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Carbon Monoxide Exposure in Workplaces, Including Coffee Processing Facilities

To the Editor:

We read the recent review of carbon monoxide (CO) poisoning by Rose and colleagues (1) with great interest. To their excellent summary, we would add mention of the burden of occupational

CO exposure: 13% of nonfatal carbon monoxide poisoning cases seen in U.S. emergency departments during a 3-year period were exposed at work (2), and CO exposure is the leading cause of fatality resulting from acute chemical inhalation among US workers (3). Because CO is odorless and tasteless, patients may not be aware of workplace CO exposures. Thus, it is important for clinicians to be familiar with occupational sources of CO.

Occupations that are known to have greater risk for CO poisoning include jobs that require working around combustion sources such as engines and fires and include mechanics, firefighters, longshore workers, diesel engine and forklift operators, and tunnel or toll booth attendants (3). Other occupations that have been largely overlooked but also carry risk for CO exposure include those as varied as warehouse workers, who use propane or diesel forklifts, and restaurant workers, such as charcoal meat grillers and indoor barbeque workers (4).

People who work at coffee processing facilities that make coffee extract are also at risk for CO poisoning (5, 6). Six workers at a coffee extract facility in Japan become symptomatic after they entered or approached a tank that stored coffee extract. One of these workers died and at autopsy was found to have a carboxyhemoglobin level of 26% (5). Subsequent measurement of CO inside the storage tank showed concentrations of 10,000–100,000 ppm, levels at which lethal carboxyhemoglobin saturation could be achieved in as little as a few minutes (5). At a coffee extract facility in the United States, a worker who entered a storage tank collapsed and was initially resuscitated with oxygen on site; his carboxyhemoglobin level was 27% at the hospital. CO concentration inside the tank was found to be 7,500 ppm (6).

We recently evaluated the potential for CO exposure at a coffee processing facility that makes whole bean and ground coffee, rather than coffee extract. Interestingly, the biggest source of CO was not from the roaster, a combustion source, but rather from the roasted beans themselves. High CO levels were observed where coffee beans were stored or ground. Notably, grinding provides a greater surface area for the off-gassing of CO that occurs after roasting (6). We found workers with personal CO exposure levels that ranged from 7 to 30 ppm when averaged over a shift, with a peak CO level of 584 ppm when a worker was standing next to an open hopper of ground coffee. Although not as high as the concentrations inside coffee extract storage tanks, as noted earlier, these exposures approach or exceed the levels recommended by the National Institute for Occupational Safety and Health (7) and, as Rose and colleagues point out, have potential health effects (1).

Recognizing an occupational source of CO has diagnostic and therapeutic implications for a patient with CO poisoning and provides an opportunity for prevention in coworkers. We encourage clinicians to consider workplace CO exposure when evaluating patients with symptoms and signs compatible with CO poisoning. Our data indicate that coffee processors should be added to the list of occupations at risk. ■

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Reply

From the Authors:

We appreciate the important data presented by Dr. Hawley and colleagues regarding occupational exposure to carbon monoxide (CO) in a number of work settings, including coffee processing facilities. As highlighted by Hawley and colleagues, CO is the most frequent cause of acute chemical exposure fatality in the workplace, and although there are myriad sources of CO, the most common exposure is from motor vehicles (1). Generators are also a very common cause of CO poisoning in the occupational setting, accounting for 13% of occupational CO-related deaths from 1992 to 2008 in one study (1–3). Of note, generators, along with gas grills and heaters operated inappropriately indoors, are the main contributors to the increase in CO poisoning during storm-related and natural disaster-related power outages (3).

Fire is one of the most common sources for CO, and perhaps the most lethal (2, 4). In one study looking at risk factors for acute mortality, more than half of CO deaths occurred from a fire source of CO (4). The National Fire Protection Association reports that in 2015, there were 1,345,000 fires reported in the United States, causing 15,700 civilian injuries and 3,280 deaths (5). Inhalational injury occurs concomitantly in more than two-thirds of lethal cases (2).

Almost 15,000 individuals suffer CO poisoning intentionally each year. Between 1999 and 2014, more than 70% of CO related deaths were from intentional versus unintentional poisonings (6). Intentional CO poisoning deaths declined in the past decade, thought to be partially a result of improving regulations for vehicle CO emissions (6), which make it more difficult to asphyxiate in a sealed environment with a motor vehicle running. More than 25% of occupational fatalities from non-fire-related CO poisoning from 1992 to 2008 were a result of intentional poisoning (1).

Education and prevention efforts are critical to controlling CO exposure (1–3). The catalytic converter has reduced CO emissions by automobiles by 75% since 1975, which has reduced unintentional motor vehicle-related CO death rates by more than 80% (2). Although

there have been multiple public campaigns and regulatory measures to increase the placement of CO alarms, there has been no study demonstrating the efficacy of CO alarms to reduce the morbidity or mortality of CO poisoning (2). Although preventing CO exposure is of critical importance, after exposure there remains no quickly administered, widely available, portable antidote for CO poisoning available (2).

For severe cases, in addition to hyperbaric oxygen therapy, discussed in detail in our review, aggressive interventions have been tested in humans, such as extracorporeal membrane oxygenation (2, 7). An ideal treatment would be a portable, quickly administered antidotal agent that could be given immediately at the site of rescue or at the emergency department (2). Roderique and colleagues have presented work in which intravenous hydroxycobalamin can increase the clearance of CO from the bloodstream (2, 8). Our group recently demonstrated a mutationally engineered recombinant neuroglobin molecule that binds to CO avidly, scavenging CO from hemoglobin (2, 9). CO scavenging may have applicability in very severe CO poisoning exposures, as recombinant neuroglobin infusion improved survival significantly by reversing hemodynamic collapse in a lethal model of severe poisoning in mice (2, 9).

The many known and new emerging sources of CO exposure, such as coffee processing facilities, demonstrate the global burden of CO poisoning. Despite increasing safety regulations and public awareness, more than 1,200 individuals die each year from CO poisoning of any source in the United States alone (6). ■

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