U.S. Department of Housing and Urban Development

# Healthy Homes Issues: Carbon Monoxide

HEALTHY HOMES INITIATIVE (HHI) BACKGROUND INFORMATION

December 2005

# Healthy Homes Issues: Carbon Monoxide

**December 2005**

Prepared for:

U.S. Department of Housing and Urban Development (HUD)
Office of Healthy Homes and Lead Hazard Control
Washington DC 20410

Prepared by:

Peter Ashley, DrPH, U.S. Department of Housing and Urban Development (HUD)
Jack Anderson, Healthy Housing Solutions
John R. Menkedick, Battelle
Maureen A. Wooton, Battelle

# Acknowledgments

We thank the following individuals for their helpful comments and information used in preparation of this document.

Albert Donnay, MHS
MCS Referral & Resources, Inc.

Ric Erdheim
National Electrical Manufacturers Association

Warren Galke, PhD
National Children's Study Program Office
National Institute for Child Health and Human Development
National Institutes of Health

Thomas H. Greiner, Ph.D., P.E., Associate Professor
Ag. and Biosystems Engineering
Iowa State University Extension

Sandra Inkster, PhD
Elizabeth W. Leland
Richard Stern
Donald W. Switzer
Susan Carlson
U.S. Consumer Product Safety Commission

Paul Patty
Underwriters Laboratories (UL)

James A. Raub
National Center for Environmental Assessment
United States Environmental Protection Agency

Robert Vanderslice, PhD
Environmental Health Risk Assessment Office
Rhode Island Department of Health

# Preface

In October 1998, in response to Executive Order 13045 on "Protection of Children from Environmental Risks and Safety Risks," the U.S. Department of Housing and Urban Development (HUD) launched the Healthy Homes Initiative (HHI). The primary goal of the HHI is to protect children from housing conditions that are responsible for multiple diseases and injuries. As part of this initiative, HUD is preparing a series of papers to provide background information to their current HHI grantees, as well as other programs considering adopting a healthy homes approach. This background paper focuses on carbon monoxide and provides a brief overview of the current status of knowledge on:

- The extent and nature of carbon monoxide in the home;
- Assessment methods for carbon monoxide in the home;
- Mitigation methods for carbon monoxide in the home; and
- Information needs in the field of carbon monoxide research.

Please send any comments to:

Peter Ashley, DrPH
U.S. Department of Housing and Urban Development (HUD)
Office of Healthy Homes and Lead Hazard Control
451 7th Street, SW
Washington, D.C. 20410
Peter_J._Ashley@hud.gov

# Table of Contents



# List of Figures and Tables

# Summary and Overview

Carbon monoxide (CO) is a poisonous gas responsible for hundreds of deaths and numerous non-fatal poisonings each year in the United States. It is a colorless, odorless, and tasteless gas that is produced as a by-product of incomplete combustion of carbon-based fuels such as natural or liquefied propane (LP) gas, kerosene, oil, gasoline, wood, or coal.

Many of the issues related to CO poisoning are familiar to those who have worked on childhood lead poisoning and other environmental health hazards. Questions arise about the health impacts of low level exposures, the actual number of people affected, technologies for measuring levels in the environment or clinical specimens, and mitigation protocols. The literature supports the following findings regarding CO hazards in the home:

HEALTH IMPACTS OF CO

- The severity of health effects from CO exposure depends on various factors, including the age and physical health status of an individual, the duration of CO exposure, and the CO concentration in the air.
- The elderly, pregnant women, fetuses, young infants, and those with certain pre-existing health problems (e.g., those with cardiac or lung conditions) are most susceptible to health effects from CO exposure.
- Both short-term exposures to high concentrations of CO and repeated longer-term exposures to lower concentrations of CO can result in serious health effects.
- Some research shows that repeated exposures to CO, even at levels previously believed to be low, are capable of producing numerous, and persistent, adverse physical, cognitive, and emotional health effects in humans.

REDUCING CO HAZARDS IN THE HOME

- Common sources of elevated CO levels in homes include malfunctioning, improperly or inadequately vented gas heating systems and other combustion appliances such as ovens and generators, and cars that are started or left running in attached garages.
- Preventing CO exposures requires routine periodic maintenance to ensure that the fumes from combustion appliances are adequately vented, as well as responsible operation of combustion appliances and motor vehicles by home occupants.
- Assessment of potential CO sources, as well as behavioral hazards, can be accomplished by occupant surveys and visual inspections of homes.
- Elevated CO concentrations can be assessed, with differing levels of accuracy, through the use of research quality and professional CO detection and monitoring devices.
- Homeowners can purchase low-cost CO alarms that are designed to warn of serious, potentially lethal levels of CO in the home.

# Residential Hazards: Carbon Monoxide

## 1.0 HEALTH IMPACTS OF CARBON MONOXIDE POISONING

Carbon monoxide (CO) is responsible for hundreds of deaths and thousands of non-fatal poisonings each year in the United States (CPSC, 2004; CDC/MMWR, 2005a). Short-term exposures to high concentrations of CO (acute exposure) and repeated longer-term exposures to relatively lower concentrations of CO (chronic exposure) can both result in serious health effects. The elderly, pregnant women, fetuses, young infants, and those with certain pre-existing health problems (e.g., those with cardiac or lung conditions) are most susceptible to health effects from CO exposure (EPA, 2000). Some research has found that repeated exposures to CO, even at levels previously believed to be low, are capable of producing numerous, and persistent, adverse physical, cognitive, and emotional health effects in humans.

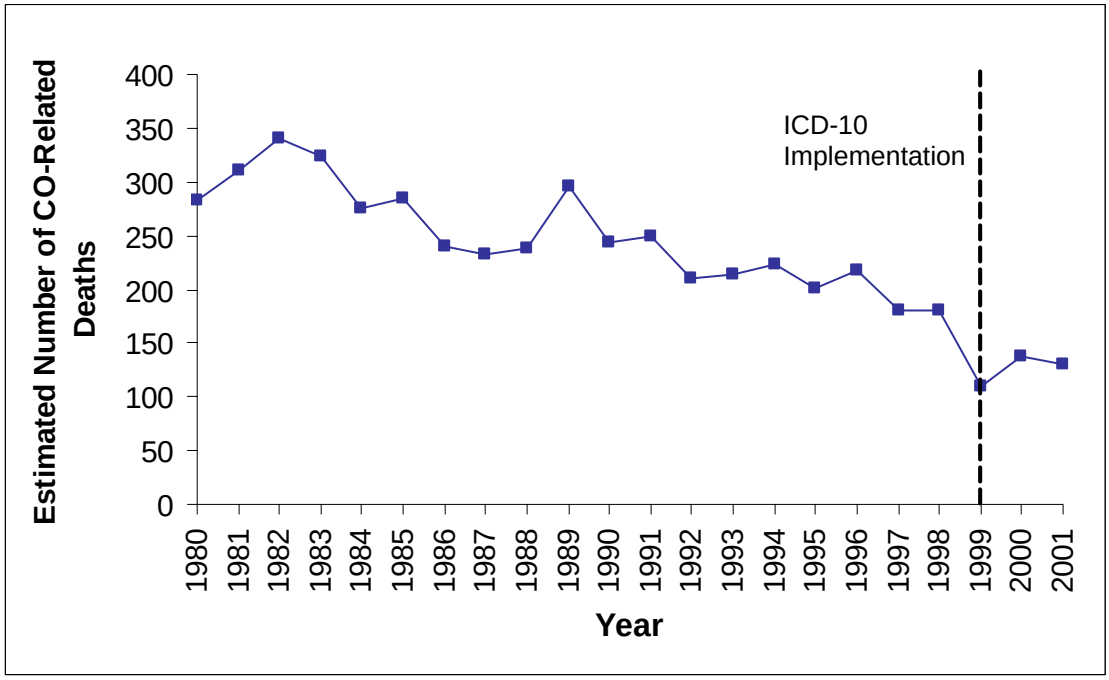
### 1.1 Prevalence of CO Poisoning

**Fatal CO Poisoning.** The number of fatal CO poisonings has generally been declining since the 1980's (see Figure 1); however, there are still hundreds of unintentional deaths per year in the U.S. from CO, with many of these deaths occurring at home (CPSC, 2004). According to the U.S. Consumer Product Safety Commission (CPSC) staff, from 1999 to 2001, the total number of unintentional non-fire CO poisoning deaths associated with the use of consumer products under the jurisdiction of the CPSC (see Table 1), excluding those associated with fire or motor vehicles, averaged about 126 annually (CPSC, 2004). These deaths were associated with various consumer products as shown in Table 1, with the majority caused by CO emissions from heating systems. The majority (66%) of these deaths occurred in the home. The remainder occurred in temporary shelters, such as campers, seasonal cabins and trailers (26%), or other places including inside automobiles, motels, etc. (8%).

Beyond CO fatalities associated with consumer products, many additional unintentional deaths occur each year as a result of CO poisoning from motor vehicle exhaust, including some deaths in homes from motor-vehicle exhaust infiltration into the living space from an attached garage. The National Highway Traffic Safety Administration (NHTSA) estimated that, between 1995 and 1997, the total number of unintentional CO poisoning fatalities from stationary motor vehicles in residential settings was 397 (126 in 1995, 149 in 1996, and 122 in 1997) (NHTSA, 2000)[1].

[1] NHTSA estimates based on analysis of National Center for Health Statistics (NCHS) 1995-1997 data.

**Figure 1. Estimated [1] Non-Fire and Non-Vehicle CO Poisoning Deaths Associated with Consumer Products, 1980 – 2001.**




Source: CPSC, 2004. See footnote for Table 1 for explanation of ICD-10.

[1] Due to changes in the International Statistical Classification of Diseases and Related Health Problems (ICD) with the implementation of the Tenth Revision (ICD-10) in 1999, there are discontinuities in comparing the estimates of CO deaths associated with consumer products in 1999 and later to prior years' estimates.

**Non-Fatal CO Poisoning.** In addition to CO poisoning fatalities, it is estimated that thousands go to hospital emergency rooms for treatment of non-fatal CO poisoning each year (Hampson, 2000). According to CPSC staff, it is not uncommon for CO incidents involving one or more fatalities to also result in one or more non-fatal CO poisoning injuries. During 2001-2003, an estimated 15,200 persons with confirmed or possible non-fire-related CO exposure were treated annually in hospital emergency departments, with most (64%) of the nonfatal CO exposures occurring at home (CDC/MMWR, 2005a). Furthermore, some researchers suggest that CO poisoning commonly goes unreported or is medically misdiagnosed because symptoms can be easily mistaken for other illnesses such as the flu (Penney, 2000; Hampson, 2000; Comstock et al., 1999) and chronic fatigue syndrome (Knobeloch and Jackson, 1999). Therefore, although there is no reliable method for estimating the number of individuals who suffer from symptoms of CO poisoning, it may be considerably larger than reported.

**Table 1 . Average Carbon Monoxide Poisoning Deaths For Each Type of Consumer Product Reported, 1994-1998 and 1999-2001[1], Excluding Fire and Motor-Vehicle Related Deaths**

| Consumer Product | 1994 | 1995 | 1996 | 1997 | 1998 | 1994-1998 Average Estimate | 1994-1998 Average Percent | 1999[1] | 2000[1] | 2001[1] | 1999-2001 Average Estimate | 1999-2001 Average Percent [2] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Total Deaths** | 223 | 201 | 217 | 180 | 180 | 200 | | 109 | 138 | 130 | 126 | |
| **Heating systems** | 177 | 159 | 163 | 135 | 128 | 152 | 76% | 49 | 82 | 75 | 69 | 54% (65) |
| Unspecified gas heating | 59 | 26 | 22 | 12 | 5 | 25 | 12% | 3 | 7 | 6 | 5 | 4% (5) |
| LP gas heating | 35 | 51 | 54 | 41 | 49 | 46 | 23% | 22 | 29 | 26 | 26 | 20% (25) |
| Natural gas heating | 24 | 31 | 19 | 46 | 57 | 35 | 18% | 19 | 37 | 28 | 28 | 22% (27) |
| Coal/wood heating | 6 | 6 | 7 | 5 | 5 | 6 | 3% | 0 | 2 | 6 | 3 | 2% (2) |
| Kerosene/oil heating | 9 | 5 | 15 | 10 | 5 | 9 | 4% | 2 | 6 | 6 | 5 | 4% (4) |
| Heating systems, not specified | 44 | 40 | 47 | 20 | 8 | 32 | 15% | 2 | 1 | 1 | 1 | 1% (1) |
| **Charcoal grills** | 15 | 14 | 19 | 23 | 16 | 17 | 9% | 17 | 8 | 12 | 12 | 10% (12) |
| **Gas water heaters** | 7 | 5 | 8 | 8 | 8 | 7 | 4% | 1 | 3 | 0 | 1 | 1% (1) |
| **Camp stoves, lantern** | 12 | 15 | 3 | 5 | 10 | 9 | 5% | 9 | 3 | 1 | 4 | 4% (4) |
| **Gas ranges, ovens** | 9 | 5 | 15 | 5 | 3 | 7 | 4% | 6 | 11 | 10 | 9 | 7% (9) |
| **Other/ multiple appliances** | 3 | 3 | 8 | 3 | 15 | 6 | 3% | 14 | 3 | 9 | 9 | 7% (9) |
| **Engine-Powered Tools** | | | | | | * | * | 13 | 27 | 23 | 21 | 16% (*) |

Source: Reproduced from CPSC, 2004 and CPSC, 2001.

