

Reducing the Risk of Accidental Death Due to Vehicle-Related Carbon Monoxide Poisoning

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ABSTRACT

Emissions of carbon monoxide (CO) from motor vehicles cause several hundred accidental fatal poisonings annually in the United States. The circumstances that could lead to fatal poisonings in residential settings with motor vehicles as the source of CO were explored. The risk of death in a garage (volume = 90 m³) and a single-family dwelling (400 m³) was evaluated using a Monte Carlo simulation with varying CO emission rates and ventilation rates. Information on emission rates was obtained from a survey of motor vehicle exhaust gas composition under warm idle conditions in California, and information on ventilation rates was obtained from a summary of published measurements in the U.S. housing stock. The risk of death ranged from 16 to 21% for a 3-hr exposure in a garage to 0% for a 1-hr exposure in a house. Older vehicles were associated with a disproportionately high risk of death. Removing all pre-1975 vehicles from the fleet would reduce the risk of death by one-fourth to two-thirds, depending on the exposure scenario. Significant efforts have been made to control CO emissions from motor vehicles with the goal of reducing CO concentrations in outdoor air. Substantial public health benefit could also be obtained if vehicle control measures were designed to take account of acute CO poisonings explicitly.

IMPLICATIONS

Significant efforts have been made to control CO emissions from motor vehicles with the goal of reducing outdoor CO concentrations. An important, but apparently incidental benefit of these controls is that the rate of accidental deaths from motor vehicle related CO poisoning has declined. Nevertheless, motor vehicle emissions still cause several hundred accidental deaths annually in the United States. Substantial public health benefit might be obtained if CO emission control measures for motor vehicles were designed to take explicit account of the risk of acute CO poisoning.

INTRODUCTION

Carbon monoxide (CO) is a highly toxic gas, emitted from incomplete combustion of carbonaceous fuels. When inhaled, CO binds reversibly with blood hemoglobin, impairing the transport and delivery of oxygen to body tissues. In the United States, CO is designated as a "criteria pollutant." States and local air districts monitor concentrations of criteria pollutants in outdoor air and, if concentrations exceed standards, develop plans to reduce airborne concentrations. The National Ambient Air Quality Standard (NAAQS) for CO is 9 ppm, based on an 8-hr average, and 35 ppm based on a 1-hr average. As of September 1997, the U.S. Environmental Protection Agency (EPA) designated 29 "nonattainment" areas for CO; about 5% of the U.S. population was living in counties that failed to meet the CO standard in 1996.¹

In the United States, motor vehicles dominate total anthropogenic emissions of CO, contributing 79% of the total estimated emissions of 90×10^9 kg y⁻¹ in 1996.¹ Considerable efforts have been made to reduce CO emissions from motor vehicles. Key control techniques include the use of catalytic converters on automotive exhaust; automotive inspection and maintenance programs; and use of reformulated fuels. These efforts have led to a significant downward trend in outdoor CO levels in most urban areas over the past decade.¹ A second important dimension of the CO problem is accidental fatal poisonings. A few hours of exposure to 1,000 ppm CO causes a blood carboxyhemoglobin (COHb) saturation of the order of 60%, which can be fatal.^{2,3} A published review of death certificates indicates that auto exhaust caused 6,600 unintentional CO poisoning deaths in the United States during 1979–1988.⁴ In 1988, vehicle exhaust caused 500 unintentional CO poisoning fatalities. Evidence suggests a beneficial effect of stricter CO emission standards on this death rate.⁵ Over the 1979–1988 period, the number of accidental deaths due to CO poisoning from vehicle exhaust dropped by about 7% per year.⁴ This period coincides with the widespread introduction of substantially

improved emission control systems into the motor vehicle fleet.

