

The Effects of Carbon Monoxide and Particulates on the Human Body

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Introduction

In the cookstove community we have now developed the means to accurately measure the concentration of the two primary indoor air pollutants, carbon monoxide (CO) and particulates. In the case of CO this can be done as a function of time, while with particulates an average concentration over a known time can be measured, and in some cases the particulate concentration as a function of time can be measured. The particulate measurements can also be broken down into a number of size ranges. Room ventilation, stratification of pollutant levels, and other factors all have an effect on pollutant concentration, in addition to the pollutant output of a stove.

This paper presents methods for relating these measured pollutant concentrations to health effects for the two primary indoor pollutants. The methods can be readily applied to a variety of situations, and can be used to answer questions such as the following.

1. What will be the health benefits of switching from an unimproved stove to an improved one? Clearly, lower pollutant levels lead to reduced health effects, but how much are the effects reduced? Similarly, can the benefits of increased room ventilation be quantified? Are these benefits worth the effort involved?
2. At what point in a stove's development does one say that the health effects are negligible for typical conditions? At this point, further efforts might be related to improving efficiency. What stoves, if any, are already at this point in their development?
3. What are the health benefits of switching to cleaner burning fuels? Are these benefits worth the effort and expense?

The goal is not to give answers to the above questions that are universally applicable, as there are too many variables that occur in a human body. Rather, the goal is to provide a means to begin to answer the questions above within some reasonable range of uncertainty.

Part 1: Carbon Monoxide

Modeling CO in the Blood

Much research has been performed over recent decades in determining what level of CO in the air leads to what level of CO in the blood, which in turn can be related to health effects. Two seminal papers are listed as Ref.'s 1 and 2. Reference 2 is reported to have given the first successful mathematical relationship between air CO and blood CO, usually noted as [COHb]. [COHb] is read as "concentration of CO in the blood". Equation 1 (sometimes called the CKF equation after the 3 initials of the authors of Ref. 2, or sometimes called the Coburn equation after the lead author) is considered the fundamental equation for relating air CO to [COHb]. It has the capability of taking a time-varying air CO level and determining the [COHb] and includes the effects of altitude and a number of other important parameters. It can also model the decrease in [COHb] after the exposure has ended.

The Coburn equation is:

$$\frac{d(CO)}{dt} = \dot{V}_{CO} + \frac{P_I(CO)}{D_L + \frac{(P_B - 47)}{\dot{V}_A}} - \frac{[COHb]}{[O_2Hb]} \frac{P_c(O_2)}{M} \frac{1}{D_L + \frac{(P_B - 47)}{\dot{V}_A}} \quad (\text{Eq. 1})$$

where:

$\frac{d(CO)}{dt}$ = the rate of change of the quantity of CO in the blood, in ml of gas per minute.

\dot{V}_{CO} = the so-called endogenous production rate of CO. The body produces a small amount of CO as part of the natural metabolic processes. This is in ml of gas per minute.

[COHb] = the concentration of CO in the blood, in ml of gas per ml of blood.

[O₂Hb] = the concentration of oxygen in the blood, in ml of gas per ml of blood.

P_c(O₂) = the pulmonary capillary pressure of oxygen in the blood, in mm Hg

M = the Haldane constant, the ratio of the affinity of blood for CO to the affinity of blood for oxygen.

P_I(CO) = the partial pressure of carbon monoxide in the air that is inspired or inhaled, in mm Hg.

P_B = the atmospheric or barometric pressure in mm Hg. The numeral 47 appears in the above equation along with the barometric pressure because the partial pressure of water

vapor at body temperature is 47 mm Hg. Thus, the quantity ($P_B - 47$) is the partial pressure of dry air inside the lung.

D_L = diffusion capacity of the lungs for CO, in ml of gas per minute per mm Hg of pressure difference.

\dot{V}_A = the alveolar ventilation rate in ml of gas per minute.

To use the above equation, one must make some assumptions. The Haldane constant varies somewhat among people, but only over a fairly small range. Reference 1 uses the number 218, which will be used throughout this study.

The barometric pressure, P_B , will be assumed to be known, and a later section of this report will look at the effects of barometric pressure.

\dot{V}_{CO} is relatively unimportant in calculating the final results, and will be assumed to be constant at 0.007 ml of gas per minute, the number recommended by Ref. 1.