[1] Due to changes in the International Statistical Classification of Diseases and Related Health Problems (ICD) with the implementation of the Tenth Revision (ICD-10) in 1999, there are discontinuities in comparing the estimate of CO deaths associated with consumer products in 1999 and later to prior years' estimates. [2] Numbers in parentheses represent percent of total excluding Engine-Powered Tools.

* Prior to 1999, estimates could not be calculated for this category.

## 1.2 Differences in Populations at Risk for CO Poisoning

Adults tend to comprise the majority of the deaths occurring from CO poisoning. For example, from 1999-2001, adults 45 years and older accounted for 58 percent of deaths, while children less than 15 years of age only accounted for an average of six percent of yearly CO poisoning deaths (CPSC, 2004). CPSC staff suggest that several factors may contribute to the higher observed incidence of CO-poisoning deaths in older adults, including pre-existing medical conditions that lower a victim's tolerance to carboxyhemoglobin (COHb, see section 1.3 below) in the bloodstream, alcohol and recreational drug use impairing ability to respond appropriately to CO hazards, and the fact that older age groups may tend to own older products that do not conform to more recent improvements in voluntary standards (CPSC, 2004). It should also be noted that the unborn fetus is also considered at increased risk from CO poisoning due to differences in fetal accumulation of CO relative to the mother (i.e., COHb levels may be much higher in the fetus) (Abelsohn et al., 2002; Liu et al., 2003)

Ralston and Hampson (2000) found that the incidence of unintentional CO poisoning differs across racial and ethnic categories. Among 586 Washington state residents treated for severe CO poisoning from 1987 to 1997, black and Hispanic populations had higher relative risks for CO poisoning than white populations (home and non-home CO poisonings included). In addition, the most common sources of CO poisoning differed by racial/ethnic category. For example, for Hispanic and black populations, about 67% and 40%, respectively, of poisonings were due to indoor burning of charcoal briquettes, while all boat-related CO deaths were in white populations (Ralston and Hampson, 2000).

## 1.3 Human Health Effects

CO is poisonous primarily because it prevents the body from using oxygen. When inhaled, CO readily combines with oxygen-binding hemoglobin in the bloodstream to form carboxyhemoglobin (COHb). This interferes with oxygen transport to the tissues and organs of the body and leads to adverse health effects (e.g., neurological impairment), particularly in sensitive organs such as the brain and heart. Eventually, at high enough levels, CO causes death by asphyxiation. The onset and severity of CO poisoning symptoms is influenced by the level and duration of reduced oxygen availability (hypoxia), as well as the sensitivity of the individual. It is possible for permanent injury, with resulting disability, to occur from a single, acute CO exposure. In addition, research indicates that CO has adverse health effects beyond those related to oxygen interference (Devine et al., 2002; Townsend and Maynard, 2002; Thom et al., 1999; Thom et al., 1997; Ryter et al., 2004).

**Chronic and Acute CO Poisoning – General Symptoms**. CO poisoning may occur as a result of both short-term (minutes to hours) exposures to high concentrations of CO (acute exposure) and longer-term exposures (repeated over days to months) to relatively lower concentrations of CO (chronic exposure). Chronic CO poisoning usually involves lower levels of CO in the bloodstream that can cause mild to moderate, nonspecific flu-like symptoms. Unfortunately, victims and/or physicians might not consider CO poisoning as the underlying problem. If CO poisoning is not diagnosed, the misdiagnosis will likely prolong the CO exposure, and could ultimately result in long-term health effects.

Individuals who suffer exposures to elevated levels of CO may be unaware of the source of their health problems because CO poisoning, both chronic and acute, can cause symptoms that vary over time and mimic common illnesses like the flu and other bacterial and viral infections. Symptoms of exposure can begin with a slight headache, subtle sensory-motor deficits, nausea, vomiting, impaired vision, fatigue, dizziness, and shortness of breath. If exposures continue, symptoms become more intense, progressing to a loss of consciousness. Survivors of CO poisoning may also have long-term neurological effects such as sensory abnormalities, personality changes, memory deficits, impaired judgment, poor concentration, and other intellectual impairments (Varon and Marik, 1997; Raub et al., 2000; EPA, 2000). In addition, symptoms may not appear until many days or even months after exposure ceases. (Townsend and Maynard, 2002).

Although the neurotoxicant effects of CO were traditionally thought to be solely a result of a lack of oxygen to the tissues (hypoxia) due to avid binding of CO to hemoglobin, recent studies of CO pathophysiology suggest that additional mechanisms beyond carboxyhemoglobin (COHb) formation are also involved, such as interference with biological pathways in cells and disruption of sensory nerve control (Devine et al., 2002; Townsend and Maynard, 2002; EPA, 2000; Thom et al., 1999; Thom et al., 1997; Ryter et al., 2004). Research (through 1999) reviewed by EPA (EPA, 2000), found a growing body of research on potential impacts of CO on vasomotor control, based on the fact that CO is continually produced by the human body as part of normal physiology and acts at very low levels as a neurotransmitter in the control of sensory nerves (EPA, 2000). EPA also found ongoing CO research focused on the ability of CO to cause disruption of intracellular and cellular level (e.g., mitochondrial) functions via free-radical-mediated changes (EPA, 2000). Although controversy exists over the role that other processes such as these may play in either acute or chronic CO poisoning, phenomena such as delayed neurological sequelae cannot be explained by hypoxia alone (i.e., after COHb levels have returned to normal and hypoxic stress is removed, symptoms would be expected to improve) (Townsend and Maynard, 2002). Other mechanisms of CO pathophysiology continue to be investigated.

**Diagnosis and Measurement of Severity of CO Poisoning.** In general, determining the level of injury caused by CO poisoning is not always possible, and as discussed below, even confirmation of CO poisoning can be difficult. Acute CO poisoning can be assessed by measurement of COHb (see below), but this is only reliable if done within hours of exposure because CO has a half-life that may be as short as 4.0 to 6.5 hours (Penney, 2001; EPA, 2000). A reliable biological marker for determining the severity of chronic CO poisoning has yet to be developed (Devine et al., 2002). For example, Devine et al (2002) identified some of the long-term health impacts of chronic CO poisoning only through extensive neurological and psychological testing.

Although there are many clinical tests that can be conducted on people with suspected cases of CO poisoning, currently the only test that measures CO directly is blood carboxyhemoglobin (COHb) saturation (Vreman et al., 2000). COHb can be determined by direct analysis of venous or arterial blood, or by measuring expired CO (in parts per million, ppm) with a breath analyzer and converting to COHb (Vreman et al., 2000). In a CO breath analysis study, Cunnington and Hormbrey (2002) found that breath analysis was rapid and results correlated well with recent CO exposure.

Measured COHb levels can be affected by a variety of factors (e.g., time, interaction with other substances in the bloodstream such as administered oxygen, physiological differences among people), and levels have been shown in many cases to correlate poorly with the signs and symptoms of acute CO poisoning (Raub et al., 2000). As a result, low COHb levels should never be solely relied upon to exclude a case of CO poisoning (Vreman et al., 2000; Penney, 2001; Cunnington and Hormbrey, 2002; Benignus et al., 1990; Raub and Benignus, 2002).

The technical literature, as well as many public outreach materials (e.g., instruction manuals that come with CO alarms), often include references to specific CO poisoning symptoms that may be expected as a function of percent COHb measured in the blood and/or as a function of CO concentrations in the air; however, such correlates between symptoms, percent COHb, and levels of CO in the air are imprecise (Donnay, 2003; WHO, 1999). Donnay (2003) investigated the origin of this information, and found that there is one original study (Sayers and Yant, 1923), which appears to serve as the basis for the currently reported COHb-symptom correlates. Donnay also found that the 1923 study did not appear to be based on human studies, and contained many caveats on usage from the original authors. For this reason, Donnay recommends that comparisons only be made in relation to normal human baseline COHb concentrations.

Baseline COHb concentrations, for nonsmokers, typically remain below 2% (EPA, 2000). According to the World Health Organization's (WHO) Environmental Health Criteria document (#213) for CO (WHO, 1999), even though measured COHb levels may be poorly correlated with symptoms, COHb levels below 10% are usually not associated with symptoms. EPA, however, also cites studies in their Air Quality Criteria for CO document that demonstrated adverse effects of CO at COHb levels as low as 2.9 to 3.0% in persons with coronary artery disease and chest pain (EPA, 2000). At higher COHb saturations of 10–30%, WHO states that neurological symptoms of CO poisoning can occur, such as headache, dizziness, weakness, nausea, confusion, disorientation and visual disturbances. Shortness of breath, increases in pulse and respiratory rates, and loss of consciousness are observed with COHb levels from 30% to 50% (WHO, 1999). When COHb levels are higher than 50%, coma, convulsions, and cardiopulmonary arrest may occur (WHO, 1999). It should be emphasized that these ranges can only provide a rough idea of the potential effects of acute CO exposure, due both to the high variability in measurement of COHb and differences in individual susceptibility to CO toxicity. Low COHb levels do not necessarily exclude a case CO poisoning (Vreman et al., 2000; Penney, 2001; Cunnington and Hormbrey, 2002; Benignus et al., 1990; Raub and Benignus, 2002).

Research has shown that COHb levels at which symptoms of CO poisoning begin to occur can vary widely with the individual (due to differences in factors such as metabolic rate, health status, smoking, or sensitivity) and the situation. For example, Sanchez et al. (1988) observed striking disparity in the symptoms of two children of similar age (27 and 28 months) exposed simultaneously to the same environment, resulting in similar COHb levels: a 27-month old child with a COHb level of 35.0% was flaccid and poorly responsive when brought to the emergency room, while a 28-month old with a COHb level of 33.6% was asymptomatic. Measured COHb levels can also be dissimilar in individuals with the same CO exposures (Sanchez et al., 1988). Even in known cases of CO poisoning, measured COHb may be unexpectedly low due to a long

time interval between leaving the site of exposure and drawing blood for measurement, resuscitation attempts (i.e., administration of oxygen), or the presence of other substances (e.g., drugs) in the bloodstream (Penney, 2001). Carboxyhemoglobin levels can decline quickly once the source of CO exposure is removed; the half-life of COHb in the body may be as short as 4.0 to 6.5 hours, with levels usually returning to background within 24 hours (Penney, 2001; EPA, 2000). Research also indicates that the unborn fetus may be especially vulnerable to CO exposure due to the higher affinity of fetal hemoglobin to CO than adult hemoglobin, as well as possible CO interference with placental transport functions; as a result, fetal COHb may significantly exceed maternal COHb (Abelsohn, et al., 2002; Liu et al., 2003). Therefore, although a high COHb level may confirm CO poisoning, low COHb cannot exclude it.

COHb levels may also become elevated for reasons other than exposure to CO from appliances and vehicles. For example, in individuals smoking one to two packs of cigarettes a day, baseline COHb concentrations average 4%, with a usual range of 3% to 8% (EPA, 2000). COHb concentrations as high as 15% have been reported in chain smokers (EPA, 2000). In addition, CO that is formed during the normal course of metabolism contributes to baseline COHb levels, including CO produced endogenously through heme degradation; metabolism of drugs; and degradation of unsaturated fatty acids, inhaled solvents, and other xenobiotics (EPA, 2000). Baseline COHb levels have also been observed to be higher in certain groups, such as untreated asthmatics and critically ill patients (EPA, 2000; Omaye, 2002). These higher CO levels were attributed to stress-induced increases in heme oxygenase (e.g., from illness, lung inflammation), which in turn create endogenous CO (EPA, 2000; Omaye, 2002).

**Research on Health Effects of Chronic and Low Level Exposures.** Although there is no clear consensus in the literature regarding the definition of a "low level" CO exposure, in general, low level CO exposure for the general population can reasonably be characterized as exposure to an air concentration of CO that is less than EPA's current National Ambient (outdoor) Air Quality Standards of 9 ppm (8-hr average) or 35 ppm (1-hr average).

Though not universally accepted, some research has found that prolonged repeated exposures to CO, even at levels previously believed to be low, are capable of producing numerous, and persistent, adverse physical, cognitive, and emotional health effects in humans (Penney, 2000; Devine et al., 2002; Liu et al., 2003). For example, Ritz and Yu (1999) investigated the potential adverse physical effects of low-level CO exposures by looking at the relationship between outdoor ambient exposure during the last trimester of pregnancy on the frequency of low birth weight among neonates (125,573 children) born 1989-1993 to women living in the Los Angeles, California area. Results of the analysis showed that exposure to ambient CO in the range of 5.5 to 7 ppm during the last trimester of pregnancy was associated with a significantly increased risk for low birth weight. In a similar study, Liu et al. (2003) reported an association between preterm birth and ambient CO exposures during the last month of pregnancy in Vancouver, Canada between 1985 and 1998. Devine et al. (2002) also observed mild, but persistent (17 and 29 months after exposure), symptoms of nervous system dysfunction in a case study of one woman chronically exposed to low level CO over the course of at least one year.