Accidental CO poisonings can occur in a variety of circumstances. In a study of unintentional deaths from CO poisonings in California, death certificates and coroners' reports from 1979 to 1988 were evaluated to determine location and cause of poisoning.⁶ Among 128 evaluations and reports of accidental CO poisonings due to vehicle exhaust, 80 incidents took place in a garage or house, 38 involved a vehicle parked outside, 4 occurred while driving, and the circumstances of the remaining deaths were unspecified. In cases where the circumstances of death were known, the most common cause of poisoning was driving into the garage (the driver was often under the influence of alcohol or other drugs) and leaving the car running (42% of deaths). Starting the car to perform vehicle maintenance (25%) or to provide heat (23%) were the next most frequent causes of death.⁷ In colder climates, it may be more common for people to start a vehicle in an attached garage in the morning to warm it up. If they return to the house and forget that the vehicle is running, accidental poisoning may occur.

Shelef⁸ has argued that reducing CO poisoning deaths may be the biggest benefit from current motor vehicle emission control programs, even though such programs are motivated by concentration standards for outdoor CO. Effective means to further reduce this death rate may exist. To establish a foundation for evaluating control strategies, a clearer understanding is needed of the circumstances in which motor vehicles may cause fatal CO poisonings.

In this paper, mathematical models are combined with experimental data to explore scenarios in which motor vehicle emissions may lead to fatal CO poisonings. Four key elements are employed in this investigation: (1) measured CO emissions from idling motor vehicles; (2) volume and ventilation rate data for residences and garages; (3) a material-balance-based indoor air quality model; and (4) a model for the formation of blood COHb as a consequence of inhalation exposure to CO. By performing a Monte Carlo simulation of CO poisoning, the risk of death due to exposure to CO from vehicle exhaust is evaluated.

METHODS

The first step in the analyses presented in this paper was to determine the indoor concentration of CO as a function of time. Motor vehicles were assumed to be operated within a well-mixed enclosed residential space with a volume of either 90 m³ and 400 m³. These are typical volumes for a two-car garage (5 m × 6 m × 3 m) and for a home with four occupants,⁹ respectively. In the second case, the source of CO would be an idling vehicle in an

attached garage. Emissions of CO from the vehicle were assumed to be constant at a rate E (g min⁻¹). Information on E for the motor vehicle fleet was obtained from random roadside surveys of motor vehicle exhaust gas composition under warm idle conditions in California.^{10,11} The indoor CO concentration rises from an initial value of zero toward a steady-state level given by a balance between the rate of emission and the rate of removal by ventilation. Information on residential ventilation rates was obtained from summaries of published measurements in the U.S. housing stock.¹² The CO emission rate and the building air-exchange rate (Q/V , where Q is the volumetric flow rate of ventilation air) were treated as independent random variables in the analysis.

A mechanistic biochemical model^{13,14} was applied to predict the accumulation of blood COHb that results from inhaling the time-varying indoor CO concentration. A fatal poisoning was considered to have occurred whenever the fractional saturation of blood hemoglobin by COHb reached 60%. For specified values of the CO emission rate, building volume, air-exchange rate, and characteristics of the exposed individual, the blood COHb level can be predicted from the coupled indoor air quality and blood COHb models.

Indoor Air Model Applied to CO

CO concentrations were predicted as a function of time by applying an indoor air quality model.¹⁵ The indoor space was assumed to be well-mixed. The governing equation for indoor CO concentration states that the rate of mass accumulation in indoor air is given by the difference between the CO emission rate and the removal rate by ventilation:

$$\frac{d(CV)}{dt} = E - QC \quad (1)$$

Here, V is the interior volume (m³), C is the indoor air concentration of CO (g m⁻³), E is the CO emission rate (g min⁻¹), and Q is the ventilation rate (m³ min⁻¹). If E and Q are constant and the initial CO concentration is zero, then eq 1 has the following analytical solution:

$$C(t) = \frac{E}{Q} \left(1 - \exp\left(-\frac{Qt}{V}\right) \right) \quad (2)$$

The model treats CO as nonreactive and neglects any supply of CO from outdoor air. A similar approach was used to predict time-varying indoor CO₂ and O₂ levels with the additional term of $+QC_o$ in eq 1, where C_o is the species concentration in outdoor air. The initial concentration of these gases was assumed to be the same as their outdoor concentrations: 360 ppm for CO₂ and 21% for

O₂. The time series of predicted concentrations for CO, CO₂, and O₂ were used as inputs to the blood COHb model.

