National Carbon Monoxide Awareness Association

AN URGENT CALL TO ACTION TO LOWER THE ALARM SET-POINT OF CARBON MONOXIDE ALARMS

Authors: Brianna Sleezer, Theresa Gamble, Charon McNabb, Dennis O'Meara, Nikki James Zellner

National Carbon Monoxide Awareness Association

AN URGENT CALL TO ACTION TO LOWER THE ALARM SET-POINT OF CARBON MONOXIDE ALARMS

Brianna Sleezer, Theresa Gamble, Charon McNabb, Dennis O'Meara, Nikki James Zellner

This white paper is an urgent call to action from the National Carbon Monoxide Awareness Association (NCOAA) to lower the alarm set-point of carbon monoxide (CO) alarms to minimize the risk of injury and death from chronic, low-level carbon monoxide exposure. Based on recommendations from the World Health Organization (WHO) and strong scientific evidence supporting those recommendations, we argue that reducing the alarm set-point of CO alarms is critical to protect individuals from undue harm resulting from chronic low-level CO exposure.

> "CO alarms are life safety devices, not injury prevention devices."
>
> ~ Consumer Product Safety Commission (CPSC)

DID YOU KNOW?

More than **95,000** CO poisonings occur annually in the US[1]

Approximately **95%** of all CO poisonings are non-fatal[1]

64% of non-fatal CO poisonings happen in the home[1]

Only **14%*** of homes in the US have properly functioning CO alarms[2]

*A more recent pilot study (conducted in 2020) in Washington, DC by Eurekafacts on behalf of the CPSC found that 67% of homes in Washington, DC had a properly functioning CO alarm.[3] However, given the study's small sample size and restricted geographic location, this statistic may not be representative of the entire US.

AUTHORS

Brianna J. Sleezer, PhD
Science and Medical Writer
Brianna holds her doctorate degree in Neuroscience from the University of Rochester School of Medicine and Dentistry. After completing her graduate work, she worked as a postdoctoral researcher at Cornell University where she studied the brain circuits regulating cognition and emotion. During her time as a neuroscientist, Brianna wrote and published numerous research papers and presented her findings at conferences across the country. Brianna now works as a science and medical writer specializing in biomedical communications within the pharmaceutical, healthcare, and biotechnology industries. Brianna provides NCOAA with a biomedical perspective and offers strategic direction for achieving the organization's goals for better screening and early detection.

Theresa Gamble, PhD
Science and Medical Writer
Theresa holds her doctorate degree in Biophysics from the University of California, San Francisco and a BS in Electrical Engineering from Massachusetts Institute of Technology. Theresa worked for seven years performing basic research in the area of structural biology, with particular emphasis on protein structure/function and HIV. She then transitioned into clinical research in the pharmaceutical industry gaining experience in project management, drug development and regulatory affairs. Currently, Theresa works for FHI 360 and holds the position of Scientist II and has responsibilities involving the development, implementation and results dissemination for international and domestic clinical trials for the HIV Prevention Trials Network (HPTN) and the HIV Vaccine Trials Network (HVTN). She is an experienced clinical researcher with extensive expertise in the field of HIV prevention, treatment, and vaccine development. Theresa is a contributing editor for NCOAA, providing expertise in the areas of medical research and technical writing.

Charon McNabb
President and Founder
Charon holds a bachelor's degree in Electrical Engineering from Lawrence Technological University. She spent 25 years in the automotive industry focused on new technology development with General Motors, Denso, and Magna International. She also worked with National Highway Traffic Safety Association (NHTSA) to drive new backup camera regulations beginning in 2018. Her engineering background and experience working with the NHTSA provide insight essential to achieving the goal of improved legislation and standards for carbon monoxide poisoning detection and prevention.

Dennis O'Meara, PMP
Director of Operations
Dennis holds a bachelor's degree in Business Administration from Concordia University. He has 40 years of experience in design, product engineering, and program management within the automotive market. Dennis has lived and worked abroad, supporting new European product launches. He also holds his PMP certification. His strong organization and communication skills have been a solid asset to NCOAA since joining the organization in 2019.

Nikki James Zellner
Director of Community Outreach
In February 2020, Nikki's life changed forever when her two young children suffered carbon monoxide poisoning at their Virginia Beach daycare. Since then, she has made headlines as an advocate and activist in the State of Virginia, in pursuit of legislation mandating carbon monoxide detectors in all educational facilities. Nikki focuses her efforts on making a significant impact in the communities she is a part of – including carbon monoxide poisoning prevention, military spouse entrepreneurship, finding success despite chronic illness, and breaking the cycle of poverty by sharing the power of entrepreneurship to underserved communities.

ACKNOWLEDGEMENTS

The authors and NCOAA would like to express their appreciation to the experts in the CO alarm industry and research community who contributed to this report, and particularly to the reviewers who made many thoughtful suggestions. We are indebted to them for their valuable contributions.

We would also like to give a special thank you to the LAUREN Project for their generous donation to help fund the development of this white paper and to the NCOAA volunteers who work tirelessly to improve CO safety.

The LAUREN PROJECT

The LAUREN (Leaders Abroad Urging Rights Equality and Non-violence) Project is a 501(c)3 organization founded to honor Lauren Moilien Johnson, who died from accidental carbon monoxide poisoning on January 5th, 2009, after a repairman put a damaged vent cap back on a roof vent, causing toxic carbon monoxide gas to back up into her apartment. The mission of the LAUREN Project is to make the world safer and more just by providing carbon monoxide detectors to those in need and by providing grants for international volunteer programs. Since 2009, the LAUREN Project has provided funding to over 140 grant recipients in more than 35 countries, totaling nearly $250,000. For more information on the LAUREN Project, please visit www.laurensproject.org.

TABLE OF CONTENTS

LIST OF TABLES

EXECUTIVE SUMMARY

Carbon monoxide (CO) is a colorless, odorless, and tasteless non-irritating gas that is imperceptible to human senses. CO poisoning is a leading cause of poisoning in the United States (US) and accounts for more than 20,000 ER visits annually.[4] The annual number of CO poisonings in the US is likely significantly higher than estimated, however, due to the imperceptible nature of CO, the wide array of CO poisoning symptoms, and a lack of robust diagnostic tools. Health effects of CO poisoning range from mild symptoms such as fatigue, dizziness, headache, confusion, and nausea to more severe symptoms such as disorientation, unconsciousness, long-term neurological disabilities, coma, cardiorespiratory failure, and death.[5] CO exposure is commonly underdiagnosed or misdiagnosed due to the nonspecific nature of the clinical effects.[6] Moreover, CO poisoning disproportionately affects marginalized communities, particularly those who are socially and economically disadvantaged.[7] As such, mitigating further CO poisonings is critical to create a healthier and more equitable nation.

In 2010, the World Health Organization (WHO) recommended a CO exposure limit of 6.11* parts per million (ppm) over the course of 24 hours.[8] The US Environmental Protection Agency (EPA) and the Agency for Toxic Substances and Disease Registry (ATSDR) published two subsequent reports further detailing the negative health effects of low-level CO exposure. [9,10] Their findings aligned with the WHO's recommendation and further stressed that the elderly and those with underlying heart disease are particularly susceptible to the adverse health effects of chronic low-level CO exposure. Despite these findings, however, many CO alarms in the US are prohibited from alarming at levels less than 70 ppm.[11] Thus, the alarm set-point for CO alarms in the US is inconsistent with the CO exposure limit supported by both national and international health organizations. According to the Consumer Product Safety Commission (CPSC), CO alarms are designed to be lifesaving devices, not long-term injury prevention devices.[12] As a result, millions of Americans are at risk for long-term debilitating diseases resulting from, or exacerbated by, chronic low-level CO poisoning.

NCOAA is initiating this urgent call to action to lower the alarm set-point of CO alarms in the US to protect the health and safety of individuals across the nation. This white paper presents the following information: current CO alarm standards and the historical events that led to the creation and adoption of these standards; the scientific data that informed the WHO's recommendation for CO exposure limits; and the need for urgent action to lower CO alarm levels.

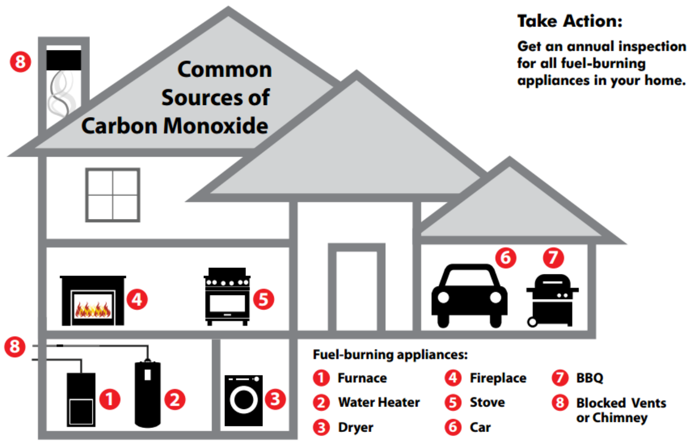
To address the challenges associated with lowering CO alarm levels, NCOAA is developing a multidisciplinary alliance of individuals and organizations that will work together to create and implement a strategic plan to improve CO safety. The purpose of this white paper is to provide background information on CO poisoning, alarms, and standards, and to serve as a springboard for further discussion and collaboration planning.

