SEG/SUM/57
October 1995

Recommendation from the Scientific Expert Group

on Occupational Exposure Limits

for Carbon monoxide

8 hour TWA	:	20 ppm (23 mg/m^3)
STEL (15 mins)	:	100 ppm (117 mg/m^3)
Additional classification	:	-

Substance:

Carbon monoxide **CO**

Synonyms	:	Carbon oxide, carbonic oxide		
EINECS N°	:	211-128-3		
EEC N°	:	006-001-00-2	Classification :	F; R12 T; R23
CAS N°	:	630-08-0		
MWt	:	28.01		
Conversion factor (20°C, 101 kPa)	:	1.17 mg/m^3 = 1 ppm		

Occurrence/use:

Carbon monoxide is a colourless odourless gas. It has a MPt of -199°C, a BPt of -191.5°C and a vapour pressure of <101 kPa at 20°C. The vapour density is 1.25 g/l at 0°C and it is explosive in the range 12.5 - 74.2% in air.

CO occurs in the atmosphere due to emission from natural sources such as atmospheric oxidation of methane, emission from oceans, marshes, forest fires, rain water and electrical storms or produced by human activities as a combustion product of organic material and industrial processes. The major source of CO emission from human activity is the incomplete combustion of motor vehicle fuels. The average concentration of CO in the atmosphere ranges from 0.06 to 0.4 ppm (0.07-0.5 mg/m^3) in the northern hemisphere. Total world-wide emission of carbon monoxide has been estimated to exceed 10^9 tonnes per annum of which about 10% may result from human activities.

The main sources of CO in occupational exposure are iron foundries, gas industries, coke ovens, distribution and use of natural gases, car manufacture and service stations. Urban CO pollution contributes to occupational exposure, particularly in high traffic areas. Peak concentrations in urban pollution may reach 150 ppm (176 mg/m^3).

Health Significance:

CO is rapidly absorbed from the alveoli of the lungs (Beard, 1982). In the blood it competes with O_2 in binding to the bivalent iron atom of haemoglobin, forming carboxyhaemaglobin (HbCO). The concentration of HbCO generated by the end of an 8h working day can be related simply to the atmospheric CO content using the formula:

$$\% \text{ HbCO} = 0.4 + p/7$$

where p = [CO] in inhaled air (ppm). This is valid for atmospheric CO concentrations up to 50 ppm (59 mg/m^3) (NAS, 1977).

CO has an even greater affinity for myoglobin, which is responsible for gas transport in muscle. CO is eliminated unchanged through the lungs with less than 1% oxidised to CO_2. Elimination is lower in aged persons, and faster in women than in men.

The critical effects of CO are on the brain, cardiovascular system and fetus. Acute intoxication, following exposure to more than 1000 ppm (1170 mg/m^3), produces progressive hypotension as a consequence of peripheral vasodilation. Heart rate increases initially then declines as HbCO saturation increases (Penney, 1990). In rabbits the threshold for myocardial damage has been established as 4 to 24h exposure to 50 ppm (59 mg/m^3) CO, which produces 4-5% HbCO saturation levels. (Thomsen and Kjeldsen, 1974). Effects on brain in the rat occur following 1 to 2 month's exposure at 50 ppm (59 mg/m^3) (Vyskocil *et al*., 1984).

A level of 46% HbCO is considered to produce fetal death (Weiler *et al*., 1983). Increased mortality and decreased birthweight were observed following exposure of pregnant rabbits to 90 and 180 ppm (105 and 210 mg/m^3) CO, corresponding to maternal HbCO concentrations of 9-19%, for 30 days (Astrup *et al.,* 1972).

There are no available data on carcinogenicity, mutagenicity or modification in the immune response.

Normal HbCO concentration in the blood of non-smokers is 0.5%, which is attributable to endogenous production from porphyrin metabolism and membrane peroxidation. No observable effects have been demonstrated in humans at concentrations under 2% HbCO. Above 2.5%, produced by 90 min exposure to 50 ppm (59 mg/m^3) CO, some authors have reported modifications of psychological and psychomotor functions (Fodor and Winneke, 1972; Groll-Knapp *et al.*, 1972, Beard and Wertheim, 1967). Other authors were unable to reproduce these results, even at higher HbCO concentrations and using more comprehensive studies with well controlled exposure (O□Donnel *et al*., 1971a, 1971b; Stewart *et al.,* 1970, 1973). At 5% levels there are observable cardiovascular effects (Zenz, 1979; Balazs *et al*., 1986). Continuous exposure to CO accelerates previous pathological conditions; it exacerbates ischaemia and vascular effects especially in coronary arteries (Atkins and Baker 1985; Ebisuno *et al*., 1986). These effects are likely to occur above 4% HbCO and, in the case of angina pectoris, above 2.7% (Anderson *et al*., 1973; Aronow and Isbell, 1973; Aronow *et al*., 1974; Sheps *et al.*, 1990). In young adults under physical exercise, a decrease in maximum O_2 consumption is produced with 5% HbCO concentrations (Horvath *et al*., 1975).

Exposure to CO may represent a particular risk for the pregnant woman and for the fetus. In pregnant women, endogenous CO production is approximately twice as high as in non-pregnant women, resulting in an average level of 0.5 to 1 % HbCO (Astrup *et al.,* 1972). In the fetus, the HbCO blood concentration is about 15 % higher than in maternal blood (Hill *et al.,* 1977). Together with an already normally low pO_2, a critical fetal HbCO concentration can easily be achieved, resulting in a hindered O_2 exchange in the placenta (Longo, 1970). Data on fetal effects at maternal HbCO concentrations of about 2-3% are not available; however, at this level a fetal risk is not expected.

Recommendation:

The studies quoted above, demonstrating that changes in CNS activity and susceptibility to cardiovascular disease start to increase when the concentration of HbCO is in the region of 5%, indicate that the limit value should not produce an HbCO concentration in excess of 4%. This correlates approximately with 30 ppm (35 mg/m^3) CO in the workplace for average exposures of 8h. Taking into account the preferred value approach, the recommended 8-hour TWA is 20 ppm (23 mg/m^3). A STEL (15 mins) of 100 ppm (117 mg/m^3) was proposed to limit accumulation of HbCO.

No "skin" notation was considered necessary.

At the levels recommended, no measurement difficulties are foreseen.

Even at a CO exposure of 20 ppm (23 mg/m^3), resulting in an HbCO concentration of about 3%, effects on groups at at extra risk, such as people with cardiovascular disease and pregnant women, cannot be ruled out.

Key Bibliography:

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