CO Emissions Data

The EMFAC model is used in California to estimate on-road vehicle emissions per unit distance traveled (i.e., g km⁻¹). Unfortunately, EMFAC model predictions are not applicable to idling vehicles because emissions normalized to distance traveled become infinite when vehicle speed is zero. Therefore, roadside emissions data collected in 1991 for 2,283 California vehicles^{10,11} were used to determine idle emission rates in the present study. The random roadside survey should capture a representative sample of vehicles because actual on-road vehicles were selected for the test, and owners did not have the opportunity to adjust their vehicles before the test. For each vehicle, inspectors measured tailpipe exhaust concentrations of CO, CO₂, and hydrocarbons (HC) with the engine idling. The engine speed at idle and engine displacement volume were also recorded. The emissions analyzer was not capable of measuring exhaust CO concentrations above 10%, so this was the maximum reported value, even though in a few cases the true concentration may have been higher. From these data and standard properties of the gasoline, the CO exhaust emissions per unit fuel consumption (g CO emitted per g fuel burned) were determined for each vehicle by carbon balance:¹⁶

$$F_{CO} = \frac{Y_{CO}}{Y_{CO_2} + Y_{CO} + 3 Y_{HC}} \left(\frac{28}{12} \right) w_c \quad (3)$$

where Y_i is the mole fraction of species i in the exhaust and $w_c = 0.87$ is the mass fraction of carbon in the gasoline. The factor of three appearing in the denominator is needed to convert from HC as propane-equivalents to carbon atoms.

The fuel consumption rate at idle, m_f (g min⁻¹), was estimated using an expression developed by Ross and An:¹⁷

$$m_f = \frac{1}{LHV} [a S + b P_b] \quad (4)$$

where $LHV = 44 \text{ kJ g}^{-1}$ is the lower heating value of gasoline, S is engine speed in revolutions per unit time, P_b is the brake-power output of the engine, and a and b are coefficients related to engine size and thermal efficiency, respectively. Since P_b is zero at idle, m_f was estimated using measured idle engine speed and displacement volume (V_d in liters) for each vehicle from the California 1991 random roadside survey, using $a = 0.29 V_d$ (kJ rev⁻¹), as recommended by Ross and An.¹⁷ The engine friction parameter, a , is ~16% higher at idle, but from the late 1970s,

when the engine map tests were conducted, to the early 1990s, improvements in engine efficiency have reduced engine friction by 20–25%.¹⁸ Exhaust emission rates of CO were computed as the product of m_f and F_{CO} , and emission rates of CO₂ were calculated similarly. The rates of O₂ consumption were calculated from the fuel consumption rate, assuming an effective molecular composition of fuel of CH₂.

The distribution of CO emission rates at idle is shown in Figure 1. Emission rates vary widely from 0.01 g min⁻¹ to 10 g min⁻¹ for most vehicles. Twelve percent of vehicles have CO emissions below the limit of detection, 0.01% CO in the exhaust.

Vehicle exhaust concentrations were also measured by the California Air Resources Board in 1994–1995.^{19,20} As part of an inspection and maintenance pilot program, registered vehicle owners were contacted at random and required to participate in laboratory testing of their vehicles, and the same parameters were measured as in the 1991 roadside tests. The 1994–1995 data set included 592 vehicles. These data were compared to the 1991 data to examine the possibility of a decline in emission rates, and therefore in the risk of CO poisoning, over the three-year period.

The distribution of CO emission rates from the 1994–1995 inspection and maintenance program is also shown in Figure 1. The difference between the emission rate distribution in 1991 and in 1994–1995 is small. Two opposing forces influence the evolution of fleet emissions: fleet turnover reduces average emissions because older, typically higher emitting vehicles are replaced with newer, lower emitting ones; and the deterioration of vehicles in the fleet causes an increase in emissions. Differences between mean and median emissions are apparent in Figure 2. Most newer vehicles have low emissions, but the