CO is a colorless, odorless, and tasteless non-irritating gas that is imperceptible to human senses.

*The WHO recommended a limit of 7 mg/m³. The value of 6.11 ppm is based on the conversion from mg/m³ to ppm, assuming a temperature of 25°C. Please also note that after the initial publication of this document, the WHO published updated air quality guidelines for multiple indoor and outdoor air pollutants, including CO. An addendum detailing the updated guidelines is provided on page 28 of this document.

CO SOURCES

Carbon Monoxide Poisoning Prevention



CO SOURCES INSIDE THE HOME

Carbon monoxide (CO) is a colorless, tasteless, and odorless gas produced by the burning of carbon-based fuels (wood, gas, petroleum, etc.) with the potential to cause significant human harm and death.[5] All homes and buildings with fuel-burning appliances and/or attached garages pose a risk for CO poisoning and proper precautions must be taken to prevent CO exposure. There are many different CO sources inside homes. These sources include:

Furnaces, Boilers, Water Heaters, and Space Heaters

Many furnaces, boilers, water heaters and space heaters are powered using combustible fuel. According to the US Energy Information Administration, over half of US households use appliances powered by combustible fuel.[13] These appliances emit CO. Since these appliances are frequently used in cold temperatures, unintentional CO poisoning is most common during colder winter months.

Portable Generators

Gas-powered portable generators can be particularly dangerous because they emit high levels of CO. A typical 5 kW portable generator releases an average of 1500 grams of CO in one hour.[14] CO poisoning due to gas-powered generators can be especially problematic during natural disasters which can prompt the use of gas-powered generators as a source of power. Research suggests that CO poisoning from portable generators can be more deadly than the natural disasters that cause people to use them. For example, among the fatalities related to Hurricane Irma in Florida, Georgia, and North Carolina in 2017, 16 were due to CO poisoning, while 11 were due to the storm itself.[15]

Did you know?

Some portable generators purchased after March 31, 2020 meet the voluntary manufacturer standards for CO and will shut off when specific CO concentrations are present near the generator. Charon McNabb, President and Founder of NCOAA, worked with the PGMA (Portable Generator Manufacturers Association) technical committee to implement new voluntary standards that better protect families and their pets from CO poisoning during emergencies.

Fireplaces

CO is produced when wood or natural gas is burned in fireplaces. Typically, if fireplaces are used and vented properly, the level of CO produced is not dangerous. However, if fumes from fireplaces are not properly exhausted, CO can rapidly accumulate inside houses and reach dangerous levels.

Gas Stoves and Ovens

As with all fuel burning appliances, gas stoves and ovens produce CO. To minimize CO production, kitchen appliances must be cleaned and maintained. Ducted range hoods should vent to the outside of the house through an exterior wall or the rooftop. Vents must be cleaned and maintained annually to ensure that CO escapes to the outside of the house.

Clothes Dryers

Gas dryers that are improperly installed or poorly vented can cause CO to accumulate inside houses. Moreover, even properly installed gas dryers can develop vent blockages over time (such as those caused by lint accumulation in the vent line or the presence of an animal nest at the vent exhaust).

CO SOURCES Continued

Vehicles in Attached Garages

Gas-powered vehicles produce large amounts of CO. For this reason, leaving vehicles idling in attached garages can be extremely dangerous and should never be done even if the garage door is open and even if vehicles are only idling in the garage for a short period of time.

WAYS TO REDUCE THE RISK OF CO POISONING INSIDE THE HOME

There are several ways to minimize the risk of CO poisoning inside the home. One way is to have all fuel-burning appliances checked and serviced annually by a qualified technician who consistently uses electronic CO detectors.

Chimneys and vent pipes should also be checked and/or cleaned every year to remove potential blockages. Gas generators should never be used inside houses, basements, or garages and should be placed at least 25 feet away from all indoor spaces. Gas ovens and stoves should not be used for indoor heating and exhaust hoods that are vented outside should always be turned on while using gas stove tops to prevent CO accumulation in kitchens. As mentioned above, it is also important to never leave a vehicle idling inside an attached garage even with the garage door open, and vehicle exhaust systems should be checked annually to prevent CO accumulation inside vehicles.

Another way to reduce the risk of home CO poisoning is to install a battery-operated or battery back-up CO alarm. Properly installed CO alarms should be located outside every sleeping area, on every level of the house, and more than 12 inches from an interior corner to allow for proper airflow.

CO alarms powered by 9-volt batteries should have the batteries replaced every 6 months.[16] For CO alarms powered by 10-year lithium batteries, the battery does not need to be replaced. All CO alarms need to be replaced after 5 to 7 years as the electrochemical sensors can wear out over time. It is also important to check the specific CO alarm to determine the exact lifetime of the product.[16]

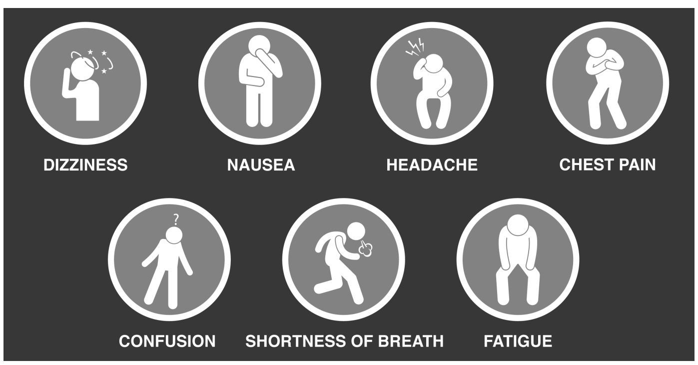
Many CO alarms sold in the US are not allowed to alarm at CO levels less than 70 ppm, yet the CO exposure limit recommended by the WHO is 6.11 ppm in 24 hours.[8,11] This recommendation is based on scientific evidence demonstrating that long-term, low-level CO exposure has a negative impact on human health. Thus, the alarm set-point for CO alarms in the US is inconsistent with CO exposure limits supported by both national and international health organizations.

According to the CPSC, CO alarms are designed to be lifesaving devices, not long-term injury prevention devices.[12] As such, millions of Americans could have long-term debilitating diseases as a result of, or exacerbated by, chronic low-level CO poisoning. According to the CDC, CO poisoning is responsible for over 20,000 emergency room visits, 4,000 hospitalizations, and 400 deaths each year in the US.[4] However, the annual number of CO poisonings in the US is likely significantly higher than estimated due to the imperceptible nature of CO, the wide array of CO poisoning symptoms, and a lack of robust diagnostic tools.[6] While everyone is susceptible to CO poisoning, some people are particularly sensitive to the effects of CO. Individuals that are most sensitive to CO include: infants, the elderly, and individuals with preexisting health conditions such as heart disease, anemia, and breathing problems.[4]

CO EXPOSURE AND HUMAN HEALTH

HEALTH EFFECTS OF ACUTE CO EXPOSURE

The exact symptoms of CO poisoning can vary widely and depend on the concentration and duration of CO exposure and the preexisting health status of each individual. Mild CO exposure typically causes dizziness, nausea, headache, chest pain, confusion, shortness of breath, and fatigue.[4,5] Due to the similarity between these symptoms and viral illnesses such as the seasonal flu, mild CO poisoning is often undetected and/or misdiagnosed.[6]

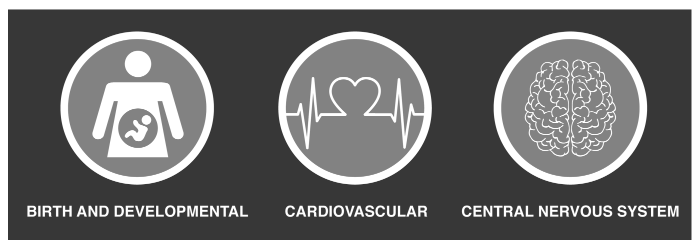


With increasing concentrations or duration of CO exposure, individuals may begin to experience shortness of breath, chest pain, syncope (fainting), tachypnea (rapid breathing), and tachycardia (rapid heart rate). Severe CO poisoning is life-threatening and can lead to reduced blood flow to the heart, irregular heartbeat, dangerously low blood pressure, heart attack, difficulty breathing or inability to breathe, seizures, unconsciousness, coma, and death.[4,5]

Another well-documented, yet poorly understood long-term health effect of CO poisoning is delayed encephalopathy (brain disease) or delayed neuropsychological sequelae (DNS). DNS is the sudden appearance of neuropsychological abnormalities after a period of recovery from initial CO symptoms. The typical features of DNS may include amnesia, fecal/urinary incontinence, gait and/or speech disturbances, anxiety, depression, and Parkinsonism.[17,18] The overall likelihood of developing DNS after CO poisoning could be as high as 40% and about 25% of all DNS cases are permanent.[19,20] Long-term health issues like DNS create significant socioeconomic consequences for those affected by CO poisoning, their families, and their communities.