Slight reductions in maximal exercise duration and performance in healthy adults, and decreased exercise tolerance and increased chest pain in individuals with coronary artery disease, have also been associated with low level (<6% COHb) CO exposure (EPA, 2000).

Yang et al. (2005) examined the associations between ambient levels of gaseous air pollutants, including CO, and hospitalization for chronic obstructive pulmonary diseases (COPD) among elderly people living in Vancouver, British Columbia, Canada, a city in which ambient air pollution levels are relatively low. Regressing the logarithm of daily counts of acute COPD hospitalization during a 5-year period from 1994 to 1998 on the daily mean levels of CO, the researchers found that CO was significantly associated with hospitalization for COPD, and the magnitude of effects was increased slightly with increasing days of exposure, with a relative risk for a 7-day average being 1.08 (Yang et al., 2005).

EPA's Air Quality Criteria for Carbon Monoxide (EPA, 2000) reviews recent research related to effects of low level exposure to CO, including several studies investigating asthma exacerbation in relation to short term ambient levels of CO and other air pollutants (Sheppard et al., 1999; Norris et al., 1999). Although exposure to ambient CO levels of just 2 to 3 ppm was correlated with asthma exacerbations and hospitalizations in several of the studies, EPA found that physiological mechanisms for CO exacerbation of asthma are unclear and epidemiologic observations on the relationship between short-term low levels of CO exposure and the frequency of respiratory disease cannot yet be interpreted with confidence (EPA, 2000). In a later study of 133 children (5-13 years of age) with asthma residing in the greater Seattle, Washington, area, Yu et al. (2000) observed a population average 30% increase in the odds of asthma symptoms for a 1-ppm increment in CO, using single pollutant models. This increase lagged 1 day. In the studies reviewed by EPA, as well as the Yu et al. (2000) study, the authors hypothesized that CO may either be a marker for other combustion products which exacerbate asthma, or may be associated with an increased susceptibility to CO that asthmatics experience with exercise induced airflow limitation (Sheppard et al., 1999; Norris et al., 1999), although these authors did not include consideration of how the exogenous CO exposures may interact with endogenous CO pathways.

More recent studies (Hwang et al., 2005; Estrella et al., 2005), have observed associations between ambient (traffic-related) CO exposures and respiratory symptoms. Hwang et al. (2005), conducted a nationwide cross sectional study of 32,672 Taiwanese school children in 2001 that compared risk of childhood asthma with air pollution monitoring data for sulfur dioxide ($SO_2$), nitrogen oxides (NOx), ozone ($O_3$), CO, and particles with an aerodynamic diameter of 10 microm or less ($PM_{10}$). Using a two stage hierarchical model adjusting for confounding factors, the researchers found that the risk of childhood asthma was positively and significantly associated with CO, as well as $O_3$ and NOx; supporting the hypothesis that long term exposure to traffic related outdoor air pollutants such as NOx, CO, and $O_3$ increases the risk of asthma in children. In contrast, the risk of childhood asthma was weakly or not related to $SO_2$ and $PM_{10}$.

Estrella et al. (2005) found evidence for a link between traffic-related CO exposure and susceptibility to acute respiratory infections (ARIs), using COHb as a marker for chronic CO exposure. Comparing data on ARIs and COHb concentrations in 960 school-age children living in urban and suburban areas of Quito, Ecuador, they found that in a random subsample of 295 children, average COHb concentrations were significantly higher in children attending schools in areas with high and moderate traffic, compared with the low-traffic area. The percentage of children with COHb concentrations above 2.5% were 1, 43, and 92% in low-, moderate-, and high-traffic areas, respectively. Children with COHb above 2.5% were also 3.25 times more likely to have ARI than children with COHb < 2.5%, and with each percent increase in COHb

above 2.5%, children were 1.15 times more likely to have an additional case of ARI. This study did not control for other traffic-related pollutants such as nitrogen dioxides.

Research on potential mild central nervous system effects, effects on the developing fetus and birth weight, and interaction with other stressors such as alcohol, heat, or other pollutants is also discussed in the EPA report, although EPA draws no conclusions at this time. In general, the literature suggests that the severity of health effects from CO depends on various factors, including the age and physical health status of an individual, the duration of CO exposure, and the CO concentration in the air. The elderly, pregnant women, fetuses, young infants, and those with pre-existing medical conditions (especially those with cardiac and pulmonary conditions) are most susceptible to health effects from CO exposure (EPA, 2000).

## 2.0 CARBON MONOXIDE HAZARDS IN THE HOME

Based on the sources and likelihood of significant exposure, the primary residential hazards and conditions associated with CO exposure and poisoning are:

- Malfunctioning or inadequately vented gas, oil, or wood burning appliances, including:
    - Water heaters
    - Central heating appliances including furnaces and boilers
    - Dryers
    - Fireplaces and woodstoves
    - Vented space heaters

- Malfunctioning or improperly operated unvented appliances including:
    - Kerosene heaters
    - Unvented space heaters
    - Ranges and ovens

- Properly functioning consumer products can pose a CO hazard when they are operated incorrectly, including
    - Charcoal/gas grills or hibachis used indoors or in confined spaces
    - Gasoline-powered electric generators used in confined spaces
    - Gasoline-powered vehicles started or left idling in attached garages, even with the garage door open

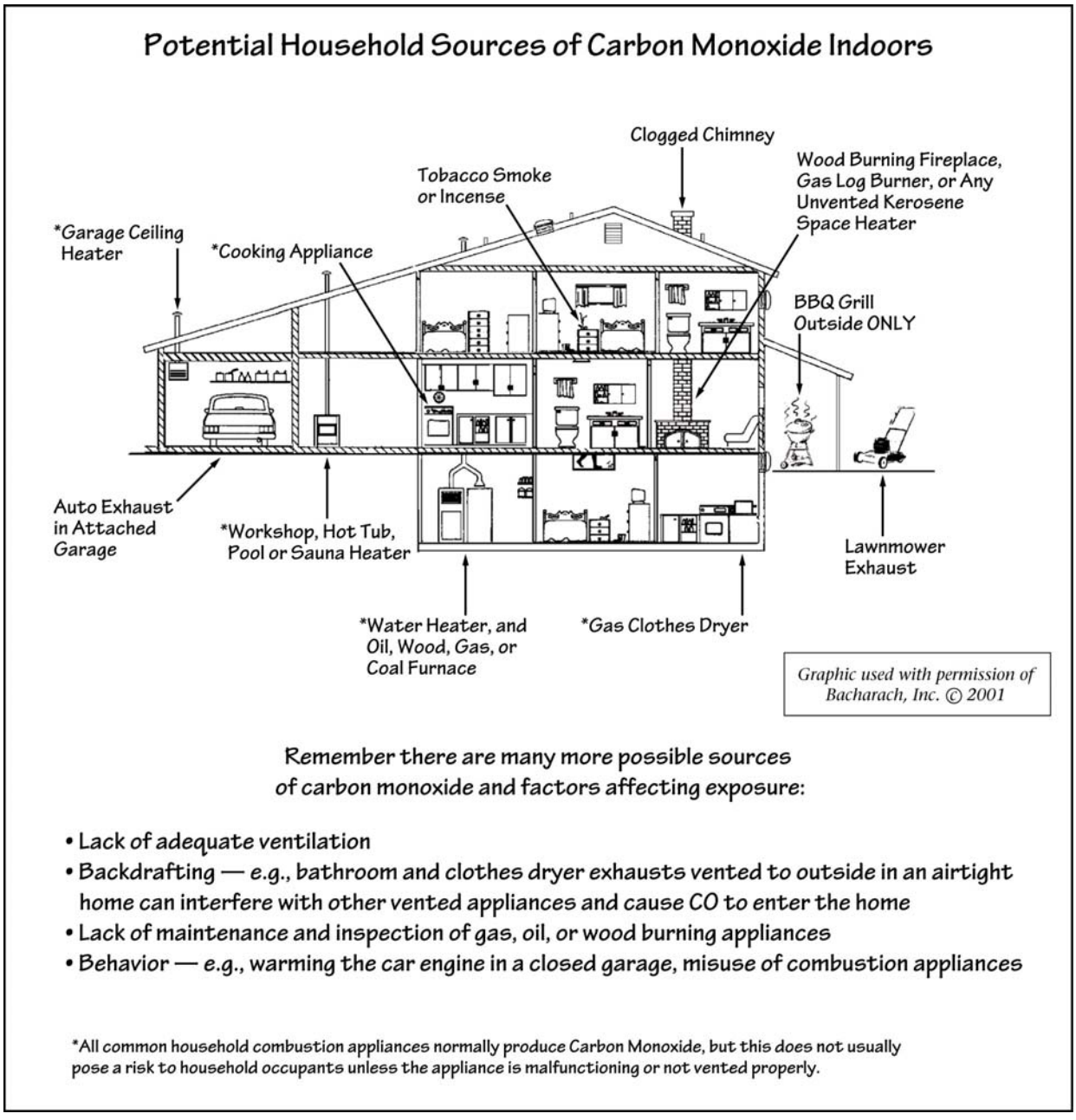
- Housing design
    - Lack of proper ventilation in attached garages (e.g., 100 cfm of continuous exhaust ventilation according to section 403.3 of the International Mechanical Code)
    - Conditions which create backdrafting
    - Lack of maintenance and yearly professional inspection of gas, oil, or wood burning appliances and their vent systems
    - Lack of CO alarms

- Behavior (e.g., idling automobiles in attached garages, using gas ovens for space heating, misuse of heating and combustion appliances, cigarette smoking)

## 2.1 Potential Residential Carbon Monoxide Sources

Figure 2 illustrates common potential sources of CO in the home. In situations where elevated CO levels are detected in a home (e.g., via a CO alarm sounding), the source, or sources, may be difficult to isolate, especially because many CO problems are intermittent in nature (Greiner and Schwab, 2000).

**Figure 2. Potential Residential Sources of Carbon Monoxide Indoors (used with permission of Bacharach, Inc. ©2001)**



Major potential CO sources in residential situations include malfunctioning or inadequately vented combustion appliances (e.g., such as furnaces, hot water heaters, fireplaces and woodstoves, and gas dryers), malfunctioning or improperly operated unvented combustion appliances designed to be used indoors (e.g., gas ovens and ranges, kerosene space heaters), charcoal or gas grills and other combustion devices that should not be used indoors (e.g., gasoline-powered generators, engines, or tools), and the start-up and idling of vehicles in attached garages. Tobacco smoke can also contribute to CO levels in indoor air, although, unless other sources are present, the increase in CO levels associated with tobacco smoke is typically insufficient to cause CO alarms to sound (EPA, 2000).

**Backdrafting.** CO levels can become elevated in buildings where backdrafting is occurring. Backdrafting occurs when the air pressure within a home is lower than the air pressure outside, a phenomenon known as house depressurization. When these conditions exist, flue combustion gasses (CO, $CO_2$, $NO_2$, etc.) can reverse direction, spilling into the living area of a home instead of traveling up a vent or chimney.

Buildings with a relatively tight envelope (few sources for air to enter) and high exhaust capacity are especially prone to depressurization. Appliances with passive ventilation via a draft hood (e.g., water heaters) may also be particularly susceptible to backdrafting. Backdrafting may be triggered by a constricted or poorly functioning chimney, improperly designed or maintained venting systems, or suction created by the operation of household equipment such as exhaust fans, clothes dryers and fireplaces (Nagda et al., 1996; CMHC, 1998). Because of the sporadic nature of some of these triggers, backdrafting is often difficult to diagnose. A simple change in wind speed or direction, turning off an exhaust fan, or opening a door or window may be enough to alleviate the depressurization and allow flue gasses to rise. Visual clues like soot on cobwebs and excess moisture can indicate a backdrafting problem. Condensation on windows and other moisture problems result from the water vapor that is produced when burning most fuels. Nagda et al. (1996) reviewed literature devoted to this subject and found that, while the causes of house depressurization and backdrafting are well understood, additional research is needed on the frequency, duration, and severity of depressurization-induced spillage events in a broad cross-section of houses.