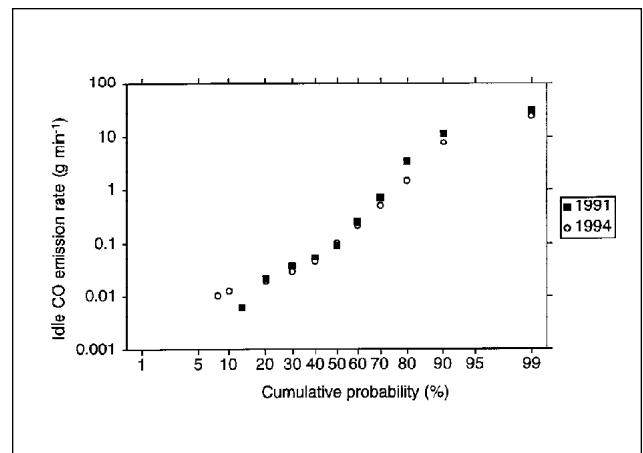


Figure 1. Cumulative probability distribution plotted on log-probability coordinates of CO emission rate under warm-idle conditions from random samples of California light-duty vehicles. Sample size of 2,283 vehicles in 1991^{10,11} and 592 vehicles in 1994–1995.^{19,20}

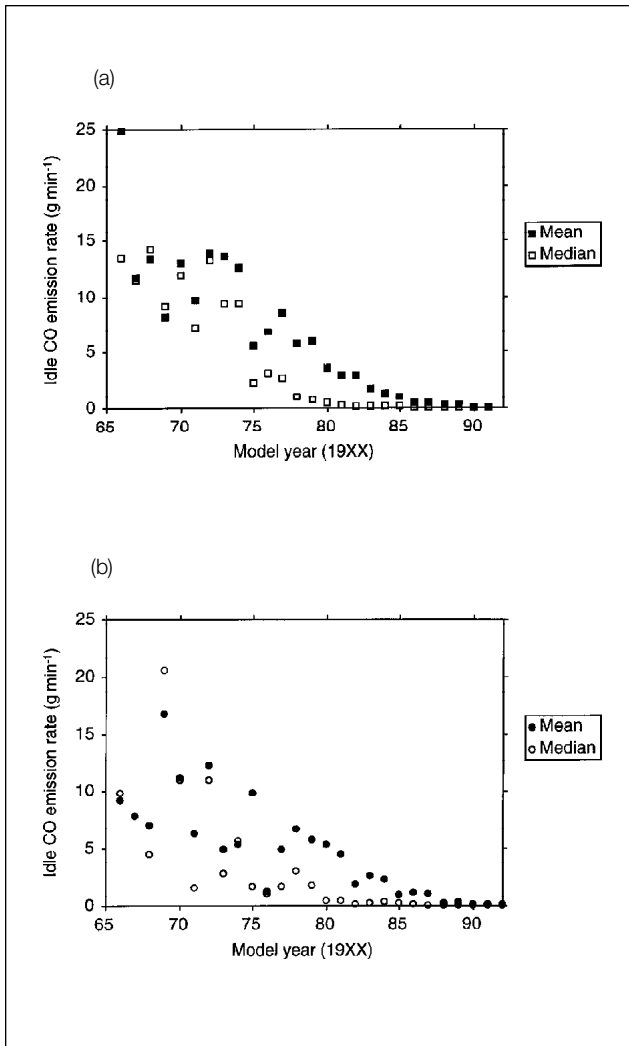


Figure 2. Mean and median warm-idle CO emission rates versus vehicle model year: (a) California 1991 random roadside survey;^{10,11} and (b) 1994-95 inspection and maintenance pilot program.^{19,20}

mean is affected by a minority of malfunctioning and otherwise high-emitting vehicles.

Building Air-Exchange Rates

A comprehensive study by Murray and Burmaster¹² of infiltration and ventilation rates in U.S. housing included measurements of the air-exchange rate of 2,844 residences in 23 states at varying times of the year. The mean air-exchange rate was 0.76 air changes per hour (hr⁻¹). Information on window and door status, heating system type, and presence or absence of air conditioning was not available. The distribution of these residential air-exchange rates is shown in Figure 3, together with the best-fit log-normal distribution. The distribution is well described by a lognormal form with a geometric mean of 0.53 hr⁻¹ and geometric standard deviation of 2.3.

These measurements indicate that air-exchange rates in U.S. housing vary over approximately 2 orders of magnitude, from 0.1 to 10 hr⁻¹, with a median of ~0.5 hr⁻¹.