HEALTH EFFECTS OF CHRONIC CO EXPOSURE

While the dangers of acute CO exposure have been known for centuries, it has more recently become apparent that chronic low-level CO exposure is also a major public health concern. Chronic low-level CO exposure has been linked to heart failure, stroke, cognitive and memory impairments, sensory-motor deficits, emotional changes, congenital defects, and low birth weight, among others (Appendices A-C).

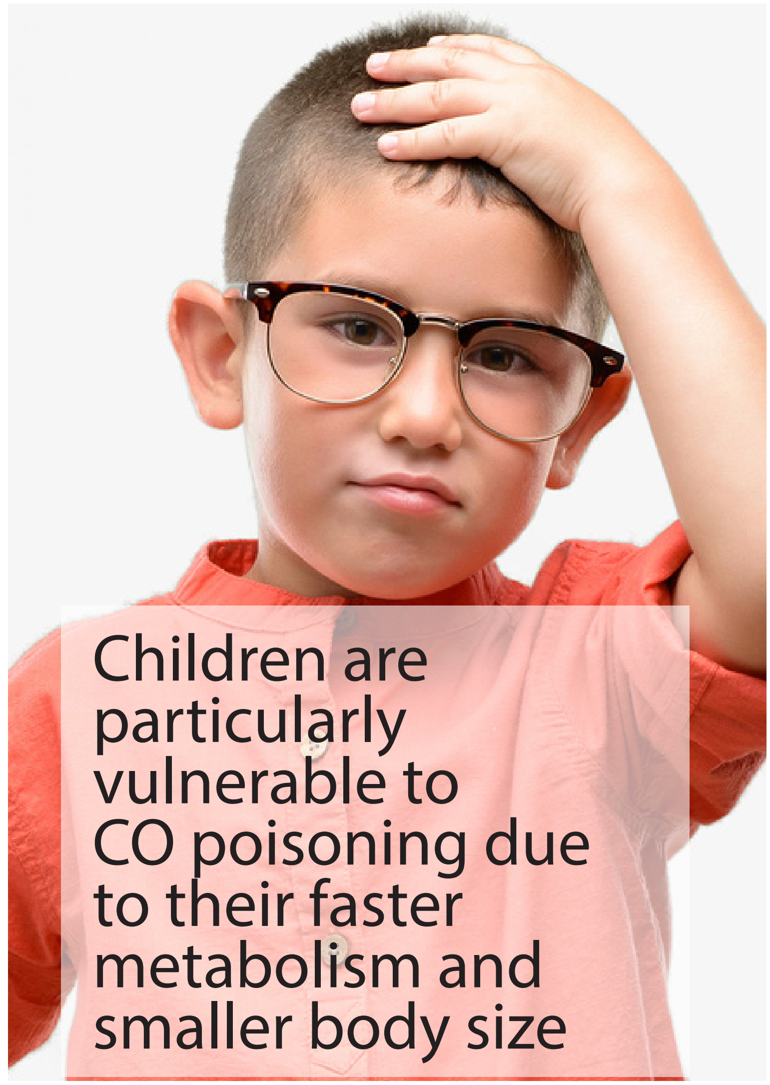


In 2010, the WHO recommended an exposure limit for chronic low-level CO of 6.11 ppm in 24 hours based on the following scientific evidence: epidemiological studies (Appendix A) which demonstrate an association between low-level chronic CO exposure and cardiovascular morbidity (i.e., heart attack, congestive heart failure, and ischemic heart disease); laboratory dose-response studies (Appendix B) which demonstrate an acute exposure-related reduction in exercise tolerance and increased symptoms related to heart disease; and retrospective and case studies (Appendix C) describing instances of long-term CO poisoning resulting in a multitude of negative physical, emotional, and cognitive health effects.[8] Taken together, these studies provide sufficient evidence to support a relationship between chronic low-level CO exposure and serious negative health outcomes, thus providing a strong basis for the WHO's recommended exposure limit of 6.11 ppm in 24 hours.

CO EXPOSURE AND HUMAN HEALTH Continued

In 2010, the EPA published a report titled Integrated Science Assessment for Carbon Monoxide.[9] This report agreed with the WHO's main conclusion that chronic low-level CO is associated with adverse health effects. Specifically, the EPA concluded that the available epidemiological data suggests a causal link between chronic low-level CO exposure and birth outcomes, developmental effects, and central nervous system (i.e., brain and spinal cord) effects.

In 2012, the ATSDR published a report titled Toxicological Profile for Carbon Monoxide.[10] This report also agreed with the WHO's main conclusion that chronic low-level CO is associated with adverse health effects and further corroborated the conclusions made by the WHO and EPA regarding the fact that chronic low-level CO seems to have particularly negative effects on the cardiovascular system, the central nervous system, and the development of fetuses and babies. Both the EPA and ATSDR further stress that elderly individuals and those with underlying cardiac disease are particularly susceptible to the adverse health effects of chronic low-level CO exposure.



CO POISONING DIAGNOSIS (AND MISDIAGNOSIS)

CO poisoning diagnosis is typically based on clinical symptoms and suspected or confirmed CO exposure. However, CO poisoning is often missed by doctors for the following reasons:

- Many of the symptoms of chronic low-level CO exposure (fatigue, headaches, dizziness, and nausea) are similar to those of other illnesses (e.g., the seasonal flu) leading to frequent misdiagnoses.
- CO poisoning symptoms are not always well correlated with CO exposure levels.
- A blood test to measure carboxyhemoglobin (COHb) levels is often used to confirm CO poisoning, but there are several problems with this medical diagnostic:
  - Humans all produce some amount of endogenous (natural) CO as a byproduct of heme catabolism. Therefore, we all have some amount of COHb in our blood naturally.[54]
  - Cigarette smoking can significantly increase COHb levels.[55,56]
  - Air pollution can increase COHb levels.[57,58]
  - CO poisoning symptoms and COHb levels are poorly correlated, especially in cases of low-level CO exposure and in smokers.[59]
  - By the time an individual sees a doctor (either through emergency room admission or through a scheduled visit with a primary care physician), they have often been removed from the source of CO for long enough that their COHb levels have dropped significantly. Thus, COHb levels measured by doctors are often an inaccurate indicator of CO exposure. New technologies such as CO breath analyzers, however, may help to diagnose CO poisoning by offering Emergency Services and First Responders a tool for earlier assessment of CO exposure.

Given the difficulties associated with CO poisoning diagnosis, the detection of low-level CO in homes is essential to prevent the adverse health effects of low-level CO exposure. Changing CO detector alarm level standards is a critical initial step in this endeavor.

US REGULATIONS AND STANDARDS FOR CO EXPOSURE AND ALARMS

CURRENT CO EXPOSURE REGULATIONS

In the US, the Occupational Safety and Health Administration (OSHA) limits CO exposure to an average of 50 ppm over an 8-hour period within workplace settings[60], while the EPA recommends an average limit of 9 ppm over an 8-hour period for ambient air.[61] The WHO has also put forth guidelines for CO exposure (Table 1), including a recommended average limit for low-level chronic CO exposure of 6.11 ppm over a 24-hour period.[8]

Table 1
WHO Guidelines for Average CO Exposure

CO (ppm)*	Time
87.29	15 minutes
30.55	1 hour
8.73	8 hours
6.11	24 hours

CURRENT CO ALARM REGULATIONS

CO alarm requirements in buildings are not regulated at the federal level. As such, these regulations vary from state to state. These regulations also differ depending on whether a building is new or existing.

