Residential Indoor Air Quality Guideline

CARBON MONOXIDE

Physical and chemical properties

Carbon monoxide is a tasteless, odourless and colourless gas at room temperature. Carbon monoxide can be produced by both natural and anthropogenic processes. It is usually formed during the incomplete combustion of organic materials.

Molecular formula	CO
Molecular weight	28 g/mol
Melting Point	-199°C
Boiling Point	-191.5°C
Conversion: ppm → mg/m^3	1.145 @ 25°C

Sources and concentrations in indoor environments

Extensive reviews on sources and concentrations of carbon monoxide have been published by the World Health Organization (WHO, 1999) and the United States Environmental Protection Agency (U.S. EPA, 2000). Carbon monoxide in the indoor environment occurs directly as a result of emissions from indoor sources (e.g., vented and unvented combustion appliances, tobacco smoke and the burning of incense) or indirectly as a result of infiltration indoors of outdoor air containing carbon monoxide (e.g., from vehicles in attached garages and busy roads nearby).

Results from studies published on the subject indicate that the presence or the use of specific sources lead to increased carbon monoxide concentrations indoors. In the absence of an indoor source, carbon monoxide concentrations are generally equivalent to average outdoor concentrations. The indoor sources, emissions and concentrations of carbon monoxide are sufficiently diverse.

The factors affecting the introduction, dispersion and removal of carbon monoxide indoors include (Health Canada, 2010):

- type, nature (factors affecting the generation rate of carbon monoxide) and number of sources;
- source use characteristics;
- building characteristics;
- infiltration or ventilation rates;
- air mixing between and within compartments in indoor spaces;
- removal rates and potential remission or generation by indoor surfaces and chemical transformation;
- existence and effectiveness of air contaminant removal systems; and
- outdoor concentrations.

Health effects

In human controlled exposure studies, carbon monoxide at low doses appears to cause a reduction in the maximum length of exercise and physical performance upon effort in healthy subjects at carboxyhaemoglobin (COHb) levels of 2.3% and 4.3%, respectively (Drinkwater et al. 1974; Raven et al. 1974; Horvath et al. 1975).

However, the data best describing the effects of carbon monoxide at low concentrations are still those for individuals diagnosed with coronary disease. Effects in these individuals include a reduction in ST segment change time on their electrocardiogram (a sign of myocardial ischemia) following carbon monoxide exposure resulting in a COHb level of over 2.4% (Allred et al. 1989b; Allred et al. 1989a; Allred et al. 1991); a reduction in the duration of the exercise caused by chest pains following carbon monoxide

exposure resulting in a COHb level of over 3.0% (Anderson et al. 1973; Sheps et al. 1987; Adams et al. 1988; Kleinman et al. 1989; Kleinman et al. 1998; Allred et al. 1989b; Allred et al. 1989a; Allred et al. 1991); and an increase in the number and complexity of arrhythmia following carbon monoxide exposure resulting in a COHb level of 6.0% or higher (Sheps et al. 1990).

Based primarily on the controlled exposure studies involving subjects diagnosed with coronary disease (the subgroup most sensitive to effects of carbon monoxide), analysis of the scientific data strongly suggests that, in order to protect the entire population, the increase in COHb level that must not be exceeded should be on the order of 1.5%, from a base level of 0.5% to 1.0% in non-smokers, resulting in a value of 2.0% to 2.5% (Health Canada, 2010).

Assessment under the *Canadian Environmental Protection Act, 1999*

Carbon monoxide is listed on the Domestic Substances List of the *Canadian Environmental Protection Act (CEPA), 1999* and has been found to meet the categorization criteria under section 73. A screening-level risk assessment following categorization has not yet taken place.

RESIDENTIAL INDOOR AIR QUALITY GUIDELINE FOR CARBON MONOXIDE

A common source of carbon monoxide exposure is tobacco smoke (mostly in smokers, but also in non-smokers as second-hand smoke). Smokers therefore have higher baseline COHb levels, and they may be exhaling more carbon monoxide into the air than they are inhaling from the ambient environment. They may even have adaptive responses due to their elevated COHb levels and, as a consequence, it is not clear whether the increases in COHb levels due to carbon monoxide exposure and their chronic COHb levels caused by smoking would be additive. Hence, the guideline is recommended for the protection of non-smokers (Health Canada, 2010).

The review of human controlled exposures studies strongly suggests that, in order to protect health, COHb levels not exceed 2.0% in blood. As this maximum COHb level was derived from studies of individuals with coronary disease, the most sensitive subgroup, it should also be protective of the entire population.

The toxicokinetic model developed by Gosselin et al. (2006) was then used to determine the carbon monoxide concentrations in air that would result in COHb levels of 2.0% in blood, under a scenario of light exercise, for different age and gender groups. This toxicokinetic model integrates the main features of the Coburn-Forster-Kane (CFK) equation (Coburn et al., 1965), but is more conservative and is considered a more precise method.

Exposure limits for 1-hour and 24-hour averaged exposure times were derived in order to protect the entire population for both short- and long-time exposures to carbon monoxide. The recommended maximum exposure limits for carbon monoxide, derived from the toxicokinetic model described above, are presented in the table below, along with the critical health effects on which they were based. Exposure to indoor air concentrations above these limits may result in COHb levels in blood above 2.0%, and potential health effects.

Residential Maximum Exposure Limits for Carbon Monoxide

Averaging time	Concentration		Critical Effects
	mg/m^3	ppm	
1h	28.6	25	• Reduction in maximum duration of exercise in health individuals
24h	11.5	10	• Reduction in ST segment in change time (sign of myocardial ischaemia) in individual with coronary artery disease

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