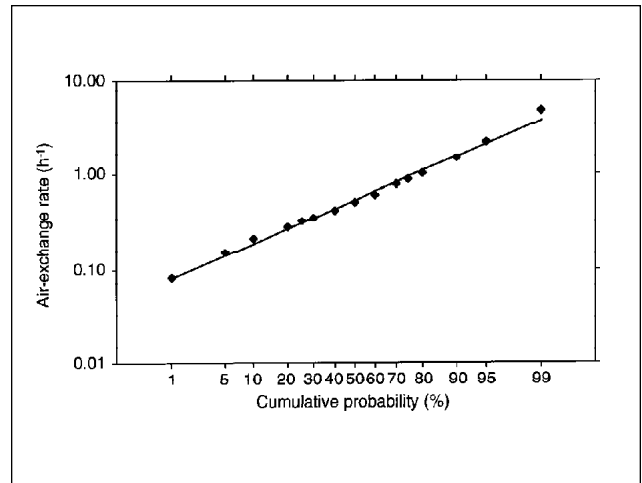


Figure 3. Cumulative probability distribution of air-exchange rates in U.S. residences.¹²

Garage ventilation rates might differ somewhat from residential values, but experimental data are scarce. We know of four studies in which garage ventilation rates were measured.²¹⁻²⁴ The geometric mean of the air-exchange rates in these six garages is 1.6 hr⁻¹, but one of the garages had an air-exchange rate of 13 hr⁻¹. If this outlier is removed from the data set, the geometric mean becomes 1.1 hr⁻¹. To address the uncertainty in the distribution of garage air-exchange rates, we examined two cases: one in which the distribution of garage air-exchange rates was assumed to be the same as that for residences, and one in which garage air-exchange rates were assumed to be three times as high as those for residences. These cases place approximate upper and lower bounds on the risk of death for CO poisoning in a garage.

Modeling Blood COHb

The model for blood COHb level is based on the Coburn-Forster-Kane (CFK) equation,¹³ which accounts for the major physiological variables that control COHb under transient and steady-state conditions: uptake following inhalation, excretion via the lungs, and endogenous production as a catabolic byproduct of heme. The CFK model has been extensively validated by comparison against experimental data, although it may not be accurate at predictions above 50-60% COHb.²⁵

Our analyses utilized a published computer model that implements a numerical solution to the CFK equation.¹⁴ This model incorporates the physiological effects of environmental changes in CO₂ and O₂.²⁶ The computer model also includes necessary physiological data for the exposed subject. For each simulation, time-varying indoor CO, CO₂, and O₂ levels, as predicted by the indoor air quality model, were supplied to the blood COHb model along with specification of the gender, age, height, and weight of the exposed subject. Our test subject was the default

subject in the COHb model, a 25-year-old male of height 178 cm and weight 70 kg. This choice was appropriate because adult males account for ~75% of accidental CO poisonings.⁴ While blood COHb levels are not as predictive of the severity of CO poisoning as are blood lactate levels, classically it has been assumed that COHb levels above 60% are fatal.³ Blood COHb levels above 60% in this analysis were recorded as fatalities, although this assumption approximates a more complex reality. When a patient died due to anoxemia or low tissue pH, predicted COHb values were not output by the model, and these outcomes were recorded as fatalities. The computer model was not designed to handle extremely high COHb values over sustained periods and in these situations, did not output a predicted COHb value or cause of death. These outcomes were also recorded as fatalities.

Monte Carlo Simulation

To generate a frequency distribution of blood COHb levels in subjects exposed to vehicle exhaust indoors, Monte Carlo simulations were performed. Four exposure scenarios were examined: 1 and 3 hr of exposure in a 90 m³ garage, and 1 and 3 hr of exposure in a 400 m³ house. The temperature was assumed to be 20 °C and the pressure 1 atm. Assuming independence of ventilation rates and vehicle emission rates, we sampled from the lognormal fit to the Murray and Burmaster data and at random with replacement from the idle CO emission rate data. Time series of indoor concentrations of CO, CO₂, and O₂ were generated using the indoor air quality model (eq 2). These time series were then input to the blood COHb model, and