Homes: 46 states and Washington, DC have enacted some level of statewide CO alarm legislation requiring CO alarms in new and/or existing residential homes. Hawaii, Kansas, Missouri, and Texas have not.[62]

Apartments: 46 states and Washington, DC have enacted some level of statewide CO alarm legislation requiring CO alarms in new and/or existing apartments. Hawaii, Kansas, Missouri, and Texas have not.[62]

Hotels/dormitories: 45 states and Washington, DC have enacted some level of statewide CO alarm legislation requiring CO alarms in new and/or existing hotels and dormitories. Hawaii, Kansas, Missouri, Nebraska, and Texas have not.[62]

Nursing homes: 37 states and Washington, DC require CO alarms in new and/or existing residential board and care facilities. Elderly individuals are especially vulnerable to chronic conditions exacerbated by CO. Exposure to CO is associated with an increased risk of hospitalization for elderly individuals with heart problems.[22,62]

Schools: 36 states have enacted some level of statewide CO alarm legislation requiring CO alarms in new and/or existing schools.[62]

Daycares: 45 states and Washington, DC require CO alarms in new or existing daycares. Young children are especially vulnerable to the effects of CO because of their smaller bodies. Children process CO differently than adults, may be more severely affected by it, and may show signs of poisoning sooner.[62,63]

Assembly occupancies: Assembly occupancies refer to places where 50 or more people gather for deliberation, worship, entertainment, eating, drinking, amusement, or awaiting transportation. Examples include: assembly halls, bus depots, churches, and restaurants. Only 4 states (Maine, Maryland, New Jersey, and New York) require CO alarms in assembly occupancies.[62]

CURRENT CO ALARM STANDARDS

Standards for home CO alarms sold in the US are voluntary, meaning that manufacturers do not have to comply with them. The Underwriters Laboratories (UL) standards for CO alarms are described in UL 2034.[11] In accordance with this document, CO alarms should alarm according to the specifications in Table 2.

Table 2
UL 2034 CO Alarm Standards

CO (ppm)	Alarm
< 70	Alarm prohibitted unless CO is continuously 30 to 69 ppm for over 30 days
70 - 149	Alarm within 60 - 240 minutes of exposure
150 - 399	Alarm within 10 - 50 minutes of exposure
> 400	Alarm within 4 - 15 minutes of exposure

*Values were converted from mg/m^3 to ppm, assuming a temperature of 25°C.

US REGULATIONS AND STANDARDS FOR CO EXPOSURE AND ALARMS Continued

HISTORICAL RATIONALE FOR CURRENT CO ALARM STANDARDS

Why are CO alarm standards so incongruous with the recommendations of national and international health organizations? To better understand this, it is important to consider key historical events.

CO detection technology began in the late 19th century when a Scottish scientist, John Scott Haldane, developed the first colorimetric analog CO gas tube analyzers. Over two decades later, in 1927, the first patent for a CO detector was awarded to Chester Gordon and James Lowe. These detectors worked by crushing a glass vial to observe a chemical reaction in the presence of CO and weren't particularly practical. It wasn't until much later, in the 1990's that CO alarms became mainstream, with the development of affordable metal oxide models.

CO poisoning incidents also began to receive heightened media attention in the 1990's. A popular television show, Rescue 911, for example, aired an episode on December 15, 1992 featuring a case of CO poisoning among a Chicago family in November 1991. All 10 members of the family died, including a mother and father and their 8 children. Another case that received a great deal of publicity was the 1994 death of tennis star Vitas Gerulaitis, who died of CO poisoning while visiting a friend in Southampton, New York.

As a result of the heightened media attention on CO poisonings, legislators and other stakeholders began to make CO safety a priority. Initial work on UL voluntary standard 2034 began in 1989, and was formally released in 1992. Shortly thereafter, in 1993, manufacturers of CO alarms introduced battery-powered alarms making them an affordable option for use in homes and small businesses.

In 1994, Chicago became one of the first cities in the US to adopt an ordinance requiring the installation of CO alarms in new and existing single-family homes that had oil or gas furnaces. This ordinance was passed on October 1, 1994. In the months that followed the installation of CO alarms across Chicago, Chicago fire departments experienced a large volume of calls related to CO alarms. Specifically, between October 1 and December 31, 1994, Chicago fire departments reported that they responded to approximately 8,600 CO alarm calls. As described further below, a number of different factors likely contributed to elevated CO levels in Chicago homes and the subsequent CO alarm activations during the winter of 1994. However, in the subsequent news coverage of this event (dubbed the "Night of Sirens"), many headlines referred to the thousands of alarms as "nuisance alarms" or "false alarms", and, as a result, many citizens and first responders began to ignore CO alarm activations altogether.

But, were these alarm activations truly "false alarms"? Were Chicago first responders and HVAC professionals adequately equipped with the necessary CO detection equipment and training to accurately detect and measure CO when responding to a CO alarm? Did the rapid evolution of CO detection technology negatively impact the reliability of CO alarms? Were thousands of Chicago residents actually exposed to dangerous levels of CO in their homes?

To collect data relevant to these and other questions, the CPSC organized a public hearing on February 21, 1996.[64] As part of this hearing, the CPSC solicited scientific, medical, and technical information from all interested parties related to the health effects of CO, the 1994 Chicago CO alarm activations, indoor and outdoor CO levels, CO alarm standards, consumers' ability to differentiate between warning signals and urgent alarms, and the needs of individuals responding to CO alarm activations. Speakers at the public hearing included nonprofit organizations, science and medical

US REGULATIONS AND STANDARDS FOR CO EXPOSURE AND ALARMS Continued

professionals, firefighters, government officials and representatives from the gas utility industry. The majority of speakers at the public hearing agreed that low-level CO exposure is a concern, particularly among susceptible populations (including pregnant women, children, individuals with preexisting health conditions, and the elderly).

Speakers at the public hearing also raised concerns related to CO alarm accuracy, repeatability, life-expectancy, humidity interference, and multiple gas interference. Several speakers believed changes made to the voluntary standards in 1995 would address these concerns.

Expert testimony further argued that the Chicago 1994 alarm activations were not actually "false alarms", but instead reflected accurate CO detection, and were likely incorrectly labeled as "false alarms" due to inadequate CO safety equipment and training for first responders to appropriately investigate CO leaks. Other factors contributing to the large number of CO activations were also discussed at the hearing, including incorrect placement of CO alarms, lack of public education, and a rare temperature inversion, which trapped pollutants (including CO) close to the ground. Moreover, despite news outlets claiming that first responders were overwhelmed by the thousands of CO alarm calls in 1994, this claim was refuted at the public hearing, with speakers indicating that the Chicago fire department had adequate resources to respond to the CO alarm activations.

Despite widespread support of low-level alarms at the public hearing and testimony from medical experts stressing the dangers of low-level CO, the CO alarm limit was increased in 1998 to a 100 ppm ceiling and then later lowered to 70 ppm. At 70 ppm, the CPSC considers CO alarms life-safety, not injury-prevention devices.[12]

Concerningly, the majority of homeowners are not aware that most home CO alarms do not protect them from the dangers of low-level CO. According to a pilot study conducted in Washington, DC by EurekaFacts on behalf of the CPSC, the majority of households (85%) believe that their CO alarm(s) will alert them if CO is present.[3]

Consequently, 25 years after the events in Chicago and the CPSC public hearing, CO alarm standards in the US leave our most vulnerable citizens unaware of the dangers of low-level CO in their homes and at risk for low-level CO poisoning; first responders and HVAC professionals still do not have adequate CO detection equipment and training to accurately and consistently detect CO in homes, and home CO alarm technology is outdated and confusing to both homeowners and first responders alike.

MOVING FORWARD

We believe that an appropriate solution to address low-level CO will require a collaborative and carefully considered approach.

Over the last few decades, a great deal of progress has been made in the areas of CO awareness, legislation, and technology. However, current CO alarm standards in the US are not sufficient to protect people from the dangers of CO in their homes. Therefore, we believe it is necessary to revisit the issue of CO alarm levels and develop a solution that protects everyone - including susceptible populations - from CO poisoning.

We also believe that an appropriate solution will require a collaborative and carefully considered approach. Thus, NCOAA is organizing a multidisciplinary working group of individuals and organizations focused on improving CO Safety. This group will work toward developing an action plan to improve CO safety, including lowering the alarm set-point of CO alarms.

To request more information about how you can help mitigate further CO poisonings and create a healthier and more equitable nation, please contact us at info@ncoaa.us.

WAYS TO SUPPORT CO AWARENESS

awareness and safety. Our mission is to initiate a global conversation on the diagnoses, treatment, and prevention of chronic and acute carbon monoxide poisoning by organizing available CO poisoning information and driving change to improve diagnostics, detection, treatment, legislation, and standards throughout the globe. You can help prevent senseless CO injuries and deaths by making a gift to NCOAA.

Supporting NCOAA is easy!

1. Visit our website at www.ncoaa.us/donations or scan the QR code below to make a secure online donation:

2. Mail your contribution to NCOAA at:
   6855 Oakhills Drive
   Bloomfield Hills, MI 48301

To make a donation by phone or to arrange an in-kind donation in support of our mission, contact us at info@ncoaa.us.