D_L varies somewhat with the size of the lungs, but will be treated here as a constant, 30. This is the value used by Ref. 1 in their sample calculations, and generally agrees with data for their test subjects given in Table 1 of Ref. 1.

$P_I(CO)$ is the primary input variable. In the cookstove community we usually work with CO concentrations in parts per million. The formula relating the CO in parts per million to $P_I(CO)$ is:

$$P_I(CO) = \frac{X_{CO}(\text{in ppm}) * P_B}{1,000,000} \quad (\text{Eq. 2})$$

It should be noted that relating CO in ppm is not a concentration, it is a molecular fraction, or mole fraction. The actual concentration will depend on air pressure. It is the concentration that is used here, and the above formula performs the conversion.

$P_c(O_2)$ the pulmonary capillary pressure of oxygen, is a function of barometric pressure, but varies in a predictable way. The relations used to calculate pulmonary capillary oxygen pressure from barometric pressure is to first calculate a partial pressure of oxygen in the inspired, or inhaled, air, given by the symbol $P_I(O_2)$. This is just the mole fraction of oxygen in air, about 0.195, times the barometric pressure. (The 0.195 will vary slightly, but can be assumed to be nearly constant.) From this number the pulmonary capillary pressure can be calculated by a regression-type formula, as given in Ref. 1.

$$P_c(O_2) = \frac{1}{0.072 - 0.00079P_I(O_2) + 2.515E - 6P_I(O_2)^2} \quad (\text{Eq. 3})$$

One must relate the quantity of CO in the body to the concentration in the blood, [COHb]. (The square brackets are the symbol for concentration.) This is done by dividing the quantity of CO in the body, in ml of gas, by the quantity of blood in the body, V_b , in ml of blood. The quantity of blood in the body is related in Ref. 1 as being proportional to body mass, with a slightly different proportionality constant for men and women.

$V_b = 74$ ml/kg of body weight for men, and

$V_b = 73$ ml/kg of body weight for women.

Since women (and children) are the main people affected by cookstoves, the value for women will be used through the rest of this report.

\dot{V}_A the alveolar ventilation rate, will vary somewhat from person to person, and will increase with increasing activity level. Ref. 1 says that, for their test subjects, the average among sedentary women was 9,000 ml/min, the average among sedentary men was 10,100, and the average among men who were mildly exercising was about 14,000. The mild exercise was pedaling a stationary bicycle at 50 cycles per minute against no resistance. The effect of alveolar ventilation will be explored in more detail, but a typical value for an active woman in the process of cooking might be about 11,000 ml/min.

The final variable to be dealt with is the most complex, the concentration of oxygen in the blood, [O₂Hb]. It is known that a gram of hemoglobin will absorb 1.38 ml of gas, but that CO and oxygen “compete” for absorption sites on the hemoglobin.

The concentration of oxygen in the blood is given by:

$$[O_2Hb] = [O_2Hb]_{\max} - [COHb] \quad (\text{Eq. 4})$$

where $[O_2Hb]_{\max}$ is in turn given by:

$$[O_2Hb]_{\max} = \frac{1.38[Hb]}{100} \quad (\text{Eq. 5})$$

where [Hb] is the concentration of hemoglobin in the blood, measured in grams per 100 ml of blood. Thus, the maximum amount of oxygen in blood is proportional to the amount of hemoglobin, and the amount of oxygen is equal to the maximum amount of oxygen, minus the amount that the CO displaces in the hemoglobin.

Equation 1 gives the change in the total quantity of CO in the blood, but this must be related to the concentration. The concentration of CO in the blood is given by the notation [COHb], though this quantity may have one of two different sets of units. It can have units of ml of gas per ml of blood, or units of % saturation. Percent saturation is the quantity usually used to correlation to various health conditions. The formulas used are:

$$[COHb] \left(\frac{\text{ml of gas}}{\text{ml of blood}} \right) = \frac{\text{Quantity of CO (ml of gas)}}{V_b} \quad (\text{Eq. 6})$$

and once the [COHb] is known in ml of gas per ml of blood, the % saturation is calculated from:

$$[COHb](\% \text{saturation}) = \frac{[COHb]}{[O_2Hb]_{\max}} \quad (\text{Eq. 7})$$

One sees that the % saturation is a strong function of the amount of hemoglobin in the blood. Ref. 1 gives the average [Hb] for women as about 14 g/100 ml, while the average for men is 15.8. It is believed that unless one's diet is completely lacking in iron or protein, that the amount of hemoglobin will not be a strong function of diet. Anemia will cause a severe deficiency of hemoglobin (Ref 4).