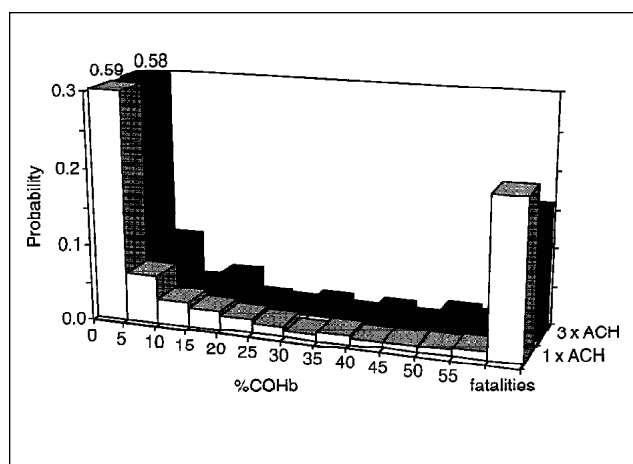


Figure 4. Distribution of predicted blood COHb levels for 3-hr exposure to motor vehicle exhaust emissions in a garage. The white bars represent the upper bound case (unmodified residential air-exchange rate distribution), and the dark bars represent the lower bound case (residential air-exchange rate distribution multiplied by a factor of three). Fatalities occur when COHb > 60%. Fatalities also include deaths due to sustained, extremely high COHb levels, anoxemia, and low tissue pH.

distributions of blood COHb levels were developed for each of the four exposure scenarios defined above. The statistic of interest was the risk of death, computed as the number of fatalities divided by the total number of runs.

The total number of runs required was determined by computing a 95% confidence interval on the risk of death. For a confidence interval of no more than plus or minus 5% of the risk of death, 500–1,500 runs were required, depending on the scenario.

RESULTS AND DISCUSSION

Figures 4 and 5 present results for the most dangerous scenario, 3 hr of exposure to vehicle exhaust in an enclosed garage, and the least dangerous scenario, 1 hr of exposure in a house. The histograms show that the chances of accidental poisoning fatalities occurring are much greater in the first case. In fact, accidental deaths do not occur in the second case, where the highest COHb level achieved is less than 60%. The smaller size of the garage allows CO concentrations to reach higher levels, and the longer exposure time allows greater uptake of CO by the exposed subject. The frequency

Table 1. Risk of death for four accidental poisoning scenarios with all vehicles and with pre-1975 vehicles removed.

Location	Exposure Duration (hr)	All Vehicles	Post-1975 Vehicles Only ^a
garage	1	3.5-7.7% ^b	1.7-5.6% ^b
garage	3	16-21% ^b	12-16% ^b
residence	1	0.0%	0.0%
residence	3	9.5%	3.1%

^a The risk of death was recalculated after removing results involving model year 1974 and earlier vehicles from the simulation.

^b A range in the risk of death in garages is presented because of uncertainty in garage air-exchange rates (see text).

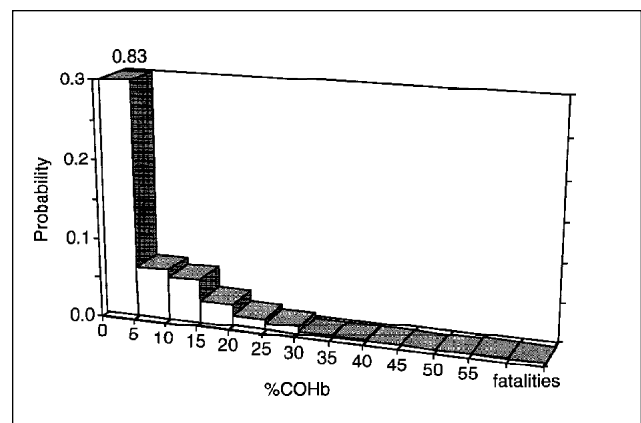


Figure 5. Distribution of predicted blood COHb levels for 1-hr exposure to motor vehicle exhaust emissions in a house.

of high but nonfatal COHb levels is also higher in the garage. In general, victims with blood COHb levels between 20 and 40% can suffer from severe headache, nausea, dizziness, vomiting, and cardiac disturbances. Severe poisonings occur at blood COHb levels between 40 and 60%, and health effects can include fainting, mental confusion, convulsions, and paralysis.³ Almost all of the exposures to CO for 1 hr in a house result in COHb levels below 40%.