Connect with us on social media and join the conversation:

REFERENCES

1. Hampson, N. B. (2016). Cost of accidental carbon monoxide poisoning: A preventable expense. Preventive medicine reports, 3, 21-24. https://doi.org/10.1016/j.pmedr.2015.11.010
2. Ryan, T. J., & Arnold, K. J. (2011). Residential carbon monoxide detector failure rates in the United States. American journal of public health, 101(10), e15-e17. https://doi.org/10.2105/AJPH.2011.300274

    Note: This study reports that approximately 38 million homes in the US have CO detectors installed; and, among the 30 home CO detectors tested, 57% failed to alarm as intended. To calculate the percent of US homes with functioning CO detectors, we divided the number of home CO detectors installed in the US in 2011 (38 million) by the US census statistic for the number of homes in the US in 2011 (118.68 million) and multiplied this by the proportion of homes with functioning CO detectors according to Ryan & Arnold, 2011 (43%). This calculation reveals that 14% of homes in the US have functioning CO detectors (as of 2011).
3. United States Consumer Product Safety Commission (2020). Consumer Product Safety Commission (CPSC) Staff's Statement on EurekaFacts Report on "SCOA Survey Findings from the Washington DC Metro Area Door-to-Door Pilot, Final Submission September 15, 2020". https://www.cpsc.gov/s3fs-public/CPSC-Survey-Revised-DiagnosticReport_11_18_20206b6.pdf
4. United States Centers for Disease Control and Prevention. (2018). Carbon Monoxide Poisoning—Frequently Asked Questions. https://www.cdc.gov/co/faqs.htm
5. United States Environmental Protection Agency, O. (2019). What is carbon monoxide? [Overviews and Factsheets]. US EPA. https://www.epa.gov/indoor-air-quality-iaq/what-carbon-monoxide-0
6. Harper, A., & Croft-Baker, J. (2004). Carbon monoxide poisoning: Undetected by both patients and their doctors. Age and Ageing, 33(2). https://doi.org/10.1093/ageing/afh038
7. National Energy Action. (2017). NEA: Understanding Carbon Monoxide Risk in Households Vulnerable to Fuel Poverty. https://www.nea.org.uk/publications/understanding-carbon-monoxide-rise-in-households-vulnerable-to-fuel-poverty/
8. World Health Organization. (2010). WHO guidelines for indoor air quality: Selected pollutants. World Health Organization. Regional Office for Europe. https://apps.who.int/iris/handle/10665/260127
9. United States Environmental Protection Agency. (2010). Integrated Science Assessment For Carbon Monoxide. https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=218686
10. Wilbur, S., Williams, M., Williams, R., Scinicariello, F., Klotzbach, J. M., Diamond, G. L., & Citra, M. (2012). Toxicological Profile for Carbon Monoxide. Agency for Toxic Substances and Disease Registry. http://www.ncbi.nlm.nih.gov/books/NBK153693/
11. Underwriters Laboratories. (2018). Standard for Single and Multiple Station Carbon Monoxide Alarms (UL - 2034). https://standardscatalog.ul.com/standards/en/standard_2034
12. United States Consumer Product Safety Commission. (2016). Carbon Monoxide Alarms. CPSC.Gov. https://www.cpsc.gov/Regulations-Laws--Standards/Voluntary-Standards/Carbon-Monoxide-Alarms
13. Energy Information Administration. (2018). Residential Energy Consumption Survey (RECS): Table HC1.1 Fuels used and end uses in U.S. homes by housing unit type, 2015. https://www.eia.gov/consumption/residential/data/2015/hc/php/hc1.6.php
14. Buyer, J. (2016, November 16). Portable Generators and Carbon Monoxide Poisoning. SAE Small Engine Technology Conference, Charleston, SC. https://www.cpsc.gov/s3fs-public/PresentationSAE_SETC.pdf
15. Issa, A., Ramadugu, K., Mulay, P., Hamilton, J., Siegel, V., Harrison, C., Campbell, C. M., Blackmore, C., Bayleyegn, T., & Boehmer, T. (2018). Deaths Related to Hurricane Irma—Florida, Georgia, and North Carolina, September 4–October 10, 2017. Morbidity and Mortality Weekly Report, 67(30), 829–832. https://doi.org/10.15585/mmwr.mm6730a5
16. First Alert. (n.d.). 6 Things to Know About Carbon Monoxide Alarms and Detectors. Retrieved February 8, 2021, from https://www.firstalert.com/on/demandware.store/Sites-firstalert-Site/default/Content-Show?cid=6-things-to-know-about-carbon-monoxide-alarms

REFERENCES Continued

17. Oh, S., & Choi, S.-C. (2015). Acute carbon monoxide poisoning and delayed neurological sequelae: A potential neuroprotection bundle therapy. Neural Regeneration Research, 10(1), 36–38. https://doi.org/10.4103/1673-5374.150644
18. Sönmez, B. M., İşcanlı, M. D., Parlak, S., Doğan, Y., Ulubay, H. G., & Temel, E. (2018). Delayed neurologic sequelae of carbon monoxide intoxication. Turkish Journal of Emergency Medicine, 18(4), 167–169. https://doi.org/10.1016/j.tjem.2018.04.002
19. Pepe, G., Castelli, M., Nazerian, P., Vanni, S., Del Panta, M., Gambassi, F., Botti, P., Missanelli, A., & Grifoni, S. (2011). Delayed neuropsychological sequelae after carbon monoxide poisoning: Predictive risk factors in the Emergency Department. A retrospective study. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine, 19(1), 16. https://doi.org/10.1186/1757-7241-19-16
20. Kim, H., Choi, S., Park, E., Yoon, E., Min, Y., & Lampotang, S. (2018). Serum markers and development of delayed neuropsychological sequelae after acute carbon monoxide poisoning: Anion gap, lactate, osmolarity, S100B protein, and interleukin-6. Clinical and Experimental Emergency Medicine, 5(3), 185–191. https://doi.org/10.15441/ceem.17.217
21. Barnett, A. G., Williams, G. M., Schwartz, J., Best, T. L., Neller, A. H., Petroeschevsky, A. L., & Simpson, R. W. (2006). The effects of air pollution on hospitalizations for cardiovascular disease in elderly people in Australian and New Zealand cities. Environmental Health Perspectives, 114(7), 1018–1023. https://doi.org/10.1289/ehp.8674
22. Bell, M. L., Peng, R. D., Dominici, F., & Samet, J. M. (2009). Emergency hospital admissions for cardiovascular diseases and ambient levels of carbon monoxide: Results for 126 United States urban counties, 1999-2005. Circulation, 120(11), 949–955. https://doi.org/10.1161/CIRCULATIONAHA.109.851113
23. Burnett, R. T., Dales, R. E., Brook, J. R., Raizenne, M. E., & Krewski, D. (1997). Association between ambient carbon monoxide levels and hospitalizations for congestive heart failure in the elderly in 10 Canadian cities. Epidemiology, 8(2), 162–167. https://doi.org/10.1097/00001648-199703000-00011
24. Chen, L., Jennison, B. L., Yang, W., & Omaye, S. T. (2000). Elementary school absenteeism and air pollution. Inhalation Toxicology, 12(11), 997–1016. https://doi.org/10.1080/08958370050164626
25. Dales, R. E., Cakmak, S., & Vidal, C. B. (2009). Air Pollution and Hospitalization for Headache in Chile. American Journal of Epidemiology, 170(8), 1057–1066. https://doi.org/10.1093/aje/kwp217
26. Hong, Y.-C., Lee, J.-T., Kim, H., Ha, E.-H., Schwartz, J., & Christiani, D. C. (2002). Effects of air pollutants on acute stroke mortality. Environmental Health Perspectives, 110(2), 187–191. https://doi.org/10.1289/ehp.02110187
27. Maisonet, M., Bush, T. J., Correa, A., & Jaakkola, J. J. (2001). Relation between ambient air pollution and low birth weight in the Northeastern United States. Environmental Health Perspectives, 109(Suppl 3), 351–356. https://doi.org/10.1289/ehp.01109s3351
28. Morris, R. D., Naumova, E. N., & Munasinghe, R. L. (1995). Ambient air pollution and hospitalization for congestive heart failure among elderly people in seven large US cities. American Journal of Public Health, 85(10), 1361–1365. https://doi.org/10.2105/ajph.85.10.1361
29. Ritz, B., & Yu, F. (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(1), 17–25. https://doi.org/10.1289/ehp.9910717
30. Stieb, D. M., Szyszkowicz, M., Rowe, B. H., & Leech, J. A. (2009). Air pollution and emergency department visits for cardiac and respiratory conditions: a multi-city time-series analysis. Environmental Health, 8(1), 1-13. https://doi.org/10.1186/1476-069X-8-25
31. Yang, C.-Y., Chen, Y.-S., Yang, C.-H., & Ho, S.-C. (2004). Relationship Between Ambient Air Pollution and Hospital Admissions for Cardiovascular Diseases in Kaohsiung, Taiwan. Journal of Toxicology and Environmental Health, Part A, 67(6), 483–493. https://doi.org/10.1080/15287390490276502
32. Yang, C.-Y. (2008). Air Pollution and Hospital Admissions for Congestive Heart Failure in a Subtropical City: Taipei, Taiwan. Journal of Toxicology and Environmental Health, 71(16), 1085–1090. https://doi.org/10.1080/15287390802114428
33. Adams, K. F., Koch, G., Chatterjee, B., Goldstein, G. M., O'Neil, J. J., Bromberg, P. A., & Sheps, D. S. (1988). Acute elevation of blood carboxyhemoglobin to 6% impairs exercise performance and aggravates symptoms in patients with ischemic heart disease. Journal of the American College of Cardiology, 12(4), 900–909. https://doi.org/10.1016/0735-1097(88)90452-4

