
ppm	parts per million
TLV	Threshold Limit Value
TWA	Time-weighted average

Carbon Monoxide Levels Measured at Facility

It is not known whether CO levels at the motocross facility were measured prior to this poisoning incident.

The facility owner's consultant set up seven locations inside the structure to monitor CO levels: four locations were near the perimeter of the motocross track, one was on the track used for remote-control cars, one was in the mezzanine (where spectators would sit), and one was in the lobby (ground level). MDCH received data collected during a seven-hour period on January 9, 2009. A maximum of seven bikes were on the track during the monitoring period. No bikes were on the track for the first and last hours of the monitoring period (MDCH case file, unpublished data, 2009). It is not known if ventilation of the facility was occurring at normal levels or if it had been adjusted as part of the sampling protocol. Table 2 shows the maximum concentrations recorded within each area.

Table 2. Maximum carbon monoxide levels recorded on January 9, 2009 at an indoor motocross facility in St. Clair County, Michigan. (Values in parts per million [ppm].)

Location	Maximum Concentration
Motocross track[1]	60
Remote-control track	43
Mezzanine	19
Lobby	31

Reference: unpublished data

Notes:

1. There were four separate monitoring locations along the perimeter of the motocross track.

The maximum concentrations occurred when the maximum number of bikes was on the track, except for the mezzanine location. Values recorded on the mezzanine showed a steady climb in the CO levels, which continued to increase during the last hour, when no bikes were on the track. This suggests a lack of adequate ventilation in this area.

Using the data from all the monitoring locations in this sampling event, the consultant calculated an average of 19.35 ppm CO over the seven hours (MDCH case file, unpublished data, 2009). When compared to the comparison values in Table 1, this might suggest that the CO levels pose no risk. However, this may not be an appropriate conclusion to make because it assumes people are present for the entire seven hours and that there is adequate ventilation in all areas. As an example, some locations along the motocross track and the remote-control track showed readings above the 4-hour AEGL-2 (33 ppm) for about three hours (data not shown). The averaging of the CO concentrations ignores the issue of short-term exposure to high concentrations that could lead to adverse health effects. Also, using an average implies that people will move randomly around the facility and not use just one area.

Area monitoring, such as was done at the facility, may not correctly represent exposure to an individual. Personal monitoring would provide more accurate information regarding exposure levels.

Estimated Carbon Monoxide Levels

The technical support document for the CO AEGLs discusses a mathematical model that can be used to back-calculate an average CO air concentration from a COHb concentration (EPA 2005). This model, and its use for this health consultation, is discussed in detail in Appendix A. Note that, as a model, the equation can only be used for purposes of estimating and cannot calculate with certainty the actual exposure experienced by the children.

Briefly, MDCH used most of the default values provided by the model but used child-specific values for the volume of blood in the body, body weight, respiration rate, total rate of ventilation, and dead space (a ventilation parameter). MDCH used the COHb value from the child who vomited (vomiting is considered a more severe symptom than headaches and nausea), which was also the highest COHb value among the three children treated. The resulting estimated CO air concentration was 17 ppm. Note that this is an *average* value, for the seven hours that the children were reportedly at the facility. If the children had arrived just after the facility opened for the day, they would not have initially been exposed. Therefore, they would have been

exposed to concentrations higher than 17 ppm to attain the average. Table 2 gives some indication of what the upper levels of exposure could have been.

Another confounding variable when estimating the CO concentration is that the COHb levels were not measured immediately after exposure stopped. It is possible that 30 to 60 minutes, if not more, elapsed before the children were seen at the emergency room. During this time, the COHb levels would be decreasing. The time required to eliminate half of the CO gas from the body (half-life) is three to five hours (EPA 2005). The COHb level in the children was likely higher when they left the facility, which would cause the estimated CO air concentration to increase. The elimination half-life for CO is discussed further in the *Toxicological Evaluation* section below.

Also, it is not known whether these children are regularly exposed to cigarette smoke. Smokers have increased background COHb levels. It is likely that children living with smokers also have higher background COHb levels, although perhaps not to the degree seen in smokers. Background COHb levels are discussed further in the *Toxicological Evaluation* section below.