Table 1 shows the risk of death calculated for the four different exposure scenarios. A randomly selected light-duty vehicle left idling in a 90 m³ garage for 3 hr has a greater than one in six chance of causing a fatal CO poisoning to an occupant of the garage. The next most deadly scenario is 3 hr in a house, closely followed by 1 hr in a garage. In Table 1, the risk of death in a garage ranges from a lower bound, calculated using air-exchange rates three times the values in the distribution of residential air-exchange rates, to an upper bound, calculated using the distribution without modification.

Older vehicles are predicted to cause a disproportionately high fraction of the deaths. Previous work found that idle CO emission rates must be of the order of 1 g min⁻¹ or higher to cause fatal poisoning in similar scenarios.²⁷ Only a fraction of the motor vehicle fleet emits CO at such a high rate, and that fraction increases strongly with vehicle age. Figure 6 shows the age distribution of all vehicles compared to the age distribution of vehicles responsible for CO poisoning in the 90 m³, 1-hr exposure scenario using the unmodified distribution of residential air-exchange rates. The figure is not significantly different for the case using higher air-exchange rates in the garage. If pre-1975 vehicles were eliminated from the vehicle fleet, the risk of death would decrease from 16–21% to 12–16% in the 3-hr exposure scenario in a garage. This model year was selected as a cutoff because in 1975,

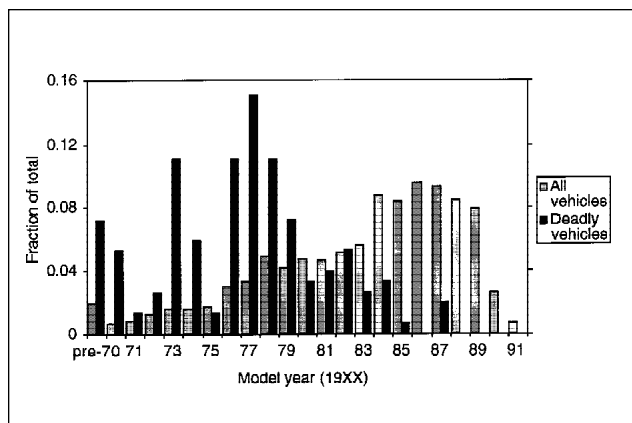


Figure 6. Age distribution of California in-use vehicle fleet in 1991 (light bars) and age distribution of vehicles causing deaths for 1-hr exposure in a garage (dark bars).

the Federal CO emission standard was lowered from 28 to 15 g mi⁻¹ and the California emission standard was lowered from 34 to 9 g mi⁻¹.²⁸ Table 1 shows the results of recalculating the risk of death after removing pre-1975 model year vehicles from the data set. Except for the scenario in which the risk of death is already zero, it decreases by a quarter to two-thirds in the other cases.

Another strategy to reduce accidental CO poisonings would be to send owners of gross-polluting vehicles, as determined by Smog Check, remote sensing, or roadside inspections, information warning them about the dangers of CO poisoning. In this context, gross-polluting vehicles might be defined as those with CO emission rates in excess of 1 g min⁻¹. In the most dangerous scenario, a 3-hr exposure in a garage, over 98% of the fatalities in the Monte Carlo simulation involved CO emission rates of at least 1 g min⁻¹. For the other 2% of fatalities, the emission rate was at least 0.44 g min⁻¹ and the ventilation rate was very low.

Other Issues

In this paper, only poisonings that occur with a vehicle parked indoors have been addressed, but fatal CO poisonings have also occurred in vehicles outdoors. These incidents typically involve a faulty or plugged exhaust system. For example, in a car parked outdoors and idling, exhaust gases may seep into the vehicle cabin through leaks in the exhaust system, or the tailpipe might become clogged with snow or mud. The vehicle may also be moving; 3% of fatal accidental poisonings in California that occurred between 1979 and 1988 were in a moving vehicle.⁷ To model such scenarios, the rate of entry of exhaust gases into the vehicle cabin and the air-exchange rate of the vehicle cabin would have to be known.