REFERENCES Continued

34. Allred, E. N., Bleecker, E. R., Chaitman, B. R., Dahms, T. E., Gottlieb, S. O., Hackney, J. D., Pagano, M., Selvester, R. H., Walden, S. M., & Warren, J. (1989). Short-Term Effects of Carbon Monoxide Exposure on the Exercise Performance of Subjects with Coronary Artery Disease. New England Journal of Medicine, 321(21), 1426–1432. https://doi.org/10.1056/NEJM198911233212102

35. Allred, E. N., Bleecker, E. R., Chaitman, B. R., Dahms, T. E., Gottlieb, S. O., Hackney, J. D., ... & Warren, J. (1991). Effects of carbon monoxide on myocardial ischemia. Environmental Health Perspectives, 91, 89-132. https://doi.org/10.1289/ehp.919189

36. Anderson, E. W., Andelman, R. J., Strauch, J. M., Fortuin, N. J., & Knelson, J. H. (1973). Effect of Low-Level Carbon Monoxide Exposure on Onset and Duration of Angina Pectoris. Annals of Internal Medicine, 79(1), 46–50. https://doi.org/10.7326/0003-4819-79-1-46

37. Ekblom, B., & Huot, R. (1972). Response to Submaximal and Maximal Exercise at Different Levels of Carboxyhemoglobin. Acta Physiologica Scandinavica, 86(4), 474–482. https://doi.org/10.1111/j.1748-1716.1972.tb05350.x

38. Horvath, S. M., Raven, P. B., Dahms, T. E., & Gray, D. J. (1975). Maximal aerobic capacity at different levels of carboxyhemoglobin. Journal of Applied Physiology, 38(2), 300–303. https://doi.org/10.1152/jappl.1975.38.2.300

39. Kleinman, M. T., Davidson, D. M., Vandagriff, R. B., Caiozzo, V. J., & Whittenberger, J. L. (1989). Effects of short-term exposure to carbon monoxide in subjects with coronary artery disease. Archives of Environmental Health: An International Journal, 44(6), 361–369. https://doi.org/10.1080/00039896.1989.9935908

40. Kleinman, M. T., Leaf, D. A., Kelly, E., Caiozzo, V., Osann, K., & O'niell, T. (1998). Urban Angina in the Mountains: Effects of Carbon Monoxide and Mild Hypoxemia on Subjects with Chronic Stable Angina. Archives of Environmental Health: An International Journal, 53(6), 388–397. https://doi.org/10.1080/00039899809605726

41. Pirnay, F., Dujardin, J., Deroanne, R., & Petit, J. M. (1971). Muscular exercise during intoxication by carbon monoxide. Journal of Applied Physiology, 31(4), 573–575. https://doi.org/10.1152/jappl.1971.31.4.573

42. Vogel, J. A., & Gleser, M. A. (1972). Effect of carbon monoxide on oxygen transport during exercise. Journal of Applied Physiology, 32(2), 234–239. https://doi.org/10.1152/jappl.1972.32.2.234

43. Devine, S. A., Kirkley, S. M., Palumbo, C. L., & White, R. F. (2002). MRI and neuropsychological correlates of carbon monoxide exposure: A case report. Environmental Health Perspectives, 110(10), 1051–1055. https://doi.org/10.1289/ehp.021101051

44. Ely, E. W., Moorehead, B., & Haponik, E. F. (1995). Warehouse workers' headache: Emergency evaluation and management of 30 patients with carbon monoxide poisoning. The American Journal of Medicine, 98(2), 145–155. https://doi.org/10.1016/S0002-9343(99)80398-2

45. Foster, M., Goodwin, S. R., Williams, C., & Loeffler, J. (1999). Recurrent acute life-threatening events and lactic acidosis caused by chronic carbon monoxide poisoning in an infant. Pediatrics, 104(3), e34–e34. https://doi.org/10.1542/peds.104.3.e34

46. Keles, A., Demircan, A., & Kurtoglu, G. (2008). Carbon monoxide poisoning: How many patients do we miss? European Journal of Emergency Medicine, 15(3), 154–157. https://doi.org/10.1097/MEJ.0b013e3282efd519

47. Khan, K., & Sharief, N. (1995). Chronic carbon monoxide poisoning in children. Acta Paediatrica, 84(7), 742-742. https://doi.org/10.1111/j.1651-2227.1995.tb13747.x

48. Myers, R. A., DeFazio, A., & Kelly, M. P. (1998). Chronic carbon monoxide exposure: A clinical syndrome detected by neuropsychological tests. Journal of Clinical Psychology, 54(5), 555–567. https://doi.org/10.1002/(sici)1097-4679(199808)54:5<555::aid-jclp1>3.0.co;2-k

49. Pinkston, J. B., Wu, J. C., Gouvier, W. D., & Varney, N. R. (2000). Quantitative PET Scan Findings in Carbon Monoxide Poisoning: Deficits Seen in a Matched Pair. Archives of Clinical Neuropsychology, 15(6), 545–553. https://doi.org/10.1016/S0887-6177(99)00051-7

50. Prockop, L. D. (2005). Carbon monoxide brain toxicity: clinical, magnetic resonance imaging, magnetic resonance spectroscopy, and neuropsychological effects in 9 people. Journal of Neuroimaging, 15(2), 144-149. https://doi.org/10.1177/1051228404273819

51. Ryan, C. M. (1990). Memory disturbances following chronic, low-level carbon monoxide exposure. Archives of clinical

REFERENCES Continued

neuropsychology, 5(1), 59-67. https://doi.org/10.1016/0887-6177(90)90007-C

52. Thom, S. R., Bhopale, V. M., Fisher, D., Zhang, J., & Gimotty, P. (2004). Delayed neuropathology after carbon monoxide poisoning is immune-mediated. Proceedings of the National Academy of Sciences, 101(37), 13660–13665. https://doi.org/10.1073/pnas.0405642101
53. Thyagarajan, M. S., Gunawardena, W. J., & Coutinho, C. M. A. (2003). Seizures and Unilateral Cystic Lesion of the Basal Ganglia: An Unusual Clinical and Radiological Manifestation of Chronic Non-fatal Carbon Monoxide (CO) Poisoning. Clinical Radiology Extra, 58(5), 38–41. https://doi.org/10.1016/S1477-6804(03)00003-7
54. Coburn, R. F. (1970). Endogenous carbon monoxide production. New England Journal of Medicine, 282(4), 207–209. https://doi.org/10.1056/NEJM197001222820407
55. Seppänen, A., & Uusitalo, A. J. (1977). Carboxyhaemoglobin saturation in relation to smoking and various occupational conditions. Annals of Clinical Research, 9(5), 261–268.
56. Russell, M. A. H. (1973). Blood carboxyhaemoglobin changes during tobacco smoking. Postgraduate Medical Journal, 49(576), 684–687. https://doi.org/10.1136/pgmj.49.576.684
57. Bono, R., Piccioni, P., Traversi, D., Degan, R., Grosa, M., Bosello, G., Gilli, G., Arossa, W., & Bugiani, M. (2007). Urban air quality and carboxyhemoglobin levels in a group of traffic policemen. Science of the Total Environment, 376(1–3), 109–115. https://doi.org/10.1016/j.scitotenv.2007.01.086
58. Goldsmith, J. R., & Cohen, S. I. (1969). Epidemiological Bases for Possible Air Quality Criteria for Carbon Monoxide. Journal of the Air Pollution Control Association, 19(9), 704–713. https://doi.org/10.1080/00022470.1969.10466551
59. Stewart, R. D., Baretta, E. D., Platte, L. R., Stewart, E. B., Kalbfleisch, J. H., Van Yserloo, B., & Rimm, A. A. (1974). Carboxyhemoglobin levels in American blood donors. JAMA, 229(9), 1187–1195. https://doi.org/10.1001/jama.1974.03230470029019
60. Occupational Health and Safety Administration. (2019). TABLE Z-1 Limits for Air Contaminants. https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.1000TABLEZ1
61. Environmental Protection Agency. (2020). National primary ambient air quality standards for carbon monoxide. https://www.ecfr.gov/cgi-bin/text-idx?SID=b0660aaaf5a672927f005b9eb10ad28f&mc=true&node=se40.2.50_18&rgn=div8
62. Johansen, N. (2021). Carbon Monoxide Detection and Alarm Requirements: Literature Review. Fire Protection Research Foundation. https://www.nfpa.org/News-and-Research/Data-research-and-tools/Detection-and-Signaling/Carbon-Monoxide-Detection-and-Alarm-Requirements-Literature-Review
63. National Center on Early Childhood Quality Assurance. (2016). Handling, Storing, and Disposing of Hazardous Materials and Biological Contaminants. https://www.researchconnections.org/childcare/resources/32401
64. United States Consumer Product Safety Commission (1996, February 21). Carbon Monoxide Detector Hearing. https://static1.squarespace.com/static/56e787dfb09f95498f2e6b60/t/604811dab081e82e2b153ce5/1615335899614/FOIA+CO+-Detector+Transcripts.pdf
65. World Health Organization. (2021). WHO global air quality guidelines: particulate matter (PM2.5 and PM10), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. World Health Organization. https://apps.who.int/iris/handle/10665/345329