**Vented Combustion Appliances.** The contribution to CO in the indoor environment from vented combustion appliances (furnaces, hot water heaters, and gas clothes dryers) is generally negligible unless the unit or ventilation system is malfunctioning, leaking, or backdrafting (EPA, 2000). Dangerous levels of CO have been noted in cases where the venting system leaked (e.g., deteriorating vent systems or chimneys), was improperly installed or designed (e.g., vents too short or at improper angles), or was otherwise malfunctioning due to factors such as blockages caused by bird nests or leaves or the occurrence of backdrafting (as discussed above). According to CPSC staff estimates for 1999-2001, some form of venting problem was noted in about 17 percent of the annual average total CO poisoning deaths and 29 percent of fatalities associated with heating systems (CPSC, 2004). In follow-up investigations of selected incidents, CPSC staff found that specific venting problems included: detached or improperly installed or maintained vents; deteriorating or collapsing chimneys; outdoor debris, birds' nests, or small animals in the chimney or flue pipe creating a blockage; blockage by soot caused by inefficient combustion (which in turn may have been caused by several factors, such as leaky or clogged

burners, an over-firing condition, or inadequate combustion air); or improperly functioning exhaust fans. Less frequently, other conditions related to furnaces included cracked heat exchangers, filter door or covers that were removed or not sealed, and dirty filters.

**Unvented Combustion Appliances.** In contrast to vented appliances like furnaces, some combustion appliances (e.g., kerosene- and propane-fueled space heaters, some gas-fueled log sets, and gas cooking ranges and ovens) are not designed to vent directly outdoors. Although the use of unvented combustion heating appliances is common throughout the United States, the percentage of adults using these devices is higher in the South, among low-income groups, among blacks, and among rural residents (CDC/MMWR, 1997).

Assessing the potential impact of unvented gas cooking ranges and ovens as a significant source of CO is difficult. According to an EPA report, because unvented gas cooking ranges and ovens are used intermittently for cooking purposes, it is not likely that their use would result in substantial increases in CO over long periods of time, except possibly in households where gas ovens are being used improperly as a primary or secondary source of heat (EPA, 2000). However, some researchers have expressed concern over the potential for high concentrations of CO for even short periods of time resulting from either extended cooking or poor burner performance, or due to practices such as covering oven floors with aluminum foil (Tsongas, 1995). Short-term peak CO concentrations of 1.8 to 120 ppm have been associated with the use of unvented gas stoves for cooking (EPA, 2000). The current American National Standards Institute (ANSI) oven flue standard for CO does not address these concerns (Greiner and Schwab, 2000) (see Table 2). However, of larger concern is the improper use of gas ranges and ovens as a primary or secondary source of heat (Slack and Heumann, 1997). Data from the National Health and Nutrition Examination Survey (NHANES) indicate that, of 83.1 million adults surveyed who used gas stoves or ovens for cooking during the years 1988 to 1994, 7.7 million had used the stoves for supplemental heating at least one time during the previous year (CDC/MMWR, 1997). Improper use of the stove or oven as a heating device was more common among rural than among urban residents, and higher among adults in the South than in any other region. In all regions, the use of stoves or ovens for heating in low-income households was approximately twice that in high-income households (CDC/MMWR, 1997).

The use of unvented space heaters can pose risks for elevated CO levels in indoor environments with inadequate ventilation (EPA, 2000). NHANES data indicate that an estimated 13.7 million adults used unvented combustion space heaters between 1988 and 1994 (CDC/MMWR, 1997). This includes an estimated 13.2% of the adult population in the southern United States (CDC/MMWR, 1997). Dutton et al. (2001) observed significant CO accumulations indoors when unvented gas fireplaces were used for extended periods of time. In one case, CO concentrations of greater than 100 ppm accumulated in under 2 hours of operation (Dutton et al., 2001). Other unvented sources can also be a hazard when used improperly in an enclosed or partially enclosed environment – such sources can include charcoal or gas grills, hibachis, or gasoline-powered tools or engines, such as portable generators, pumps, or power washers. When generators are used in garages or in living areas of homes CO concentrations can rise to greater than 1000 ppm in a very short time. Two Centers for Disease Control and Prevention (CDC) investigations of deaths and emergency department visits attributed to CO poisoning after hurricanes in 2004 and 2005 in the states of Florida, Alabama, Louisiana, and Mississippi determined that misplacement of portable, gasoline-powered generators (e.g., indoors, in

garages, or outdoors near windows) was responsible for nearly all of these CO exposures (CDC/MMWR, 2005b; CDC/MMWR, 2005c). Further research has also reported associations between extreme weather, such as ice storms leading to prolonged losses of power, and cases of CO-poisoning related to the use of alternative heating and cooking sources (Ghim and Severance, 2004; Broder et al., 2005; Hampson and Zmaeff, 2005).

**Automobiles in Attached Garages.** CO can also potentially be drawn into a house from any combustion source being operated in an attached garage, including motor vehicles, lawn mowers, or grills. Even if the garage doors are open, CO can seep into the house, particularly in situations where backdrafting is occurring (e.g., CO seepage into homes from attached garages during cold winter months due to temperature differentials). In an Iowa State Study, researchers found that after only two minutes of warming-up an automobile in an opened garage, CO concentrations in the garage rose to 575 ppm (ISU, 1998; Greiner and Schwab, 1998). Within one minute, measurable levels of CO seeped into the house and after only 45 minutes the level in the house rose to 23 ppm. Eight hours later CO concentrations still remained above 9 ppm (ISU, 1998). Although ambient levels of CO from automobile exhaust have decreased dramatically (approximately 90 percent between 1965 and 1992) due to progressively tighter motor vehicle emission controls and the introduction of the catalytic converter in 1975 (EPA, 2000; Mott et al., 2002), even well-tuned engines will produce large concentrations of CO for the first minute or two of operation (ISU, 1998; Burch et al., 1996). Catalytic converters do not work efficiently until they are warmed up (to about 300 ˚C), which usually takes one to three minutes after starting a cold engine (ISU, 1998; Burch et al., 1996). While a properly working catalytic converter typically reduces the concentration of CO in automobile exhaust to fewer than 100 ppm, the CO concentration released by a cold or otherwise non-working converter can be up in the tens of thousands of parts per million, potentially up to 80,000 ppm (ISU, 1998; Lucas, 1953). As a result, many acute CO poisoning episodes continue to occur from exposure to automobile exhaust. For example, NHTSA estimated that between 1995 and 1997, the total number of unintentional CO poisoning fatalities from stationary motor vehicles in residential settings was 397 (NHTSA, 2000). CDC also reports that some motor-vehicle-related CO deaths in garages have occurred even though the garage doors or windows have been open, suggesting that passive ventilation may not be adequate to reduce risk in semi-enclosed spaces (CDC/MMWR, 1996).

CO researchers at Iowa State University recommend installation of exhaust fans in attached garages to prevent CO from entering the house and speed the removal of CO from the garage, but also emphasize that even with a garage exhaust fan (or with the garage door open) it is not safe to operate any sort engine in the garage (ISU, 1998). Standards such as those included in the International Mechanical Code (ICC, 2003), which require attached residential garages to have 100 cfm of continuous exhaust per bay (Section 403.3), have been established to help prevent buildup of toxics like CO in these situations; however, standards such as these have not been consistently adopted or implemented across the country at this time.

## 2.2 Behavioral Hazards and Lack of Prevention

Many unintentional CO poisonings in the home are the result of occupant behavior and lack of knowledge about potential sources of CO (e.g., using ovens for secondary heating sources),

preventive measures (e.g., use of alarms, furnace maintenance), and the proper response to a suspected problem. Common behavioral hazards include:

- Improper use, cleaning, and maintenance of gas ranges and ovens (e.g., blocking secondary air ports with aluminum foil, using range burners or ovens to heat the home, not turning on exhaust ventilation prior to turning on oven or range top burners);
- Improper use of propane, natural gas or charcoal barbecue grills, portable generators, or any gasoline-powered tool (i.e., using indoors or in an attached garage);
- Unsafe behaviors where attached garages are present (e.g., starting a vehicle in a closed garage, idling the car in or near an attached garage (i.e., car should be pulled out immediately onto the driveway, then the garage door closed to prevent exhaust fumes from being drawn into the house);
- Lack of regular cleaning of the clothes dryer and other ductwork and outside vent covers for blockages such as lint, snow, or overgrown outdoor plants;
- Lack of CO alarms, or improper use, placement, or maintenance of CO alarms;
- Lack of regular chimney flue cleaning, regular inspection and maintenance (by a professional when necessary) of gas or other fuel-burning appliances, etc.
- Lack of mechanical exhaust ventilation in attached garage and kitchen, as required by Section 403.3 of the International Mechanical Code.

A questionnaire-based study of 1003 respondents representing households in the continental United States found that although most respondents reported having a smoke alarm (97%), only 29% had a CO detector (Runyan et al., 2005).

It should also be noted that in 2001 an estimated 18 percent of CO victims were noted as having used alcohol or recreational drugs during the time period surrounding the incident (CPSC, 2004). Alcohol and recreational drug use can act as a central depressant causing dulled reactions, potentially impairing a person's ability to react appropriately to a CO hazard (CPSC, 2004).

# 3.0 METHODS USED TO ASSESS CARBON MONOXIDE HAZARDS IN THE HOME

A variety of methods are available for assessing CO hazards in the home. Assessment of potential CO sources, as well as behavioral hazards, can be accomplished by occupant surveys and visual inspection. Elevated CO concentrations can be assessed, with differing levels of accuracy, through the use of various CO monitors (e.g., research quality and professional CO monitoring devices). Homeowners can also purchase low-cost CO alarms that warn of serious, potentially lethal levels of CO.

## 3.1 Surveys and Visual Inspection

Occupant surveys and visual inspection can be used to evaluate housing conditions, as well as behavioral factors, that contribute to CO hazards. For example, in addition to visually inspecting combustion equipment, first responders to CO alarms may survey occupants about the activities occurring in the house at the time the alarm sounded.

Surveys and inspections are also used to identify inappropriate use of equipment (e.g., cooking ranges used for heating, space heaters in violation of codes, etc.) or other occupant behavior that might affect CO exposure, such as idling a vehicle or operating a generator in an attached garage. Commonly recommended points for homeowner education regarding CO hazards and behavior are discussed under mitigation methods in section 4.1 below.

Housing conditions assessed through surveys and visual inspection may include housing design (e.g., the presence of an attached garage), installation of appropriate ventilation devices, presence of appliances that may contribute to CO exposure, or visual evidence of problems with equipment, chimneys, flues, vents, or ventilation. For example, visual evidence of the backdrafting of combustion gases includes soot, scorched surfaces, and melted fittings near the vent (CMHC, 1998). Observations such as excess condensation on windows, and practices such as lining ovens with aluminum foil are also visual indications of possible CO problems. Recommendations for regular professional inspection of equipment and relevant housing conditions are discussed under mitigation methods in section 4.2 below. Checklists help ensure that all potential sources are investigated.

## 3.2 Analytical Methods for Assessing CO

**Field Monitors and Research Instruments.** A variety of field instruments are commercially available for investigation of residential CO levels, many available at a cost of less than $300-500. Currently, the assessment of CO levels in homes by researchers and professional investigators (e.g., from the gas utility) is most often conducted using a commercial analyzer equipped with an active sampler and either a nondispersive infrared (NDIR) absorption sensor, or an electrochemical cell sensor.

The NDIR method, which is generally accepted as the most reliable, continuous method for measurement of CO in ambient air, is based on the specific absorption of infrared radiation by the CO molecule and is extremely sensitive over wide concentration ranges. The most sensitive, commercially available analyzers using NDIR technology are able to detect minimum CO concentrations of about 0.02 ppm (EPA, 2000). The EPA-designated reference methods for collecting CO measurement data for National Ambient Air Quality Standards (NAAQS) are automated methods using NDIR technology (EPA, 2000). Portable analyzers using NDIR technology are available for home assessments with the capability of measuring extremely low-levels of ambient CO (i.e., down to ppb). However, they are expensive (e.g., up to tens of thousands of dollars) and are typically used only in research settings where the extra sensitivity is needed.

Although the electrochemical sensor technology, which is based on the measurement of electrical currents generated as a result of chemical reactions that occur in the presence of CO, is less sensitive and more susceptible to interferences (from water vapor and other gases) than NDIR technology, it is less expensive (e.g., typically a few hundred dollars) and sufficiently sensitive (some down to 1ppm) for the identification of CO poisoning hazards[2]. Therefore, for

[2] For example, available automated data logging CO analyzers include: PocketCO (accuracy +/- 10% of reading) for $129, http://www.quantumfields.com/Pocketco.html ; HOBO CO Datalogger (accuracy +/- 7% of reading, no digital display) for at $220, www.onsetcomp.com/Products/Product_Pages/Other_HOBOs/co_data_logger.html;

routine CO screening, or in situations where the goal is to identify higher concentrations of CO that represent a health risk, palm-held electrochemical sampler/analyzers are most commonly used. Electrochemical sensors are also used in certain types of home CO alarms. Upper-end (e.g., up to a few thousand dollars) electrochemical sensor analyzers are available that, with frequent recalibration, can exhibit sensitivities comparable to NDIR. The normal performance range expected for automated CO analyzers is 0 to 1,000 ppm, with some instruments available that offer higher or lower ranges for specific uses. For example, a CO analyzer with a range up to about 1,000 ppm might be needed to monitor CO levels in a parking garage. In comparison, sensors used in home CO alarms are intended for the purpose of providing warning of potentially dangerous CO levels (generally above 70 ppm) and therefore do not need such a large range.