Toxicological Evaluation

CO is a tasteless, odorless and colorless gas. It is produced by both natural and man-made processes, the main source being incomplete combustion of fuels. Environmental exposures may occur while traveling in motor vehicles, working, smoking, visiting urban locations associated with combustion sources, or cooking and heating with domestic gas, charcoal or wood fires. Ambient CO concentrations generally are below 9 ppm (when averaged over 8 hours), but may have short-term peaks up to 50 ppm on heavily traveled roads. Concentrations inside motor vehicles are generally around 9-25 ppm and occasionally over 35 ppm. The levels in homes are usually lower than 9 ppm but may peak at 18 ppm with gas stoves, 30 ppm with wood combustion, and 7 ppm with kerosene heaters (EPA 2005).

When CO is inhaled and absorbed by the lungs, it combines with hemoglobin to form COHb, which causes the hemoglobin molecule to become less able to bind with oxygen. Although the rate of CO binding with hemoglobin is about one-fifth slower and the rate of dissociation (separating) from hemoglobin is an order of magnitude slower than the respective rates for oxygen, CO's chemical affinity for hemoglobin is about 245 times greater than that of oxygen, thus making it bind very tightly. The elimination half-life of CO in the body is 3 to 5 hours when breathing room air. Increased oxygen pressure helps to dislodge the CO molecule from the hemoglobin. One hundred percent oxygen at atmospheric pressure reduces the half-life to about 80 minutes (or to as little as 44 minutes in children), whereas hyperbaric (greater than atmospheric pressure) oxygen reduces the half-life to about 20 to 25 minutes (EPA 2005).

Adults with coronary artery disease and unborn fetuses are more susceptible than healthy adults to *lethal* effects of carbon monoxide. For *nonlethal* effects, subjects with coronary artery disease and children constitute susceptible subpopulations. Exposure can cause earlier onset of angina and changes in the electrocardiogram and increased frequency of arrhythmias in heart patients. In children, it can cause syncopes (fainting) and long-lasting neurotoxic effects (EPA 2005).

During CO exposure, the partial pressure of oxygen in the body's tissues is lowered, which can result in hypoxia (insufficient oxygen). Although the brain has a higher requirement for oxygen than the heart, the coronary (heart) circulation, in contrast to the cerebral (brain) circulation, must supply an even greater amount of oxygen during periods of generalized tissue hypoxia. Under these circumstances, the heart is forced to increase both its rate and its output to meet the oxygen demands of the body. Exposed heart patients, with diminished coronary circulation caused by their condition, would near the point of myocardial (heart muscle) tissue hypoxia, which can ultimately lead to myocardial infarction (heart attack; EPA 2005).

Case reports on stillbirths after CO poisoning of pregnant women indicated measured maternal COHb of about 22-25% or higher. Observed nonlethal fetal outcomes include preterm births and cerebral palsy. Fetal hemoglobin has a higher CO affinity and slower CO elimination than adult hemoglobin (EPA 2005), and since the fetus exists in a low-oxygen state, there is no room for the oxygen level to fall. The fetus is unable to increase cardiac output in response to increased CO exposure. (ACGIH 2001).

Acute signs of CO poisoning in children, such as headache, nausea, vomiting, and lethargy, have been reported at COHb levels as low as 7%. (Healthy adults may not experience these symptoms until COHb levels reach 20-30%.) Follow-up studies of CO-poisoned children suggest that long-lasting (up to 11 years post-exposure) neurotoxic effects occurred when the children's COHb level exceeded 20%. These effects included chronic headache; amplified negative behavior (increased nervousness, irritability or anxiety); difficulty with spelling, arithmetic, and speech; difficulty concentrating; some incoordination; and spatial organization problems. Not all children suffered from these long-term effects, and some symptoms resolved over time (EPA 2005).

Humans produce small amounts of carbon monoxide endogenously (within their bodies), leading to a normal background COHb level of about 0.5 to 0.8%. Smokers are exposed to higher CO levels than are non-smokers and have an average background COHb level of 4% (range of 3 to 8%; EPA 2005). Children living with smokers likely have an elevated background COHb level.