The Coburn equation is a non-linear differential equation that has no explicit solution, even under the simplest assumptions. However, a computer can easily be used to solve the equation. A spreadsheet program can be used, as was done in this study. The general procedure for solving the equation would be:

1. Assume the initial blood CO concentration in % saturation.
2. Calculate the initial blood CO concentration in ml of gas per ml of blood.
3. Calculate the total CO in the blood, in ml of gas.
4. Assume values for the other variables such as CO in the air, alveolar ventilation rate, hemoglobin concentration, etc.
5. Use Eq. 1 to calculate the rate of change of total CO in the blood during the first time step.
6. Multiply this rate of change by the length of the time step to get the change in total CO at the end of the first time step.
7. Add this to the total CO value at the beginning of the first time step (from step 3 of this process).
8. Recalculate the concentration of CO in ml of gas per ml of blood.
9. Divide the total CO by V_b then divide this value by $[O_2Hb]_{\max}$ to get the CO concentration in % saturation.
10. Go through steps 5-9 for each time step to get the [COHb] at the end of each time step.

11. Find the maximum value of COHb concentration in % saturation. Health effects will be correlated to this value based on the table below.

Table 1: Health effects produced by various levels of [COHb].

[COHb] (% Saturation)	Effects
0.4	Normal value for non-smokers
5	Typical value in heavy smokers
Below 10	No significant effects
10+	Headache and impaired manual coordination, changes in visual evoked response by electroencephalogram.
10-20	Heavy head
20-30	Headache, dizzy, weak
30-40	Pass out
40-50	Coma
50-60	In deadly peril
60+	Death

Most of Table 1 comes from Ref. 3. There is anecdotal evidence that different humans respond differently to various levels of CO. This is almost certainly true, but it is unknown how much variation is present within humans. Also anecdotally, some people reportedly develop a tolerance to CO. This tolerance may be psychological rather than physical, that is, people get accustomed to suffering.

Typical Results

Before looking at specific cooking situations, some general rules of thumb might be generated. If one assumes a 110 lb woman (50 kg) with an alveolar ventilation rate of 11,000 ml/min (active, but not working hard) with an initial [COHb] of 1%, and a barometric pressure of 750 mm Hg (slightly above sea level) some general results can be calculated. A CO level of 170 ppm will produce a blood CO level of 10% saturation in an hour. This is about the level where significant physiological effects begin to appear.

The peak blood CO concentration at 170 ppm is about 22% saturation. As a rough approximation, in the first hour the blood CO level will rise about 50% of the way to its peak value, and in the second hour it will rise most of the rest of the way to its peak value.

An approximate correlation for the peak CO at the end of a cooking process is:

$$Peak [COHb] \approx 10 \frac{AverageCO}{170} \frac{Time}{60} \frac{50}{m} \quad (Eq. 8)$$

where

m = body mass in kg

Average CO = average CO in ppm

Time = time duration of cooking task in minutes

Peak [COHb] = the peak CO concentration in the blood in % saturation.

This equation applies for CO levels below about 700 ppm, for time durations up to about 90 minutes, for both men and woman but probably not for children with their smaller lungs, and for healthy people who are active but not exercising hard.

Comparison to Standards

Various standards exist in the world for allowable exposures to CO. These standards give an allowable constant level of CO for a given time, the allowable level of CO decreasing as the time gets longer. The standards are summarized in the table below, which comes from Ref. 5.

Table 2: Allowable CO exposures around the world.

Time	Allowable CO level in ppm (The approximate peak blood CO in % saturation is in parentheses.)			
	World Health Organization	US Environmental Protection Agency	US Occupational Safety and Health Administration	Indian Factories Act
15 minutes	100 (1.96%)			400 (6.7%)
1 hour	30 (1.98%)	35 (2.25%)		
8 hours	10 (1.73%)	9 (1.57%)	50 (7.6%)	50 (7.6%)

As with the previous calculations, the calculated CO levels here also assume a 50 kg female who is moderately active. The assumed initial CO level was 0.4% saturation, which is the typical value for non-smokers who haven't been exposed to CO for some time. Data from this table supports the idea that blood saturations in the 7% range produce insignificant effects.