The CO emission rate was assumed to be constant in this model, but in reality, it may vary over time. Oxygen depletion in a tightly sealed garage could perturb the air-to-fuel ratio in the engine and cause a clean vehicle to become a gross polluter.²⁹ On the other hand, if a vehicle ran out of gasoline, emissions would drop to zero.

In this model, we have not included the effect of a cold start on CO emissions. When a cold vehicle is started, the fuel-air mixture is intentionally enriched to facilitate ignition and to improve cold engine operation, and the automobile's catalytic converter is not warm enough to function efficiently. During the first few minutes of operation, the vehicle usually emits much higher levels of CO and other pollutants until the catalyst reaches a temperature above 200–370 °C.³⁰ An incremental cold-start emission factor of 16 g CO per start has been measured in an underground parking garage.³¹ In a 90 m³

garage, this extra CO would temporarily increase indoor CO concentrations by up to 153 ppm, and the excess CO would eventually decay away due to ventilation. By itself, this amount of CO would not be enough to kill someone, but it could push blood COHb levels somewhat higher. We ran Monte Carlo simulations for exposure to CO in a garage with the addition of an initial CO concentration of 153 ppm for all vehicles and found that the risk of death did not change for the 1-hr scenario but that it increased from 16–21% to 17–26% for the 3-hr scenario. Additionally, if a cold vehicle is started and then left to idle for an extended period of time, the catalyst may never reach a high enough temperature to operate effectively. The stabilized CO emissions may then remain at a higher level than if the vehicle were operating in a fully warmed mode. This latter effect is not easily modeled and was not included in this analysis.

Another potentially confounding factor in this analysis is the distribution of garaged vehicles versus vehicle age. This study has assumed that all vehicles tested in the random roadside emissions inspection are equally likely to be parked in an enclosed garage, but it is possible that a higher fraction of newer vehicles are kept in garages because of socioeconomic factors. If this were true, then the risk of death from CO poisoning has been overestimated because the older vehicles, which are responsible for a disproportionately high fraction of deaths, would be less likely to be parked in enclosed garages.

Federal standards for light-duty motor vehicle CO emissions have decreased from 51 g mi⁻¹ in 1968 to 3.4 g mi⁻¹ in 1981–1993,²⁸ and the number of accidental deaths from CO poisonings decreased 7% per year from 1979 to 1988,⁴ probably due at least in part to tighter vehicle emission standards. However, this progress in reducing the rate of death from accidental CO poisoning may be slowed by the growing popularity of vehicles with larger engines, for example, sport utility vehicles, in the United States. These vehicles have larger engines and higher fuel consumption rates at idle, and therefore they have a greater potential to become gross polluters and cause CO poisoning if the emission control system deteriorates.

Several control measures might be applied to reduce the risk of CO poisoning: more durable catalytic converters and oxygen sensors; improved vehicle inspection and maintenance programs; CO alarms in garages and residences; and CO sensors on motor vehicles coupled to engine ignition systems. To prevent CO poisoning deaths, it appears particularly important to find and fix (or retire) high-emitting vehicles, or at least to notify owners of the gross polluters about

the high risk of CO poisoning. In fact, greater efforts to educate all vehicle owners about the danger of idling a vehicle indoors might also help reduce the risk of accidental death from CO poisoning.

CONCLUSIONS

The risk of death from CO poisoning has been evaluated for indoor spaces in the presence of an idling motor vehicle. Vehicle emission rates, residential building ventilation rates, and a blood COHb model were combined to study the risk of accidental CO poisonings. For independent, randomly selected vehicles from the 1991 California fleet and nationwide residential air-exchange rates, there is a ~20% chance that an adult male exposed to exhaust emissions from an idling vehicle in a 90 m³ garage for 3 hr will die due to CO poisoning. Vehicle CO emission rates vary widely, and about 25% of California vehicles have the potential to cause accidental poisoning because their CO emission rates exceed ~1 g min⁻¹. Older vehicles are expected to be responsible for a disproportionately large fraction of deaths.

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