Children's Health Considerations

Children may be at greater risk than adults from exposure to hazardous substances at sites of environmental contamination. Children engage in activities such as playing outdoors and hand-to-mouth behaviors that could increase their intake of hazardous substances. They are shorter than most adults, and therefore breathe dust, soil, and vapors found closer to the ground. Their lower body weight and higher breathing and intake rates result in a greater dose of hazardous substance per unit of body weight. The developing body systems of children are immature and more at risk of permanent damage if toxic exposures occur during critical postnatal growth stages. Injury during key periods of prenatal growth and development could lead to malformation of organs (teratogenesis), disruption of function, and premature death. Exposure of the mother could lead to exposure of the fetus, via the placenta, or affect the fetus because of injury or illness sustained by the mother (ATSDR 1998). Thus, children can experience substantially greater exposures to toxicants in soil, water, or air than adults can.

Children, along with adults, use this indoor motocross track. As discussed in the *Toxicological Evaluation*, children may experience symptoms of CO poisoning at lower COHb levels than adults. Children generally have a higher respiration rate than adults, so they would inhale more CO over time, resulting in higher COHb levels sooner. Children represent a population of special concern at this site.

Pregnant women may not be participating in motocross events at this site but may be present as spectators, remote-control car racers, or employees. While their COHb levels should not increase to the point where a stillbirth could occur, nonetheless they may be putting their unborn child at risk for negative health effects. Therefore, unborn fetuses also represent a population of special concern at this site.

Other Populations of Concern

Although people with coronary heart disease would not likely participate in motocross activities, they may be present at this facility as spectators or as remote-control car racers. Increased COHb levels due to CO exposure may put these people at risk of cardiovascular effects, especially if they are exerting themselves, such as climbing the stairs to the mezzanine.

Customers of the motocross facility may use the track sporadically, allowing recovery between subsequent exposures. Employees of the facility, however, would have repeated exposures and may not return to their baseline COHb levels in between. This would lead to an increasingly narrow margin between no apparent effects and symptoms being expressed. Although MIOSHA will be addressing employee safety and health in their evaluation, this concern is mentioned here for completeness.

Community Health Concerns

MDCH received one phone call regarding this site from a local citizen. The caller stated that she had heard about the CO poisoning incident and was concerned about continued use of the facility.

Conclusions

ATSDR public health hazard categories are described in Appendix B.

Conditions at this indoor motocross facility posed an urgent public health hazard in the past. Three children who breathed the air while using the track for seven continuous hours showed symptoms of CO poisoning shortly after leaving the track. Poisoning was confirmed by COHb testing at an emergency room. Longer exposure or exposure to higher levels of CO may have resulted in more severe, and possibly lasting, effects.

Current and future conditions at this facility pose no public health hazard. According to the facility's website, the track was closed permanently on or about February 24, 2009.

Conditions at other indoor motorized sport arenas pose an indeterminate public health hazard. To determine whether or not indoor air conditions at similar facilities pose a risk to people, one should consider:

- ►the adequacy of ventilation,

►the types of engines in use (which would affect rate of emissions),
►duration of engine use (full-day use by motorized go-carts or motorbikes versus half-day use by a"monster" truck rally versus intermittent use by a zamboni [ice-grooming] machine for an ice rink),
►the duration of exposure, and
►the health of the individuals in the facility during motorized events.

It is possible that a CO level of 35 ppm would be not adequately protective of susceptible populations, especially if those persons spend a long time at the facility or are in an area of the facility with insufficient air flow.

Recommendations

1. There are no recommendations for the facility used in this case study, since it is now closed.
2. For other indoor motorized sport arenas, evaluate airflow and monitor CO levels during occupancy of the facility. Stop motorized activities and ventilate the facility if the indoor air concentration of CO exceeds 27 ppm. (Although this is the AEGL-2 value that is applied for an 8-hour exposure period and not all occupants may be present for 8 hours, athletes may be breathing at an increased rate.) It may be prudent to evacuate the facility. If so, reoccupancy should occur only when air monitors read 0 ppm. Otherwise, CO levels would build back up too soon and corrective action would have to be repeated.
3. Inform workers at and users of these facilities about the dangers of CO exposure. Emphasize that heart patients, unborn fetuses, and children are most at risk. If deemed necessary and appropriate by MIOSHA, provide personal badge-type monitors to employees.