APPENDIX A

EVIDENCE SUPPORTING THE WHO'S RECOMMENDATION: EPIDEMIOLOGICAL STUDIES

Study	Details
Barnett et al,[21] 2006	This study found a significant relationship between the number of hospital admissions for cardiovascular disease among elderly people and CO concentration in outdoor air pollution in Australia and New Zealand between 1998 and 2001. For each 0.9 ppm increase in the daily maximum 8-hour CO level, there was a 2.2% increase in hospital admissions for cardiovascular disease.
Bell et al,[22] 2009	This study found a significant relationship between the number of hospital admissions for cardiovascular disease among people aged 65 and older and CO concentration in outdoor air pollution in 126 US urban cities between 1999 and 2005. For each 1 ppm increase in the daily maximum 1-hour CO level, there was a 0.96% increase in hospital admissions for cardiovascular disease.
Burnett et al,[23] 1997	This study found a significant relationship between the number of hospital admissions for congestive heart failure among elderly individuals and CO concentration in outdoor air pollution in 10 Canadian cities between 1981 and 1991. The relative risk of hospital admission for congestive heart failure associated with an increase of 1 to 3 ppm in the daily maximum 8-hour CO level was 1.065.
Chen et al,[24] 2000	This study found a significant relationship between elementary school absenteeism and CO concentration in outdoor air pollution in Washoe County, Nevada from 1996-1998. For each 1 ppm increase in the highest daily 1-hour maximum CO level, there was a 3.8% increase in elementary school absence rate.
Dales et al,[25] 2009	This study found a significant relationship between the number of hospital admissions for headache and CO concentration in outdoor air pollution in 7 Chilean urban cities between 2001 and 2005. The relative risk of hospital admission for headache associated with an increase of 1.15 ppm in CO was 1.1.
Hong et al,[26] 2002	This study found a significant relationship between stroke mortality (i.e., number of deaths per day) and CO concentration in outdoor air pollution in Seoul, Korea between 1995 and 1998. CO levels ranged from 0.4 to 3.4 ppm (average = 1.2 ppm).
Maisonet et al,[27] 2001	This study found a significant relationship between the frequency of low birth weight among neonates and CO concentration in outdoor air pollution in Northeastern US cities between 1994 and 1996.
Morris et al,[28] 1995	This study found a significant relationship between the number of hospital admissions for congestive heart failure among people aged 65 and older and CO concentration in outdoor air pollution in 7 US cities between 1986 and 1989. The relative risk of hospital admission for congestive heart failure associated with an increase of 10 ppm in the daily maximum 1-hour CO level ranged from 1.10 in New York to 1.37 in Los Angeles.
Ritz & Yu,[29] 1999	This study found a significant relationship between the frequency of low birth weight among neonates and CO concentration in outdoor air pollution in Los Angeles between 1989 and 1983. CO levels ranged from 0.65 to 6.70 ppm (average = 2.24 ppm).
Stieb et al,[30] 2009	This study found a significant relationship between the number of hospital admissions for myocardial infarction/angina and CO concentration in outdoor air pollution in 7 Canadian cities during the 1990s and early 2000s. For each 0.7 ppm increase in the daily 24-hour average CO level, there was a 2.6% increase in hospital admissions for myocardial infarction/angina.
Yang et al,[31] 2004	This study found a significant relationship between the number of hospital admissions for cardiovascular disease and CO concentration in outdoor air pollution in Kaohsiung, Taiwan between 1997 and 2000. CO levels ranged from 0.2 to 1.7 ppm (average = 0.8 ppm).
Yang et al,[32] 2008	This study found a significant relationship between the number of hospital admissions for congestive heart failure and CO concentration in outdoor air pollution in Taipei, Taiwan between 1996 and 2004. CO levels ranged from 0.12 to 3.66 ppm (average = 1.26 ppm).

APPENDIX B

EVIDENCE SUPPORTING THE WHO'S RECOMMENDATION: CO DOSE-RESPONSE STUDIES

Study	Details
Adams et al,[33] 1988	This study examined the effect of CO inhalation on exercise capacity in adults with coronary artery disease. The average level of COHb was 6%. Average exercise duration was significantly lower in subjects who inhaled CO compared to those who inhaled air, and subjects who inhaled CO were more likely to experience angina earlier during exercise.
Allred et al,[34] 1989 Allred et al,[35] 1991	These studies examined the effect of CO inhalation on exercise capacity in adults with coronary artery disease. The postexercise level of COHb ranged from 2% to 3.9%. Following CO exposure, the amount of time until ST-segment change (an indicator of heart disease) and the amount of time until the onset of angina were reduced during exercise.
Anderson et al,[36] 1973	This study examined the effect of CO inhalation on exercise capacity in adults with angina. The average level of COHb ranged from 2.9% to 4.5%. Following CO exposure, average exercise duration prior to the onset of pain was reduced, the duration of pain was prolonged (with 100 ppm CO, but not 50 ppm CO) exposure, and ST-segment changes were worsened.
Ekblom & Huot,[37] 1972	This study demonstrated that, during maximal treadmill exercise, CO inhalation decreased maximum physical performance and maximum oxygen uptake in healthy adults. COHb levels ranged from 7% to 20%.
Horvath et al,[38] 1975	This study examined the critical threshold at which COHb levels influence maximum aerobic power (VO^2 max) in healthy adults. The results demonstrated that the threshold for significant VO^2 max reduction was approximately 4.3% COHb.
Kleinman et al,[39] 1989	This study examined the effect of CO inhalation on exercise capacity in adults with angina. The average COHb level prior to CO exposure was 1.5%, while the average post-CO exposure level was 3.0% (after 1 hour exposure to 100 ppm CO). Following CO exposure, average exercise duration prior to the onset of pain was reduced and oxygen uptake was reduced.
Kleinman et al,[40] 1998	This study examined the effect of CO inhalation on exercise capacity in adults with angina. Following CO exposure, average exercise duration was reduced, oxygen uptake was reduced, and the average exercise duration prior to the onset of pain was reduced.
Pirnay et al,[41] 1971	This study found that CO inhalation significantly increased heart rate while reducing maximum oxygen uptake during moderate exercise in healthy adults.
Vogel & Gleser,[42] 1972	This study examined oxygen transport in healthy adults at rest and during exercise when exposed to 225 ppm CO-air mixture. During exercise, maximal O^2 uptake was reduced by 23% following CO exposure.