Other methods also commonly used to assess CO levels include gas chromatography/canister sampling methods for measuring low level background CO levels, and passive samplers (e.g., badges and spot detectors) used to monitor personal exposure to CO. Regardless of which method is used, and as with all field instrumentation, accurate results are dependent on appropriate training for those using the instrument, as well as routine maintenance/calibration of the instrument. Badges and spot detectors provide measurements of exposure based on color change, and can't be calibrated or reset to zero after exposure since they offer no digital display. Using multiple instruments with differing vulnerabilities to interference is one method of verifying suspect readings.

**CO Concentrations Indoors.** CO concentrations in the indoor environment vary based on the source emission rate, use pattern (i.e., intermittent or constant use), ambient outdoor CO concentration, air exchange rate, building volume, and air mixing within the indoor compartments (EPA, 2000). Generally, all-electric homes have lower CO readings than homes that have combustion appliances, although even all-electric homes may still contain several potentially hazardous sources of CO (e.g., fireplaces, electric ovens in self-cleaning mode, and attached garages) (http://www.karg.com/CO%20Protocol.htm; Wilson, et al., 1993; Colome, et al., 1994). Average indoor CO levels typically vary from 0.5 to 5 ppm (Wilson, et al., 1993). Studies conducted by Wilson and Colome investigated a random sample of residences in California for the purpose of estimating a statewide distribution of indoor CO concentrations. Based on this analysis, the estimated 95$^{th}$ percentile value of 48-hour average CO concentrations in California residences was 5.8 ppm. The estimated 95$^{th}$ percentile value for the maximum 10-minute exposure was 18.6 ppm (Wilson, et al., 1993). These values provide some context for determining when an indoor CO concentration is abnormally high in comparison to average levels.

Transiently elevated CO levels in homes caused by intermittent sources, such as appliances used only occasionally or downdrafting, may be difficult to detect. For example, although average long-term concentrations of CO from gas cooking stoves are not expected to be significant due to their intermittent use, short-term peak CO concentrations up to 120 ppm have been associated with these stoves (EPA, 2000).

---

Gasman II (with digital display and datalogging options that can be set by user) for $510, http://www.ceainstr.com/pdf_datasheets/gasman2_Info.pdf.

**Available Criteria for Comparison to Measured CO Concentrations.** CO hazard levels are typically expressed as airborne concentrations in parts per million (ppm) and duration of exposure. Table 2 shows available standards and guidelines for comparison to measured CO levels, although it should be noted that each of these was created for purposes other than assessing residential CO levels.

**Table 2. Selected Standards and Guidelines[1] for Carbon Monoxide**

| Criterion | Agency & Purpose |
|---|---|
| 9 ppm | ▪ EPA's National Ambient (outdoor) Air Quality Standard – 8-hr average |
| | ▪ World Health Organization's outdoor air limit – 8-hr average |
| ≤ 11 ppm | ▪ Health Canada's Exposure Guideline for Residential Indoor Air – acceptable short-term exposure range, 8-hr average |
| ≤ 25 ppm | ▪ Health Canada's Exposure Guideline for Residential Indoor Air – acceptable short-term exposure range, 1-hr average |
| 30 ppm | ▪ Lowest CO level that UL and CSA allow home CO alarms to display, must not alarm in less than 30 days |
| 35 ppm | ▪ EPA's National Ambient (outdoor) Air Quality Standard – 1-hr average |
| 50 ppm | ▪ OSHA's 8-hr time-weighted average exposure for workers |
| | ▪ EPA's Significant Harm Level for ambient CO per 8 hr time-weighted average |
| 70 ppm | ▪ UL and CSA false alarm resistance point at 60 minutes (1 hr) of exposure |
| | ▪ Level at or above which UL and CSA home CO alarms must go off when exposed for 60-240 minutes (1-4 hrs) |
| 75 ppm | ▪ EPA's Significant Harm Level for ambient CO per 4 hr time-weighted average |
| 125 ppm | ▪ EPA's Significant Harm Level for ambient CO per 1 hr |
| 150 ppm | ▪ Level at or above which UL approved CO alarms must go off within 10-50 minutes of exposure |
| 200 ppm | ▪ NIOSH ceiling concentration for workers at which immediate evacuation is recommended |
| | ▪ (Air free) Level of CO allowed inside water heater flue by ANSI standard |
| 400 ppm | ▪ Level at or above which UL approved CO alarms must go off within 4-15 minutes of exposure |
| | ▪ (Air free) Level of CO allowed inside furnace flue by ANSI standard |
| 800 ppm | ▪ (Air free) Level of CO allowed inside oven flue by ANSI standard |

[1] For comparison: Average indoor CO levels typically vary from 0.5 to 5 ppm (Wilson, et. al., 1993). During smog episodes, atmospheric levels of CO, both indoors and outdoors can climb to 5 to 10 ppm (EPA, 2000).
ANSI = American National Standards Institute; CSA = Canadian Standards Association (refers to CSA Std. 6.16-01); NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; UL = Underwriters Laboratories (refers to UL Std. #2034, Second Edition, dated October 29, 1996, with revisions through June 28, 2002).

*Ambient Carbon Monoxide Standards.* Under the Clean Air Act, EPA issued a CO standard for outdoor air of 9 ppm averaged over 8 hours and 35 ppm averaged over 1 hour (*Federal Register*, August 1, 1994). These National Ambient Air Quality Standards (NAAQS) for outdoor air are intended to be protective for all segments of the population (including sensitive

populations). Areas that fail to meet the NAAQS two or more times in a year must implement special air pollution control measures. These standards are also used as the basis for EPA's Air Quality Index. Ambient CO levels that exceed the Index trigger warnings to those most sensitive to CO: "People with cardiovascular disease, such as angina, should limit heavy exertion and avoid sources of CO, such as heavy traffic." Warnings increase with increasing CO levels. If outdoor CO levels reach three- to five-fold the standard (an extraordinarily unusual finding), Air Quality Index warnings are extended to members of the general public who are advised to avoid heavy exertion. EPA has also defined Significant Harm Levels (SHL) for ambient CO as 50 ppm/8h average, 75 ppm/4h average, and 125 ppm/1h (40 CFR part 51.151). SHL are ambient pollutant concentrations that EPA defines as levels that cause significant and imminent harm to the general public. There is no EPA standard for CO in indoor air.

*Occupational Standards for Carbon Monoxide.* In contrast to EPA's standards that apply to more vulnerable members of the general public, occupational standards and guidelines pertain to healthy adult workers. The Occupational Safety and Health Administration (OSHA) standard for exposure to CO prohibits worker exposure to no more than 50 ppm, averaged over an 8-hour workday (29 CFR 1910.1000, Table Z-1; OSHA, 2002). The National Institute for Occupational Safety and Health (NIOSH) recommends that CO levels to which workers are exposed should not exceed a ceiling concentration of 200 ppm (NIOSH, 1972).

*Carbon Monoxide Alarm Standards.* Although there are no mandatory national standards in place for CO alarms, the quality of CO alarms available for purchase today is greatly influenced by self-imposed industry performance criteria, which provide recommended performance requirements for alarms, as well as general criteria for their construction and testing. The U.S. CPSC recommends that consumers purchase home alarms that meet specifications established by Underwriters Laboratories (UL) 2034 standard for CO detectors/alarms, "Single and Multiple Station Carbon Monoxide Detectors" (UL, 2001) or the Canadian Standards Association CAN/CSA 6.19-01, and the previous International Approval Services IAS 6-96. All three organizations are well respected standards developers and their standards are equally acceptable to the CPSC staff.

The current UL 2034 standard is the second edition, dated October 29, 1996, with revisions through and including June 28, 2002. The UL specifications require alarms to sound before an active individual would experience an estimated dose causing 10% COHb. Because COHb levels are a function of both the level of CO in the air and the duration of exposure, among many other factors, specifications for CO alarms are defined by the CO level in air and the amount of time the level is maintained. As shown in Table 2, under UL Standard 2034 (revised 2001) alarms must sound within 60-240 minutes (1-4 hours) if 70 or more parts per million (ppm) CO are present; within 10-50 minutes if 150 or more ppm CO are present; and within 4-15 minutes if 400 or more ppm are present (UL, 2001). In order to limit false alarms, home alarm specifications also identify levels at which CO alarms must not sound. These performance criteria were established in response to incidents like the one in Chicago in December 1994 in which thousands of home CO alarms sounded simultaneously during a smog episode and the ensuing calls to fire companies overwhelmed the 911 system. Detectors from the early 1990's alarmed at levels of concern for those with cardiovascular disease or other risk factors for CO effects, but, given the unintended consequences on the 911 system, were considered unacceptable for the general population. In response, under the revised UL Standard 2034 (UL,

2001), alarms must be exposed to a minimum of 30 ppm CO for at least 30 days before they may sound (UL, 2001).

These alarm criteria are consistent with the use of CO alarms to warn residents of serious, life threatening levels of CO. These criteria, however, are purposefully not designed to warn of unhealthy ambient conditions addressed by EPA's Air Hazard Index or compliance with occupational standards and ceiling recommendations. Currently manufactured CO alarms that meet the UL standard must not display the CO concentration below 30 ppm, and starting in 2007 will only be required to be accurate within 30 percent of the actual CO concentration. CO alarms are not designed for low-level CO monitoring and are not appropriate for that use. For comparison, Table 2 also shows the American National Standards Institute (ANSI) standards for combustion appliances, which have remained unchanged since they were first established in 1925.

## 3.3 Home Carbon Monoxide Alarms

Exposure to moderate concentrations of CO over several hours can be as dangerous as exposure to higher CO levels for a few minutes. Therefore, while CO alarms are designed primarily to provide early warning of potentially dangerous high-level exposures, they also offer some protection against lower levels by monitoring CO levels over time.

**CO Alarm Technologies and Performance.** There are many different types and brands of CO alarms available on the market today for home use, typically costing about $20 to $60. Alarms that use household current typically employ a solid-state sensor that measures CO on a periodic basis. Battery-powered alarms usually use a passive sensor technology that reacts to prolonged exposure to CO gas. The most common CO sensor technologies include: colorimetric reagent (i.e., biometric or biomimetic) sensors, in which a change in the color of a gel-coated disc sounds an alarm; metal oxide semiconductor (MOS) sensors, which determine CO levels by reaction with a heated metal (tin oxide); and electrochemical cell sensors, in which a chemical reaction with CO creates an electrical current, setting off an alarm. Other established technologies for CO detection, such as gas chromatography, mass spectrometry, ion mobility spectroscopy, and NDIR absorption sensors used for research and professional monitoring, are currently not available for low-cost home use. According to Kwor (2000), colorimetric and metal oxide type sensors dominated the home consumer market until about 1997, when the market share of electrochemical CO alarms began to grow rapidly. The colorimetric alarms tend to have the lowest cost, and the MOS alarms have the longest life (Kwor, 2000). The review by Kwor (2000) concludes that the electrochemical alarms "exhibit the best overall combination of cost and performance" (Kwor, 2000).

A simplified overview of selected properties and the relative performance of the three primary CO sensor technologies currently used in home CO alarms is presented in Table 3. This overview serves as only a very general comparison of sensor technologies available for home use. Research has indicated that brand-to-brand variation in CO alarm performance is not conclusively related to the particular sensing technology used, whether colorimetric, semiconductor or electrochemical (Clifford and Siu, 1998; Kwor, 2000). All three sensor technologies are capable of meeting UL requirements. There is no recommendation as to which technology the alarm must use to meet the standard.