Public Health Action Plan

MDCH is willing to work with facilities in crafting language for educational materials so that facility workers and users understand the potential health risks of CO.

If any citizen has additional information or health concerns regarding this public health consultation, please contact the MDCH Division of Environmental and Occupational Epidemiology at 1-800-648-6942. ATSDR and MDCH remain available for further consultation on this site.

Preparers of Report

Michigan Department of Community Health
Division of Environmental Health

Christina Bush, Toxicologist
Toxicology and Response Section

ATSDR Region 5 Office

Mark Johnson
Office of Regional Operations

ATSDR Division of Health Assessment and Consultation

Trent LeCoultre, Technical Project Officer
Cooperative Agreement Program Evaluation Branch

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1998. ATSDR, Division of Health Assessment and Consultation. Guidance on Including Child Health Issues in Division of Health Assessment and Consultation Documents. July 2, 1998.

Agency for Toxic Substances and Disease Registry (ATSDR). 2009. Minimal Risk Levels (MRLs) for Hazardous Substances. http://www.atsdr.cdc.gov/mrls/. Last updated January 14, 2009. Accessed February 20, 2009.

American Conference of Governmental Industrial Hygienists (ACGIH). 2001. Carbon Monoxide: TLV® Chemical Substances 7th Edition Documentation. Publication #7DOC-109.

American Conference of Governmental Industrial Hygienists (ACGIH). 2008. TLV®/BEI® Resources. http://www.acgih.org/tlv/. Last updated January 30, 2008. Accessed February 20, 2009.

Lauwerys RR, Hoet P. Biological monitoring of exposure to organic substances. In: Industrial chemical exposure: guidelines for biological monitoring. 3rd ed. CRC Press (www.crcpress.com); 2001. p. 474-477.

Michigan Occupational Safety and Health Administration (MIOSHA). Part 301 - Air Contaminants. Lansing, MI: Department of Consumer and Industry Services, Director's Office, Occupational Health Standards; 2002 Jan. BSR-STD-1224. http://www.michigan.gov/documents/CIS_WSH_part301_35589_7.pdf. Accessed February 20, 2009.

Numa AH, Newth CJ. 1996. Anatomic dead space in infants and children. J Appl Physiol 80:1485-1489.

Occupational Safety and Health Administration (OSHA). Permissible Exposure Limits (PELs) for air contaminants. Washington, DC: Department of Labor, Unified Agenda, Proposed Rule Stage, 1218-AB54-2228. 1999 Jan. http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=4536&p_table=UNIFIED_AGENDA. Accessed February 13, 2009.

Occupational Safety and Health Administration (OSHA). Occupational safety and health guideline for carbon monoxide. Washington, DC: Department of Labor. http://www.osha.gov/SLTC/healthguidelines/carbonmonoxide/recognition.html. Accessed February 20, 2009 (last update not indicated).

U.S. Environmental Protection Agency (EPA). 2005. Interim Acute Exposure Guideline Levels (AEGLs): Carbon Monoxide (CAS Reg. No. 630-08-0). Developed for the NAS/COT Subcommittee for AEGLs. http://www.epa.gov/oppt/aegl/pubs/tsd306.pdf. Last updated February 2005. Accessed January 13, 2009.

U.S. Environmental Protection Agency (EPA). 2008a. AEGL Program. http://www.epa.gov/oppt/aegl. Last updated December 11, 2008. Accessed February 20, 2009.

U.S. Environmental Protection Agency (EPA). 2008b. Child-Specific Exposure Factors Handbook. Washington, DC: National Center for Environmental Assessment, Office of Research and Development; 2008 Sept. EPA/600/R-06/096F. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=199243

Certification

This Health Consultation was prepared by the Michigan Department of Community Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures. Editorial review was completed by the cooperative agreement partner.