APPENDIX C

EVIDENCE SUPPORTING THE WHO'S RECOMMENDATION: RETROSPECTIVE, CASE, AND ANIMAL STUDIES

Study	Details
Devine et al,[43] 2002	This case study discussed a case of CO poisoning in a 45-year-old woman. Exposure was due to a leak in a kitchen where she worked as a cook for at least one year before the leak was discovered. She had difficulty reading, writing, speaking, and with word retrieval. Brain imaging revealed bilateral lesions of the basal ganglia.
Ely et al,[44] 1995	This case study described CO poisoning in 30 warehouse workers. Exposure was due to inhalation of exhaust from a propane-fueled forklift. The workers demonstrated variable degrees of confusion and difficulties concentrating.
Foster et al,[45] 1999	This case study discussed a case of CO poisoning in a female infant. Exposure was due to a kerosene space heater. At 3 to 4 weeks old, the child was hospitalized; she exhibited loose stools, cough, labored breathing, respiratory distress, and wheezing. At 2 months old, she again had breathing difficulties and wheezing. The mother administered nebulized albuterol, but the infant developed apnea, circumoral cyanosis, and stiffness of the extremities and the father had to administer CPR. At 3 months old, the infant was again hospitalized due to respiratory distress and apnea. After this hospitalization, the fire department checked the CO levels in the house and found CO levels of 0.43% near the space heater and 0.13% in the infant's bedroom.
Keles et al,[46] 2008	This retrospective study examined 323 emergency room patients with CO poisoning between 2002 and 2003. The most common symptoms included headache, nausea, dizziness, fainting, and seizures. Average COHb level was 26.3 ± 11.5%.
Khan & Sharief,[47] 1995	This case study discussed a case of CO poisoning in two children, ages 4 and 5. Exposure was due to a partial blockage of the exhaust outlet for a family's living room heater. The children were brought to their family physician due to recurrent headaches, lethargy, sleepiness, and mood abnormalities. The children's COHb levels were 20% and 13%.
Myers et al,[48] 1998	This case study discussed 7 cases of chronic CO poisoning with exposure durations ranging from 3 months to 3 years of exposure. These cases were: 1.) A case of a 23-year-old woman with an 18-month exposure to CO from a faulty clothes dryer hookup. She exhibited fatigue, headaches, fever, throat pain, nausea, diarrhea, heart palpitations, sleep problems, weight loss, tinnitus, chest pain, irritability, emotional lability, depression, difficulty with arithmetic, blurred vision, and ataxia. 2.) A case of a 36-year-old woman with combined methylene chloride and CO poisoning due to open cans of oil-based paint next to her furnace. Her symptoms included neck and shoulder spasms, fatigue, chest pain, nausea, headaches, tinnitus, sensitivity to light, reductions in short-term and long-term memory, slow thought, and difficulty with arithmetic. 3.) A case of a 44-year-old woman with an 8-month exposure to CO in her home resulting from a faulty water heater ventilation system. The woman had a history of personality disorder and possible temporal lobe seizures, headaches, and irritability. During the period of CO exposure, she developed extreme paranoia, increased irritability, difficulty with arithmetic, difficulty remembering words, impaired short-term memory, nausea, vomiting, worsening of headaches, and disorganized thoughts. 4.) A case of a 67-year-old man with a 3-month CO exposure. Exposure was due to a wood-burning stove in his garage. During the period of exposure, he experienced bad dreams, headaches, dizziness, tinnitus, and arm numbness. His symptoms persisted for 2 months after discontinuing use of the stove.

APPENDIX C Continued

Study	Details
Myers et al,[48] 1998 continued	5.) A case of a 63-year-old woman with CO exposure between 1963 and 1965. During that period of exposure, she was diagnosed with a psychiatric disorder and severe depression. Between 1988 and 1989 she was re-exposed due to a faulty furnace ventilation system and faulty chimney fireplace in her condominium. She was exposed to CO for approximately 16 months. Her symptoms included fatigue, nausea, headaches, disorientation, short-term memory loss, difficulty with arithmetic, difficulty driving and following directions, depression, and emotional lability. 6.) A case of a 43-year-old woman who was exposed to CO for approximately 1 year due to an improperly installed furnace. Her symptoms included weight loss, muscle twitches, headache, nausea, and dizziness. One year after the diagnosis, she reported continued symptoms such as muscle weakness, joint pain, urinary incontinence, memory difficulties, breathing difficulties, and tremors. 7.) A case of a 43-year-old woman exposed to CO in her condominium for 3 months due to a faulty gas furnace. Her symptoms included headaches, nausea, vomiting, dizziness, coughing, and shortness of breath, poor balance, falling, and sensitivity to light. Two months after moving into her condominium, she experienced blurry vision that was serious enough that she had difficulty passing the vision portion of her driving test, despite having had no prior vision difficulties. She also experienced difficulties with reading and reading comprehension, memory recall, and simple arithmetic.
Pinkston,[49] 2000	This case study discussed a case of CO poisoning in two middle-aged adults. Exposure was due to a faulty furnace, which resulted in daily CO exposure over a period of 3 years. Formal neuropsychological tests revealed disorganization, indecisiveness, mental passivity, and problems in planning and goal formulation. PET brain imaging revealed frontal cortex, hippocampus, temporal lobe, visual cortex, and somatosensory cortex abnormalities.
Prockop,[50] 2005	This case study described a case of CO poisoning in 9 people due to a faulty gas heater in an apartment building. The 9 individuals' COHb levels varied from 14.2% to 56%. One of the individuals died, while 3 others were in a coma following the exposure. Follow-up symptoms included impaired attention, problem solving, reduced abstract thinking, intellectual impairment, and Parkinsonian symptoms.
Ryan,[51] 1990	This case study discussed a case of CO poisoning in a 48-year-old woman. Exposure was due to a furnace that had been releasing 180 ppm CO. The woman had a 3-year history of constant headaches, lethargy, and memory problems. She had no problem remembering distant past but did have difficulty remembering new information. Occasionally, she also had periods of mental confusion and depression. After the furnace was replaced, her memory problems persisted. Formal neuropsychological tests confirmed her reports of verbal and visual memory impairments.
Thom,[52] 2004	This study in laboratory rats found that CO exposure in rats (1,000 ppm for 40 minutes followed by 3,000 ppm for 20 minutes) impaired learning.
Thyagarajan et al,[53] 2003	This case study described a case of CO poisoning in a 37-year-old woman. Exposure was due to a faulty heating appliance. The woman was exposed daily for over 7 years. She experienced persistent tiredness, headache, cognitive and personality changes, and depression. A brain MRI revealed abnormalities in the basal ganglia and hippocampus.

APPENDIX D

LIST OF ABBREVIATIONS

APU: Auxiliary Power Unit
A device on a vehicle that serves as an additional energy source for non-propulsion related functions. APUs are commonly gas-powered.

ATSDR: Agency for Toxic Substances and Disease Registry
A federal agency that is part of the US Department of Health and Human Services.

CO: Carbon Monoxide
A colorless, tasteless, and odorless gas produced by the burning of carbon-based fuels (wood, gas, petroleum, etc.) with the potential to cause significant human harm and death.

COHb: Carboxyhemoglobin
A biological complex formed in mammals when carbon monoxide binds to red blood cells. Our bodies all produce some amount of endogenous (natural) CO and, therefore, we all have some level of COHb in our bodies. COHb is commonly used as a marker for CO poisoning given that CO poisoning increases COHb levels. However, there are several problems with using COHb as an indicator of CO exposure. For example, cigarette smoking can significantly increase COHb levels[55,56], air pollution can increase COHb levels[57,58], and CO poisoning symptoms and COHb levels are often poorly correlated, especially in cases of low-level CO exposure and in smokers.[59] Moreover, by the time individuals are seen by a doctor (either through emergency room admission or through a scheduled visit with a primary care physician), they have often been removed from the source of CO for long enough that their COHb levels have dropped significantly.

CPSC: Consumer Product Safety Commission
An independent US government agency. The CPSC works to prevent injury from consumer products, develops safety standards for consumer products, and conducts research related to the safety of consumer products.

EPA: Environmental Protection Agency
An independent US government agency that focuses on environmental protection.

OSHA: Occupational Safety and Health Administration
A federal agency that is part of the US Department of Labor

PPM: Parts per million
A unit used to measure the number of gas particles of a particular type that exists in a mixture, per one million total gas particles.

UL: Underwriters Laboratories
A global safety certification company that has developed over 1,000 product standards in the US. UL 2034 is the UL guideline for current carbon monoxide alarm standards.

WHO: World Health Organization
An agency of the United Nations responsible for international public health.

APPENDIX E

FURTHER READINGS

1. Penney, D. G. (Ed.). (2019). Carbon monoxide. CRC Press.
2. Penney, D. G. (Ed.). (2007). Carbon monoxide poisoning. CRC Press.
3. Penney, D. G. (Ed.). (2000). Carbon monoxide toxicity. CRC Press.
4. Penney, D.G. et al. (1999). Environmental Health Criteria 213: CARBON MONOXIDE, 2nd ed., World Health Organiza tion.
5. All-Party Parliamentary Carbon Monoxide Group. (2015). Carbon Monoxide: From awareness to action. Available from: https://www.policyconnect.org.uk/media/1008/download

ADDENDUM

2021 WHO AIR QUALITY GUIDELINES

Shortly after the initial publication of this document, the WHO published updated air quality guideline (AQG) levels for multiple indoor and outdoor air pollutants, including CO.[65] In the report, the WHO recommends a 24-hour AQG of 4 mg/m^3 (3.49 ppm, assuming a temperature of 25°C) for CO. They further recommend an interim target of 7 mg/m^3 (6.11 ppm, assuming a temperature of 25°C). Interim targets are proposed levels that serve as intermediate goals for pollution reduction and are intended for use in areas with high pollution levels.