## Table 3. Selected Properties of the Primary Sensor Technologies for Residential CO Alarms

| Sensor Property | General Performance of Sensor Type [a] | | |
|---|---|---|---|
| | **Colorimetric** | **Metal Oxide Semiconductor** | **Electrochemical** |
| ***Basic Operation Principles*** | | | |
| | Gel-coated discs darken in the presence of CO; color change sounds an alarm | Heated metal (tin oxide) reacts with CO to determine the levels of CO; must connect to house power | Chemical reaction with CO creates an electrical current, setting off an alarm |
| ***Durability*** | | | |
| **Lifetime** | > 5 years (Data being collected) | 5 - 10 years | > 5 years (Data being collected) |
| **Short-term stability** | Unknown; difficult to assess | Fair | Good |
| ***Performance*** | | | |
| **Resolution and Accuracy [b]** | Fair | Fair | Good |
| **Sensitivity drift** | Unknown | Moderate | Moderate |
| **Response time** | Fair | Fair | Good |
| **Immunity to false alarms [c]** | Fair | Good | Good |
| **Immunity to false negatives [d]** | Good | Good | Good |
| **Temperature and humidity dependence** | Fair | Fair | Good (humidity)<br>Fair (temperature) |
| **Selectivity [e]** | Good | Good | Good |
| **Immunity to poisoning [f]** | Good | Good | Good |
| ***Consumer Preferences*** | | | |
| **Power consumption** | Low | High | Low |
| **Sensor cost** | Low | Low | Low |
| **Primary advantages** | Simple, lowest cost | Long-life, extensive performance data available (longest history of field usage) | Reasonable cost, low power consumption, good performance |
| **Primary disadvantages** | Interference (temperature, humidity, other gases), difficult to reset quickly after CO exposure, rarely equipped with digital displays, early models had shorter lifetimes | High input power, interference (temperature, humidity), inaccuracy | Temperature and humidity dependence, lack of long-term sensitivity data (relatively recently developed technology) |

[Table adapted from Kwor, 2000 and Clifford and Dorman, 1996]
[a] There are commercial alarms available that meet UL-2034, CAN/CGA-6.19-M93 and CAN/CSA 6.19-01 requirements for all three sensor technologies. The U.S. Consumer Product Safety Commission (CPSC) recommends that consumers purchase and use an alarm that meets the latest requirements of the UL or CAN/CSA or CGA standard. There is no recommendation as to which technology the alarm must use to meet the standard. [b] Resolution and accuracy = reflects the detection limit and how close the measured value is relative to the true CO level. [c] False alarm = detector alarms even though CO level is low. [d] False Negative = detector fails to alarm when CO level is high. [e] Selectivity = ability to distinguish between CO and other gases. [f] Immunity to poisoning = resistance to interference from other substances or pollutants in indoor air.

UL specifications for CO alarms address not only CO exposures that trigger alarms, but also problems with interference and concerns about reliability. Because many alarm technologies are also susceptible to interference from pollutants commonly found in indoor environments, UL 2034 standards specify minimum allowable interference levels for methane, butane, heptane,

ethylacetate, isopropyl alcohol, carbon dioxide, ammonia, ethanol, toluene, trichlorethylene and acetone. Two recent studies identified some problems with CO alarms not meeting the UL specifications for sensitivity and selectivity (Clifford and Siu, 1998; Kramer and Tikalsky, 2000), but there is no widespread indication of problems with CO alarms fulfilling their intended use – to warn of potentially dangerous CO levels, generally above 70 ppm. With respect to maintenance of long-term performance, UL specifications require certain levels of performance for 3000 hours of operation.

Longer-term field evaluations of CO alarms (models available at the end of 1999), including sensitivity testing over time, are ongoing by organizations such as UL. Preliminary results of UL research suggest that, regardless of the sensor technology used, most alarms perform within UL standards, and all provide effective signaling protection (Patty, 2001, personal conversation; Moloney, 2001). Based on current information, the major questions that remain unanswered concern how long CO alarms are actually remaining in use in the field and the performance characteristics for different types of CO alarms after several years of use.

**Protocols for CO Alarm Response and Evaluation.** If a CO alarm sounds, most residents call their local emergency service or utility company for assistance. Professionals who respond to CO alarms generally use field monitors that feature digital displays of CO levels to investigate the cause of the alarm sounding and to advise residents about the hazard posed by the levels of CO found. With no generally recognized standards for acceptable levels of CO in indoor air, advice to residents often reflects the professional judgment of the individual responding to the alarm.

Guidelines and protocols for responding to CO alarms have been developed by several groups including federal agencies (CPSC, 2003a; see Appendix A), trade associations (e.g., Building Performance Institute or BPI), municipal first response teams, and private industry (Scott Instruments, 2002). Although not identical, these protocols share many similarities. CPSC recommends that, in the event of a CO alarm activation, the residents should go outdoors or to a neighbor's house immediately and not ventilate the house (to allow for identification of the CO source), unless someone is unconscious or cannot leave. Emergency responders (911) should be called immediately. As with any hazardous substance emergency, first responders need to follow protocols for safe entry once on the scene. Field instruments, as well as visual clues and occupant survey, are used to evaluate conditions.

# 4.0 METHODS USED TO MITIGATE CARBON MONOXIDE HAZARDS IN THE HOME

Intervention methods for prevention of residential CO poisoning include:

- Education and outreach to consumers about CO poisoning symptoms and CO source control (including safe behaviors and proper maintenance of combustion appliances).
- Education and outreach to professionals about home CO poisoning, including symptoms and correct observation of home conditions that pose potential or actual CO hazards.
- Installation of home CO alarms and implementation of standard protocols for alarm response.

- Installing ventilation for, or improving existing ventilation of, combustion appliances.
- Replacing combustion with non-combustion appliances.

## 4.1 Education and Outreach to Home Occupants

Education of home occupants regarding the potential sources of CO, actions to take to avoid CO exposure, and the proper response to a suspected problem are primary means of reducing CO hazards in the home. For example, as most acute CO poisoning episodes occur from exposure to automobile exhaust, residents need to be educated about the risk posed by starting and idling vehicles in unvented garages attached to the home. This risk can be substantially reduced by installing continuous mechanical exhaust ventilation (an exhaust fan) as required by Section 403.3 of the International Mechanical Code.

In an effort to reduce injury and deaths associated with CO poisonings, numerous federal agencies (EPA, CDC, CPSC, USDA) as well as private and non-profit groups, have created educational materials to increase awareness of the symptoms and sources of CO poisoning. Commonly recommended points for homeowner education regarding CO hazards and behavior include those focusing on proper use and maintenance of propane, natural gas, or charcoal equipment (appliances, grills, generators, etc.) or any gasoline-powered tool; safety where attached garages are present; the importance of regularly cleaning the clothes dryer and other ductwork and checking outside vent covers for blockages; proper use, placement, and maintenance of CO alarms; and the need to have all gas or other fuel-burning appliances and chimneys inspected and maintained regularly by a professional.

Other outreach efforts include timely press releases and reminders issued by CPSC to consumers about CO hazards posed from bringing grills, hibachis, and gasoline-powered generators into the home or garage during storms or other events that cause power outages.

## 4.2 Education and Outreach to Professionals

In addition to home occupants, the education of health care providers (e.g., visiting nurses), professionals who respond to CO alarms or conduct home inspections (e.g., utility company inspectors), and professionals who service combustion appliances (e.g., heating contractors) regarding home CO poisoning is also essential. These groups can both serve as effective conveyors of risk information to home occupants and serve important roles in diagnosing CO problems in a home, for example, through early recognition of symptoms of CO poisoning in home occupants or correct observation of home conditions that pose potential or actual CO hazards.

**Outreach to Health Care Providers about CO Poisoning Symptoms and Prevalence.** Studies on the misdiagnosis of patients with CO poisoning demonstrate the need for outreach to medical professionals on CO poisoning (Comstock et al., 1999). Because of the relatively non-specific flu-like symptoms of CO poisoning (e.g., headache, nausea, lethargy, confusion, dizziness, agitation, etc.), it often may be misdiagnosed (Comstock et al., 1999). For example, in an investigation of 34 (45%) of 75 manufacturing plant employees that experienced symptoms of CO poisoning (primarily headaches) while at work, failure to diagnose illness

correctly in the first employees evaluated resulted in some CO-intoxicated employees being sent back to work and further exposure and in continued exposures to other workers at the plant (Comstock et al., 1999). Of ten ill employees evaluated at three local emergency departments, CO poisoning was initially diagnosed (and then later dismissed as erroneous) in only three workers (Comstock et al., 1999). Grand rounds, i.e., lectures at hospitals for physicians and others, have been an effective tool for educating medical providers about other environmental health hazards. Reliable data on the prevalence of CO-related morbidity could also be a useful tool for demonstrating the need for medical providers to educate themselves about the dangers of CO.

**Outreach to Home Inspection Professionals: Guidance on Assessing Appliance, Ventilation, and Backdrafting Problems.** Numerous organizations, such as the CPSC and the Canada Mortgage and Housing Corporation (CMHC), as well as several commercial organizations, provide guidance to professionals who conduct overall CO investigations in the home, including emergency response and routine preventive inspections. In November 2003, CPSC published the following guidance, "Responding to Residential Carbon Monoxide Incidents: Guidelines for Fire and Other Emergency Response Personnel" (see Appendix A). Other examples include the Building Performance Institute's (BPI) draft protocol, "Carbon Monoxide Analyst Protocol," (http://home.att.net/~cobusters1/coprotocol.htm) and R.J. Karg Associates' "Chicago Protocol: A Protocol for the Testing of Carbon Monoxide Emissions" (http://www.karg.com/CO%20Protocol.htm).

It is reported that few of the currently available CO inspection protocols recommend measuring CO levels inside an attached garage as part of a CO investigation in the home (Donnay, 2005). If elevated levels of CO are found in an attached garage during an investigation, this suggests a vehicle in the garage as a source for elevated CO levels in the home.

According to the CPSC, inspections of homes by a professional (e.g., heating contractor or Gas Company) should include a careful look at the following sources of CO:

- *Furnaces, water heaters, boilers, and stoves.* If they burn natural gas, heating oil, wood or other kinds of fuel, these appliances are potential sources of CO. Typical appliance (e.g., furnace, stove, fireplace) problems that cause the release of CO in homes, many of which are hard for a homeowner to identify, include: cracked heat exchangers; insufficient air for proper combustion; and maladjusted burners.
- *Chimneys, flues, and vents.* Flues and chimneys should be inspected before each heating season for leakage and for blockage by creosote or debris. Creosote buildup or leakage could cause black stains on the outside of the chimney or flue. These stains can mean that pollutants are leaking into the house. (Specific methods for assessing backdrafting are described in Section 2.0). All vents to furnaces, water heaters, or boilers should be checked to make sure they are not blocked, loose, or disconnected. Snow and ice also create the potential for vent blockages. Owners and residents should know where all of their vents exhaust and be aware of those areas where heavy snow or ice can impact proper operation.

- *Improper ventilation.* Fuel burning appliances require adequate ventilation. A supply of fresh air is important to help carry pollutants up the chimney, stovepipe, or flue, and is necessary for the complete combustion of any fuel.
- *High Temperature Plastic Venting (HTPV) pipes.* Consumers should have the vent pipes on their natural gas or propane heating systems inspected for the presence of HTPV pipes. The HTPV pipes could crack or separate at the joints and leak CO into the home. In 1998, virtually the entire furnace and boiler industry, together with the manufacturers of HTPV pipes, joined with CPSC to announce a vent pipe recall program.

Another target area for professional assessment is the potential for backdrafting problems, particularly in tight homes that are especially susceptible to backdrafting due to house depressurization. If a backdrafting problem is suspected, a professional heat contractor should check the house and heating systems. Small temperature-sensitive strips called "Backdraft Indicators" can be attached to the draft diverter (which regulates the flow of air in HVAC systems) to detect backdrafting of exhaust gases (ISU Extension Publication, 1996). A chimney flow test may also be conducted by holding a smoke indicator (such as an incense stick) near the draft hood of a gas furnace or water heater, and watching the direction of smoke movement at the draft hood or damper, both with and without exhaust fans and other exhaust equipment in the house turned on (CMHC, 1999). If the smoke moves into the house, a spillage problem may be present. Ways to reduce house depressurization (i.e., reducing indoor and outdoor pressure differences) include shutting off exhaust fans or avoidance of running several simultaneously, sealing return ducts or closing return registers in the basement, opening supply registers in the basement, opening doors between rooms, closing fireplace dampers, and where a furnace or water heater is enclosed in a small separate room, allowing air to move freely between the furnace room and the rest of the house. The only fail-proof way to eliminate backdrafting of combustion gases, however, is to install direct vent appliances that do not have open draft diverters.

Various guidance documents with suggested protocols for conducting safety testing of combustion appliances, including spillage and CO emissions, have been developed, including:

- ASTM Standard E1998-99, "Standard Guide for Assessing Depressurization-Induced Backdrafting and Spillage from Vented Combustion Appliances";
- Section H of the National Fuel Gas Code (ANSI Z223.1/NFPA 54);
- ASHRAE 62.2 Appendix A, Checking the Venting of Combustion Appliances; and
- Canada General Standards Board- 51.71-95, "The Spillage Test Method to Determine The Potential for Pressure Induced Spillage from Vented, Fuelfired, Space Heating Appliances, Water Heaters and Fireplaces";
- Iowa State University, Agricultural and Biosystems Engineering Extension, provides numerous factsheets on combustion appliance inspection, as well as other information on CO hazards (see, for example, "Carbon Monoxide Poisoning - Checking for Complete Combustion," ISU Extension Pub # AEN-175, available at http://www.abe.iastate.edu/human_house/aen175.asp).

## 4.3 Carbon Monoxide Alarms

Along with regular inspection of combustion appliances, properly working CO alarms can provide home occupants with warning when indoor CO levels reach dangerous levels. For example, in a study of unintentional CO poisoning deaths in New Mexico (1980 through 1995), Yoon et al. (1998) found that 49% of residential CO deaths occurred when the occupants were sleeping, and estimated that (of the victims without the presence of alcohol in their blood) approximately half (78) of the deaths could have been prevented if audible CO alarms were used and functioned properly. Research by the Home Safety Council indicates that only 35 percent of American homeowners had a CO detector in their home (Home Safety Council, 2003). Donnay (2005) however, questions whether the UL CO alarm standards are protective enough (e.g., in light of NIOSH recommendations for workers that recommend immediate evacuation of workers above 200 ppm), especially for sensitive groups such as those with cardiac and pulmonary health problems, and pregnant women.