Technical Project Officer, Cooperative Agreement Program Evaluation Branch (CAPEB), Division of Health Assessment and Consultation (DHAC), ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.

for Greg Ulirsch

Team Leader, CAPEB, DHAC, ATSDR

Appendix A. Mathematical Model for Estimating Carboxyhemoglobin and Exposure Concentrations

In conferring with MIOSHA staff and an occupational health physician about this carbon monoxide (CO) poisoning case, MDCH obtained equations that these persons used for back-calculating CO concentrations in the air based on carboxyhemoglobin (COHb) in the blood.

MIOSHA Equation
MIOSHA, using an Excel® spreadsheet, employs a variant of a mathematical model developed by Coburn, Forster and Kane (called the CFK model; C. Passamani, MIOSHA, personal communication, 2009):

$$CO = \frac{1{,}316[(0.0046)(2\text{^}[\theta / T])C - (0.007)B + (0.007B - 2.3D)e\text{^}[-2.3t / 5{,}500B]}{1 - e\text{^}[-2.3t / 5{,}500B]}$$

CO is the concentration of CO in parts per million (ppm).

θ (Theta) is the time between the end of CO exposure and the blood sample being taken, in minutes (min). For this exercise, MDCH assumed the children's blood was tested one hour (60 min) after leaving the motocross facility.

T is a recovery-time factor that considers what corrective measures have been taken to lower the COHb levels. It considers whether the victim breathed only air following exposure, received 100% or hyperbaric oxygen, or if there was a combination of measures taken. For this exercise, MDCH assumed that, after the seven-hour exposure, the children breathed regular air for one hour, then received 100% oxygen for two hours. This resulted in a total weighted recovery time of 160 min.

C is the blood COHb, in %, at the time of the blood samples. For this exercise, MDCH used the highest COHb measured, 13.6%.

B is a numerical activity factor applied to rest, light, or heavy physical work. For this exercise, MDCH assumed that participation in motocross activities qualifies as light physical work. The numerical factor applied for light work is 0.065.

D is the background COHb level. MDCH assumed that the children came from non-smoking families, making the value for D equal to 0.0015 (per MIOSHA's spreadsheet).

t is the time of exposure to CO in minutes. For this exercise, it is assumed that the amount of time spent at the facility, 420 min (7 hours), equals the exposure time.

Inserting the values above into the MIOSHA spreadsheet results in an average CO concentration of 114 ppm.

Lauwerys and Hoet (2001) Equation
An occupational health physician suggested a simpler equation, first hypothesized by Lauwerys and Hoet (2001):

$$CO = \frac{10{,}000\,XCOHb}{tXK}$$

CO is the concentration of CO in ppm.

COHb is the percent COHb. For this exercise, MDCH used the highest COHb measured, 13.6%.

t is the time of exposure to CO in minutes. For this exercise, it is assumed that the amount of time spent at the facility, 420 min (7 hours), equals the exposure time.

K is an activity constant for rest, light, or heavy physical work. For this exercise, MDCH assumed that participation in motocross activities qualifies as light physical work. The numerical factor applied in this equation is 5.

Inserting the values above into the Lauwerys and Hoet equation results in an average CO concentration of about 65 ppm.

Modified CFK Equation Used by EPA AEGL Program
The previous two exercises are more useful when applied to occupational, and therefore adult, settings, not to children. Children are smaller and breathe more air than adults. Based on anatomic and physiological differences, they may be more susceptible to toxic injury than adults. MDCH selected an equation that would allow substitution of child-specific parameters.

The technical support document for the CO Acute Exposure Guideline Levels (AEGLs) discusses another variant of the CFK model. To solve for an air concentration of CO based on COHb, the equation is as follows:

$$P_{CO} = [\frac{\Delta(COHb)_t}{\Delta t} - \frac{V_{CO}}{V_b} + \frac{COHb_{t-1} \times P_{O_2}}{M \times B \times V_b(OHb_{max} - COHb_{t-1})}] \times B \times V_b$$

P_{CO} is the partial pressure of CO in the air inhaled. In the equation, the P_{CO} result is in millimeters mercury (mm Hg). That value is then multiplied by 1,316 to convert to ppm.

$\Delta COHb_t$ is the milliliters (ml) of CO per ml of blood at time t (min). To convert %COHb to ΔCOHb, subtract the background %COHb from the reported blood %COHb level, multiply by 100, and divide by OHb_{max} (discussed below). This exercise uses the 13.6% COHb reported for one of the children and assumes a background COHb of 0.8%. The resulting ΔCOHbt is 0.0272.

Δt is the exposure duration. For this exercise, it is assumed that the amount of time spent at the facility, 420 min (7 hours), equals the exposure time.

V_{CO} is the rate of endogenous CO production. The default value for the equation is 0.007 ml/min.

V_b is the blood volume. The support document presented V_b for a 70-kilogram (kg) man of 5,500 ml, which is about 79 ml/kg, and V_b for a 20-kg five-year-old of 1,500 ml, which is about 75 ml/kg. For this exercise, the average proportion, 77 ml/kg, was used.

$COHb_{t-1}$ is the ml of CO per ml of blood at an earlier time, in this case before the children were exposed at the track. This would be the background level. For this exercise, 0.8% was assumed to be background and converted by multiplying by 100, then dividing by OHb_{max} (discussed below). This value results in 0.0016 ml CO/ml blood.

P_{O2} is the average partial pressure of oxygen in the lung capillaries. The default value for the equation is 100 mm Hg.

M is the ratio of affinity of blood for CO to that for oxygen. The default value for the equation is 218.

B consists of several factors and is derived using the following equation:

$$B = \frac{1}{D_L} + \frac{P_L}{V_A}$$

D_L is the diffusivity of the lung for CO. The default value for the equation is 30 ml/min mm Hg.

P_L is the barometric pressure minus the vapor pressure of water at body temperature. The default value for the equation is 713 mm Hg.

V_A is the alveolar ventilation rate, consisting of several factors, and is derived using the following equation:

$$V_A = V_E - f \times V_D$$

V_E is the total rate of ventilation. MDCH consulted the EPA's Child-Specific Exposure Factors Handbook (EPA 2008b; Table 6-13 in the handbook) and selected the 50th percent Upper Confidence Limit of the mean for male children engaged in medium-level activity. This value is 21,600 ml/min.

f is the respiration rate. MDCH consulted the Child-Specific Exposure Factors Handbook (EPA 2008b; Table 6-40) and selected the average for children ages 6-12, "waking" (versus "sleeping"). This value is 39.1 respirations per minute.

V_D is the anatomic dead space. An article out of the Pediatric Critical Care Division of the Los Angeles Children's Hospital discusses anatomic dead space in children (Numa and Newth 1996). The authors hypothesize that mean extrathoracic anatomic dead space is age-related and equals

0.8 ml/kg in children over 6 years. They argue that mean intrathoracic anatomic dead space is not age-related and equals 0.8 ml/kg. According to the Child Specific Exposure Factors Handbook (EPA 2008b; Table 8-4), the average weight for a 6- to 11-year-old male child is 29.6. Thus, V_D for this exercise is 54.168 ml.

Using the values above for the V_A equation, V_A equals 19,482.03 ml/min. Inputting this and the other values above into the B equation results in B equaling 0.069931 (unitless).

OHb_{max} is the ml of oxygen per ml blood under normal conditions. The default value for the equation is 0.2.

Inserting the above values into the equation for P_{CO} results in a value of 0.012925 mm Hg. Multiplying this value by 1,316 results in an average CO concentration of 17.00907 (17) ppm.

There are several considerations to bear in mind regarding this calculation:

1. Almost all the values used for this exercise are assumptions, which are based on various databases (discussed at length in the Child-Specific Exposure Factors Handbook). Even the exposure duration of 420 minutes (7 hours) assumes that the children were exposed to some concentration of CO for the entire duration. It is possible that the children arrived at the facility right at opening, before any bikes were running, so initial exposure, out of the 420 minutes, may have been 0 ppm. A shorter actual exposure time would result in a higher estimated CO air concentration.