Clifton et al. (2001), using a novel method that involved analysis of national media clipping data, studied CO exposures in the US and the role of CO detectors in prevention of CO-related deaths. Comparing nonfatal outcomes attributable to the presence of CO detectors and case fatality rates among cities with and without CO detector ordinances, the researchers found that cities with CO detector ordinances showed lower case fatality rates as reported in the media than those cities without ordinances ($P < .001$). There were 1,008 (24.2%) survivors who attributed their survival to the presence of a CO detector. The authors also note that despite its limitation, the use of a media clipping service may provide insight into CO poisoning demographics. Similarly, the CDC, in coordination with local emergency physicians and fire department authorities, conducted an investigation of the effectiveness of a CO alarm ordinance in Mecklenburg County, North Carolina (2002 population: 722,367) after a 2002 ice storm caused 78.9% of county households to lose power (CDC/MMWR, 2004). The ordinance, adopted in September 2000, requires a CO alarm in the majority of residences; all-electric residences without attached garages (35.4% of all homes) were exempt. The CDC investigation found that of the 124 cases of symptomatic CO poisoning reported over the next 9 days, 96.2% of the severe poisonings occurred in homes with no reported functioning CO alarm. As a result of these findings, on October 8, 2003, Mecklenburg County officials amended the ordinance to require alarms with battery back-ups in all residences. The CDC researchers note that officials in other communities should consider enacting such alarm ordinances to prevent CO poisonings (CDC/MMWR, 2004).

CPSC recommends that all homes have one CO alarm that meets the requirements of UL 2034, IAS 6-96, or CAN 6-19-01 installed in the hallway near every separate sleeping area of the home. CO alarms should be installed according to the manufacturer's instructions (e.g., alarms should not be covered by furniture or draperies). For earlier warning, some gas utilities (e.g., Baltimore Gas & Electric in Maryland), recommend installing additional CO alarms in and any area with a fireplace, furnace or fuel-burning appliance, or in an attached garage[3].

[3] BG&E Service Express newsletter, Dec 2000, "Protecting Yourself Against Carbon Monoxide Poisoning."

# 5.0 CURRENT RESEARCH AND INFORMATION GAPS

### Prevalence of CO poisoning

While detailed information is available on CO-related fatalities, no nationwide studies have been conducted to determine the prevalence of elevated CO exposures in the general population. Topics of consideration for future research in this area include:

- Current CO poisoning prevalence among groups with greater sensitivities to CO;
- Current CO poisoning prevalence among patients with flu-like symptoms.

### CO Sources, Exposures and Health Effects

The following are some topics on which additional research would be of value to better understand and ultimately, further reduce, current residential exposures.

- Research on health effects associated with chronic exposures to low levels of CO or intermittent exposures to medium or higher levels of CO;
- Research regarding current CO exposure models and accurate prediction of high-level and low-level CO exposures;
- Research on the contribution of nonambient sources to total human exposure to CO;
- Data on actual CO levels founds in homes with various types of CO sources (to better inform HHI grantees about the range of CO levels they may encounter);
- Investigative information after combustion appliance failures and other CO poisoning accidents (i.e., the source of CO, the reason(s) CO entered the structure, the health outcome of the exposure, measures needed to correct the problem, the magnitude of the problem);
- Research/survey information on the prevalence of excessive CO emissions from combustion equipment and the primary cause of failure (i.e., is it improper design, installation, maintenance, or use?);
- Information on the frequency and cause of vent failure (e.g., failure of the owners to have the vent maintained, failure of professionals to inspect and repair, etc?);
- Cost-effective options for venting currently unventilated garages and kitchens and information on the benefits of such ventilation; and
- Research into the incidence and severity of delayed neurological sequelae in individuals with confirmed CO poisoning.

### Home CO Alarms

- Evaluation of the overall performance of CO alarms, including reliability both at the time of purchase and throughout their lifetime;
- Evaluation of the performance of CO alarms in response to cumulative lifetime exposures to other indoor air contaminants that may compromise their functioning;
- Evaluation of the length of time consumers should retain a CO alarm;
- Continued research into various sensing technologies that may be employed in CO alarms;
- Cost-benefit analyses of CO alarm use and other intervention options from a public health program perspective.

# References


Abelsohn, A., Sanborn, M.D., Jessiman, B.J., Weir, E. 2002. Identifying and managing adverse environmental health effects: 6. Carbon monoxide poisoning. CMAJ. 166(13): 1685-1690.

American Gas Association. Fact Sheet: Combustion Emissions for Gas Ranges. Arlington, VA. http://www.aga.org

Benignus, V.A., Muller, K.E., Malott, C.M. 1990. Dose-effects functions for carboxyhemoglobin and behavior. Neurotoxicol Teratol. 12(2):111-8.

Broder, J., Mehrotra, A., and J. Tintinalli. 2005. Injuries from the 2002 North Carolina ice storm, and strategies for prevention. Injury. 36(1): 21-6.

Burch, S.D., Keyser, M.A., Colucci, C.P., Potter, T.F., Benson, D.K., Biel, J.P.. 1996. Applications and Benefits of Catalytic Converter Thermal Management, Presented at SAE Fuels & Lubricants Spring Meeting (Dearborn, MI), May 7, 1996. Available online at: http://www.nrel.gov/vehiclesandfuels/energystorage/pdfs/saecatl.pdf

CAN/CSA-6.19-01 Residential Carbon Monoxide Alarming Devices. Canadian Standards Association, Mississauga, Ontario, CN (1999). Available at www.csa.ca

CDC/MMWR. 2005a. Unintentional non-fire-related carbon monoxide exposures – United States, 2001-2003. Morbidity and Mortality Weekly Report, Jan 21, 2005. 54(2): 36-9. Centers for Disease Control and Prevention.

CDC/MMWR. 2005b. Early Release: Carbon Monoxide Poisoning After Hurricane Katrina --- Alabama, Louisiana, and Mississippi, August–September 2005. Early Release September 30, 2005 / Vol. 54 / http://www.cdc.gov/mmwr/preview/mmwrhtml/mm54e930a1.htm

CDC/MMWR. 2005c. Carbon monoxide poisoning from hurricane-associated use of portable generators--Florida, 2004. Morbidity and Mortality Weekly Report, July 22, 2005. 54(28): 697-700. Centers for Disease Control and Prevention.

CDC/MMWR. 2004. Use of carbon monoxide alarms to prevent poisonings during a power outage--North Carolina, December 2002. Morbidity and Mortality Weekly Report, March 12, 2004. 53(9): 189-92. Centers for Disease Control and Prevention.

CDC/MMWR. 1997. Use of Unvented Residential Heating Appliances – United States, 1988-1994. Morbidity and Mortality Weekly Report, December 26, 1997. 46(51): 1221-1224. Centers for Disease Control and Prevention.

CDC/MMWR. 1996. Deaths from Motor-Vehicle Related Unintentional Carbon Monoxide Poisoning — Colorado, 1996, New Mexico, 1980–1995, and United States, 1979–1992. Morbidity and Mortality Weekly Report, November 29, 1996. 45 (47): 102. Centers for Disease Control and Prevention.

Clifford, P.K. and D.J. Siu. 1998. Performance Testing of Residential CO Detectors. Prepared for Gas Research Institute, Chicago, IL.

Clifford, P.K. and M.G. Dorman. 1996. Test Protocols for Residential Carbon Monoxide Alarms, Phase I. Prepared by Mosiac Industries for Gas Research Institute, Chicago, IL.

Clifton, J.C. 2nd, Leikin, J.B., Hryhorczuk, D.O., and E.P Krenzelok. 2001. Surveillance for carbon monoxide poisoning using a national media clipping service. Am J Emerg Med. 2001 Mar; 19(2): 106-8.

CMHC. 1998. The Clean Air Guide: How to Identify and Correct Indoor Air Problems in Your Home. Canada Mortgage and Housing Corporation.

CMHC. 1999. Combustion Gases in Your Home: Things you should know about combustion spillage. 7pp. Canada Mortgage and Housing Corporation. http://www.cmhc-schl.gc.ca/

*Code of Federal Regulations.* Revised as of July 1, 1998. Occupational Safety and Health Standards, Subpart Z--Toxic and Hazardous Substances, Table Z-1. Limits for Air Contaminants. 29 CFR 1910.1000.

Colome, S.D., Wilson, A.L. and T. Yian. 1994. California residential indoor air quality study. Volume 2. Carbon monoxide and air exchange rate: an univariate and multivariate analysis. Chicago, Il. Gas Research Institute. GRI-93/0224.3.

Comstock, R.D., et al. 1999. Carbon Monoxide Poisoning Associated with Use of LPG-Powered (Propane) Forklifts in Industrial Settings -- Iowa, 1998. Morbidity and Mortality Weekly Report, December 17, 1999. 48 (49): 1121-1124.

CPSC. 2004. Non-Fire Carbon Monoxide Deaths Associated with the Use of Consumer Products: 2001 Annual Estimates. U.S. Consumer Product Safety Commission, Division of Hazard Analysis, May 13, 2004.

CPSC. 2003. Non-Fire Carbon Monoxide Deaths Associated with the Use of Consumer Products: 1999 and 2000 Annual Estimates. U.S. Consumer Product Safety Commission, Division of Hazard Analysis. July 31, 2003.

CPSC. 2003a. Responding to Residential Carbon Monoxide Incidents: Guidelines for Fire and Other Emergency Response Personnel. U.S. Consumer Product Safety Commission, November 2003. Access: www.cpsc.gov/library/foia/foia04/os/resident.pdf

CPSC. 2001. Non-Fire Carbon Monoxide Deaths Associated with the Use of Consumer Products: 1998 Annual Estimates. U.S. Consumer Product Safety Commission, Division of Hazard Analysis. Bethesda, MD.

CPSC. 1999. CO Poisoning. *In:* U.S. Consumer Product Safety Commission, Consumer Product Safety Review. Fall 1999, Vol. 4, No. 2. http://www.cpsc.gov/cpscpub/pubs/cpsr_nws14.pdf

Cunnington, A.J., and P. Hormbrey. 2002. Breath analysis to detect recent exposure to carbon monoxide. Postgrad Med J. 2002 Apr; 78(918): 233-7.

Devine, S.A., Kirkley, S.M., Palumbo, C.L., and R.F. White. 2002. MRI and neuropsychological correlates of carbon monoxide exposure: A case report. Environmental Health Perspectives. 110(10): 1051-1055.

Donnay, A. 2005. Personal communication. November 9, 2005.

Donnay, A. 2003. Carbon Monoxide Exposure and Carboxyhemoglobin (letter). Environmental Health Perspectives. 111(10): A511-2; author reply A512. http://ehpnet1.niehs.nih.gov/docs/2003/111-10/correspondence.html

Dutton, S.J., Hannigan, M.P., and S.L. Miller. 2001. Indoor pollutant levels from the use of unvented natural gas fireplaces in Boulder, Colorado. J. Air & Waste Management Assoc. 51:1654-1661.

EPA. 2000. Air Quality Criteria for Carbon Monoxide. U.S. Environmental Protection Agency, National Center for Environmental Assessment. June, 2000. EPA 600/P-99/001F.

Estrella, B., Estrella, R., Oviedo, .J, Narvaez, X., Reyes, M.T., Gutierrez, M., and E.N. Naumova. 2005. Acute respiratory diseases and carboxyhemoglobin status in school children of Quito, Ecuador. Environ Health Perspect. 113(5):607-11.
*Federal Register*. 1994. National ambient air quality standards for carbon monoxide – final decision. 59 FR 38:906. August 1, 1994.

Ghim, M., and H.W. Severance. 2004. Ice storm-related carbon monoxide poisonings in North Carolina: a reminder. South Med J. 97(11): 1060-5.

Greiner, T.H. and C.V. Schwab. 2000. Approaches to Dealing with Carbon Monoxide in the Living Environment. In: Carbon Monoxide Toxicity (D.G. Penney, ed.). CRC Press, Boca Raton, FL.

Greiner, T.H. and C.V. Schwab. 1998. Carbon Monoxide Exposure from a Vehicle in a Garage. Thermal Performance of the Exterior Envelopes of Buildings VII. December 1998, Florida.

Hampson, N.B. 2000. Carbon Monoxide Poisoning and Its Management in the United States. In: Carbon Monoxide Toxicity. (D.G. Penney, ed.) CRC Press, Boca Raton, FL.

Hampson, N.B. and J.L. Zmaeff. 2005. Carbon monoxide poisoning from portable electric generators. Am J Prev Med. 28(1): 123-5.