2. The COHb levels measured at the emergency room (13.6% in this exercise) do not reflect the maximum COHb levels, which most likely would have occurred at the point the exposure ended. It is not known how much time passed between the children leaving the facility and being tested at the hospital, so the maximum COHb level cannot be estimated with any certainty.

3. The 17-ppm value is the *average* value over the exposure duration (420 minutes). Based on data collected by the owner's consultant, CO levels at the facility depend on, among other factors, number of bikes on the track and physical location within the facility (see Table 2 in the main body of this document). It is likely that the children were exposed to a range of concentrations, not 17 ppm consistently.

4. It is not known whether the children live with smokers and are regularly exposed to secondhand smoke. This would increase their background COHb levels (represented by $COHb_{t-1}$ in the equation). If this were the case, the estimated CO air concentration, to raise the COHb level to 13.6%, would be higher (e.g., a background COHb level of 3% would result in 28.6 ppm CO, and a background of 8% would result in 57.4 ppm CO).

5. A child with a lower total rate of ventilation (represented by V_E), such as a spectator (light activity versus the medium activity assumed above), might only be breathing at a rate of 11,300 ml/min (EPA 2008b; Table 6-13). The estimated CO air concentration, to raise the COHb level to 13.6%, would increase to 24 ppm.

6. A heavier child would increase the estimated CO air concentration needed to raise the COHb level to 13.6%. For instance, the mean body weight of a 6- to 11-year-old child, *regardless of sex*, is 31.8 kg, per Table 8-1 in the Child-Specific Exposure Factors Handbook (EPA 2008b). This raises the estimated CO air concentration only slightly, to 18 ppm. However, a child on the upper end of body weight for his group (51.4 kg is the 95th percent Upper Confidence Limit for boys; EPA 2008b; Table 8-4), would result in an estimated CO air concentration of 27 ppm.

Therefore, this exercise in back-calculating potential CO air concentrations should not be considered evidence of the exposure that occurred to these children. Rather, it can be used in approximating average CO levels, what factors may cause a person to be more susceptible to the toxic effects of CO (lower body weight), and determining what an acceptable evacuation/-reoccupancy level may be at this facility.

Appendix B. ATSDR Public Health Hazard Categories

Depending on the specific properties of the contaminant(s), the exposure situations, and the health status of individuals, a public health hazard may occur. Sites are classified using one of the following public health hazard categories:

Urgent Public Health Hazard

This category applies to sites that have certain physical hazards or evidence of short-term (less than 1 year), site-related exposure to hazardous substances that could result in adverse health effects. These sites require quick intervention to stop people from being exposed. ATSDR will expedite the release of a health advisory that includes strong recommendations to immediately stop or reduce exposure to correct or lessen the health risks posed by the site.

Public Health Hazard

This category applies to sites that have certain physical hazards or evidence of chronic (long-term, more than 1 year), site-related exposure to hazardous substances that could result in adverse health effects. ATSDR will make recommendations to stop or reduce exposure in a timely manner to correct or lessen the health risks posed by the site.

Indeterminate Public Health Hazard

This category applies to sites where critical information is lacking (missing or has not yet been gathered) to support a judgment regarding the level of public health hazard. ATSDR will make recommendations to identify the data or information needed to adequately assess the public health risks posed by this site.

No Apparent Public Health Hazard

This category applies to sites where exposure to site-related chemicals might have occurred in the past or is still occurring, but the exposures are not at levels likely to cause adverse health effects. ATSDR may recommend any of the following public health actions for sites in this category:

- cease or further reduce exposure (as a preventive measure)
- community health/stress education
- health professional education
- community health investigation.

No Public Health Hazard

This category applies to sites where no exposure to site-related hazardous substances exists. ATSDR may recommend community health education for sites in this category.

For more information, consult Chapter 9 and Appendix H in the 2005 ATSDR Public Health Assessment Guidance Manual (http://www.atsdr.cdc.gov/HAC/PHAManual/index.html).