Home Safety Council. 2003. *Press Release*: Carbon Monoxide (CO) Poses Silent And Serious Threat For Families During Winter Months -- Home Safety Council Research Shows Only 35 Percent Of American Homes Are Protected By CO Detector. October 17, 2003. http://www.homesafetycouncil.org/media/media_w023.aspx

Hwang, B.F., Lee, Y.L., Lin, Y.C., Jaakkola, J.J., and Y.L. Guo. 2005. Traffic related air pollution as a determinant of asthma among Taiwanese school children. Thorax. 60(6):467-73.

ICC. 2003. 2003 International Mechanical Code. International Code Council. http://www.iccsafe.org/e/prodshow.html?prodid=3300L03

International Approval Services - U.S., IAS U.S. Requirements for Carbon Monoxide Alarms for Residential Use, Second Edition, June 1, 1998, No. 6-96, International Approval Services - U.S., Cleveland, Ohio.

ISU Extention. 1998. Carbon Monoxide Poisoning: Garages. Iowa State University Extension Publication # AEN-207. Author: Dr. T. Greiner, Dept. of Agricultural and Biosystems Engineering. Access: http://www.abe.iastate.edu/human_house/aen207.asp

ISU Extension. 1996. Carbon Monoxide Poisoning: Downdrafting (Backdrafting). Iowa State University Extension Publication # AEN-165. Author: Dr. T. Greiner, Dept. of Agricultural and Biosystems Engineering. Access: http://www.abe.iastate.edu/human_house/aen165.asp

ISU Extension. 1997. Carbon Monoxide Poisoning - Checking for Complete Combustion. Iowa State University Extension Publication ISU Extension Pub # AEN-175. Author: Dr. T. Greiner, Dept. of Agricultural and Biosystems Engineering. Access: http://www.abe.iastate.edu/human_house/aen175.asp

Knobeloch, L. and R. Jackson. 1999. Recognition of chronic carbon monoxide poisoning. WMJ. 98(6):26-9.

Kramer, J.M. and S.M. Tikalsky. 2000. Carbon Monoxide Response Survey Analyses: Final Supplement 1994-99. Prepared by Resource Strategies, Inc. for Gas Research Institute. Chicago, IL. GRI-99/0238.

Kwor, R. 2000. Carbon Monoxide Detectors. In: Carbon Monoxide Toxicity (D.G. Penney, ed.). CRC Press, Boca Raton, FL.

Lanphear, B.P., Aligne, C.A., Auinger, P., Weitzman, M., and R.S. Boyd. 2001. Residential exposures associated with asthma in US children. Pediatrics. 107(3): 505-511

Liu, S. Krewski, D., Shi, Y, Chen, Y, and R.T. Burnett. 2003. Association between gaseous ambient air pollutants and adverse pregnancy outcomes in Vancouver, Canada. Environmental Health Perspectives. 111:1773-1778.

Lucas, GHW. 1953. Symptoms and Treatment of Acute Poisoning. New York: MacMillan Company, p 124.

Moloney, S. 2001. Carbon Monoxide Alarm Field Study (Summary). Underwriters Laboratories, Inc., October 29, 2001.

Mott, J.A., Wolfe, M.I., Alverson, C.J., Macdonald, S.C., Bailey, C.R., Ball, L.B., Moorman, J.E., Somers, J.H., Mannino, D.M., and S.C. Redd. 2002. National Vehicle Emissions Policies and Practices and Declining US Carbon Monoxide-Related Mortality. JAMA 288(8); 988-995.

Nagda, N.L., Koontz, M.D., Billick, I.H., Leslie, N.P. and D.W. Behrens. 1996. Causes and consequences of backdrafting of vented gas appliances. Journal of the Air & Waste Management Association. 46: 838-846.

NHTSA. 2000. Fatalities Associated With Carbon Monoxide Poisoning From Motor Vehicles, 1995-1997. Research Note, April 2000. National Highway Traffic Safety Administration, Department of Transportation. http://www-nrd.nhtsa.dot.gov/pdf/nrd-30/NCSA/RNotes/2000/CO_FINAL.pdf

NIOSH. 1972. Criteria for a Recommended Standard: Occupational Exposure to Carbon Monoxide. National Institute for Occupational Safety and Health (NIOSH), CDC, DHHS. DHHS (NIOSH) Publication No. 73-11000.

Norris, G., Young-Pong, S.N., Koenig, J.Q., Larson, T.V., Sheppard, L. and J.W. Stout. 1999. An association between fine particles and asthma emergency department visits for children in Seattle. Environ Health Perspect. 107: 489-493.

Omaye, S.T. 2002. Metabolic modulation of carbon monoxide toxicity. Toxicology. 180:139-150.

OSHA. 2002. OSHA Fact Sheets: Carbon Monoxide Poisoning. Occupational Safety & health Administration, U.S. Department of Labor. Access (October, 2004): http://www.osha.gov/OshDoc/data_General_Facts/carbonmonoxide-factsheet.pdf

Patty, P. 2001 and 2002. Personal communication with Paul Patty of Underwriters Laboratories (UL). Northbrook, IL. July, 27, 2001 and January 9, 2002.

Penney, D.G. 2001. Carbon Monoxide Poisoning: The Essentials of Carboxyhemoglobin (from Carbon Monoxide Headquarters online). Updated01/17/02. Access http://coheadquarters.com/COHb5.htm.

Penney, D.G. 2000. Chronic Carbon Monoxide Poisoning. In: Carbon Monoxide Toxicity. (D.G. Penney, ed.) CRC Press, Boca Raton, Fl.

Ralston, J.D. and N.B. Hampson. 2000. Incidence of severe unintentional carbon monoxide poisoning differs across racial/ethnic categories. Public Health Reports. 115:46-51. U.S. Department of Health and Human Services.

Raub, J.A. and V.A. Benignus. 2002. Carbon monoxide and the nervous system. Neurosci Biobehav Rev. 26(8):925-40.

Raub, J.A., Mathieu-Nolf, M., Hampson, N.B., and S.R. Thom. 2000. Carbon monoxide poisoning – a public health perspective. Toxicology 145:1-14.

Raub, J.A.. 2000. The Setting of Health-Based Standards for Ambient Carbon Monoxide. In: Carbon Monoxide Toxicity (D.G. Penney, ed.). CRC Press, Boca Raton, FL.

Ritz, B. and F. Yu. 1999. The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. Environmental Health Perspectives. 107: 17-25.

Runyan, C.W., Johnson, R.M., Yang, J., Waller, A.E., Perkis, D., Marshall, S.W., Coyne-Beasley, T., and K.S. McGee. 2005. Risk and protective factors for fires, burns, and carbon monoxide poisoning in U.S. households. Am J Prev Med. 28(1): 102-8.

Ryter, S.W., Morse, D., Choi, A.M. 2004. Carbon monoxide: to boldly go where NO has gone before. Sci STKE. 2004(230):RE6.

Sanchez, R., Fosarelli, P., Felt, B., Greene, M., Lacovara, J., Hackett, F. 1988. Carbon monoxide poisoning due to automobile exposure: Disparity between carboxyhemoglobin levels and symptoms of victims. Pediatrics. 82(4): 663-666.

Sayers, R.R. and W.P. Yant. 1923. Dangers and Treatment of Carbon Monoxide Poisoning. Reports of Investigations, Serial No. 2476. Washington, DC: Department of the Interior, Bureau of Mines.

Scott Instruments. 2002. CO Hazards: a First Response Guide. Scott Instruments, Exton, PA. Available online at http://www.scottinstruments.com/public/library.cfm

Sheppard, L., Levy, D., Norris, G., Larson, T.V. and J.Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology. 10:23-30.

Slack, H.H. and M.A. Heumann. 1997. Use of unvented residential heating appliances – United States, 1988-1994. Morb. Mortal. Wkly. Rpt. 46:1221-1224.

Thom, S.R., Ohnishi, S.T., Fisher, D., Xu, Y.A., Ischiropoulos, H. 1999. Pulmonary vascular stress from carbon monoxide. Toxicol Appl Pharmacol. 154(1):12-9.

Thom, S.R., Xu, Y.A., Ischiropoulos, H. 1997. Vascular endothelial cells generate peroxynitrite in response to carbon monoxide exposure. Chem Res Toxicol. 10(9):1023-31.

Townsend, C.L. and R.L. Maynard. 2002. Effects on health of prolonged exposure to low concentrations of carbon monoxide. Occup Environ Med. 59: 708-711.

Tsongas, G. 1995. Carbon Monoxide From Ovens: A Serious IAQ Problem. Home Energy Magazine Online, September/October 1995. Access: http://hem.dis.anl.gov/eehem/95/950907.html#95090701

UL. 2002. (Update). Underwriters Laboratories (UL) 2034 Standard for Single and Multiple Station Carbon Monoxide Alarms, Second Edition, October, 1996. ISBN 0-7629-274-9 [First Edition April 1992, Second Edition October 29, 1996, revised October 15, 1997 and June 28, 2002]. Underwriters Laboratory, Inc. Northbrook, IL.

Varon, J., and P.E. Marik. 1997. Carbon Monoxide Poisoning. The Internet Journal of Emergency and Intensive Care Medicine. Access: www.ispub.com

Vreman, H.J., Wong, R.J., and D.K. Stevenson. 2000. Carbon Monoxide in Breath, Blood, and Other Tissues. In: Carbon Monoxide Toxicity (D.G. Penney, ed.). CRC Press, Boca Raton, FL.

Wilson, A.L., Colome, S.D., and Y. Tian. 1993. California residential indoor air quality study. Volume I: methodology and descriptive statistics. Appendices. Chicago, Il. Gas Research Institute. GRI-93/-224.2.

WHO. 1999. Carbon Monoxide. Environmental Health Criteria #213. Geneva: World Health Organization. http://whqlibdoc.who.int/ehc/WHO_EHC_213.pdf

Yang Q, Chen Y, Krewski D, Burnett RT, Shi Y, McGrail KM. 2005. Effect of short-term exposure to low levels of gaseous pollutants on chronic obstructive pulmonary disease hospitalizations. Environ Res. 99(1):99-105. Epub 2004 Nov 21.

Yoon, S., Macdonald, S., Parrish, G. 1998. Deaths from unintentional carbon monoxide poisoning and potential for prevention with carbon monoxide detectors. JAMA. 279(9): 685-687.

Yu, O., Sheppard L, Lumley T, Koenig JQ, Shapiro GG. 2000. Effects of ambient air pollution on symptoms of asthma in Seattle-area children enrolled in the CAMP study. Environ Health Perspect 108:1209-1214.

# Appendix A. Additional Internet Resources

In addition to the references and links appearing in the reference list above, the following table provides selected links with additional information on carbon monoxide and associated issues.

| Sponsoring Organization/Topic | Internet Web Site Address |
|---|---|
| American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. | http://www.ashrae.org/ |
| Canada Mortgage and Housing Corporation (Healthy Housing & Sustainability Projects) (Combustion Gases Link) | http://www.cmhc-schl.gc.ca/en/index.cfm (http://www.cmhc-schl.gc.ca/en/imquaf/hehosu/index.cfm) (http://www.cmhc-schl.gc.ca/en/burema/gesein/abhose/abhose_ce02.cfm) |
| Canadian Standards Association (CSA) | http://www.csa.ca/ |
| Carbon Monoxide Headquarters | http://www.coheadquarters.com/CO1.htm |
| Centers for Disease Control and Prevention | http://www.cdc.gov/ |
| Environmental Health Watch | http://www.ehw.org/ |
| Gas Technology Institute | http://www.gri.org/ |
| Health Canada's Exposure Guidelines for Residential Indoor Air Quality | http://www.hc-sc.gc.ca/ewh-semt/pubs/air/exposure-exposition/index_e.html |
| HUD's Office of Healthy Homes and Lead Hazard Control | http://www.hud.gov/offices/lead/ |
| International Approval Services (IAS) | http://www.approvals.org/ |
| Iowa State University Extension | http://www.abe.iastate.edu/human_housing.asp |
| Home Safety Council | http://www.homesafetycouncil.org/ |
| NIOSH Criteria for a Recommended Standard: Occupational Exposure to Carbon Monoxide | http://www.cdc.gov/niosh/73-11000.html |
| National Fire Protection Association | http://www.nfpa.org/ |
| National Safety Council Indoor Air Program | http://www.nsc.org/ehc/indoor/iaq.htm |
| Occupational Safety and Health Administration | http://www.osha.gov/SLTC/healthguidelines/carbonmonoxide/index.html |
| Safer Child, Inc. – Indoor Pollution and Home Safety | http://www.saferchild.org/indoor.htm |
| Underwriters Laboratories (UL) | http://www.ul.com/ |
| U.S. Consumer Product Safety Commission | http://www.cpsc.gov/ |
| U.S. EPA Indoor Air Quality Home Page | http://www.epa.gov/iaq/ |
| U.S. EPA Air Quality Criteria for Carbon Monoxide | http://www.epa.gov/ncea/coabstract.htm |
| U.S. Fire Administration | http://www.usfa.fema.gov/ |