A Comparison of Stoves

It is informative to use the techniques presented here to calculate typical CO levels for some test conditions. Of course, the CO exposure (the amount of CO in the air) is a function of the CO output of the stove, the ventilation level of the room, the size of the room, the height of the ceiling, and other factors.

The 4 test stoves described here were all tested in the Aprovecho test kitchen under severely limited ventilation conditions. The 4 stoves were:

1. An open fire.
2. A Uganda 2-pot stove with chimney.
3. A rocket stove.
4. A charcoal stove from Mali.

Each stove was tested 3 times for a typical cooking task, that is, bringing 5 liters of water to a boil and simmering for 30 minutes. Of the 3 tests, a test was chosen with mid-levels of CO, that is, not the worst test nor the best. The exception to this was the Uganda stove, where the worst test was chosen. Since this stove had a chimney it was believed that CO levels would be so low that the stove would produce minimal blood CO even under the worst conditions.

The human body variables assumed in this analysis are a 50 kg woman who will have 3650 ml of blood; breathing with an alveolar ventilation rate of 11,000 ml/min, which is appropriate to modest activity; an altitude a little above sea level ($P_B=750$); an initial CO level of 1% saturation, which is appropriate for a non-smoker who retains a small amount of blood CO from a previous cooking event; and with 14 g of hemoglobin per 100 ml of blood, which is appropriate to a healthy adult female.

Figure 1 shows the room and blood CO levels for the open fire. We see that the peak room CO is about 720 ppm, and the average room CO level is about 500 ppm for about 60 minutes. The peak blood CO level is about 28 % saturation, which is in the level of headache, dizzy, weak from Table 1.

Figure 2 shows the results for a rocket stove. We see that the peak room CO is about 190 ppm, and that the average CO is about 120 ppm for 70 minutes. The peak blood CO is a little over 7 % saturation, which will produce no significant effects.

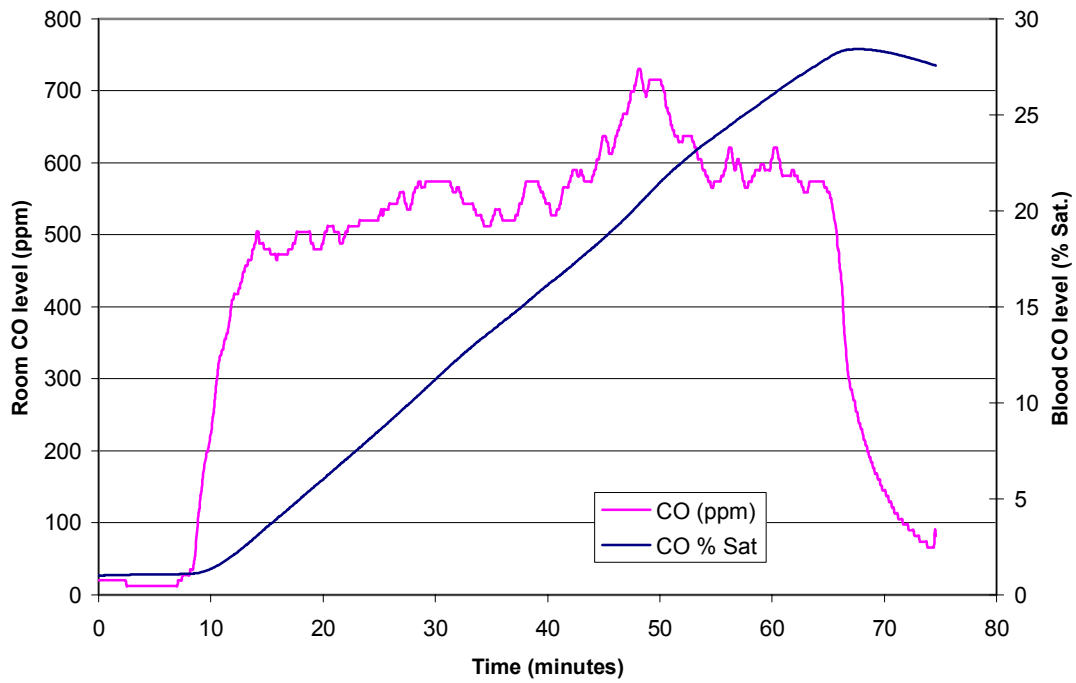


Figure 1: Results for the open fire.

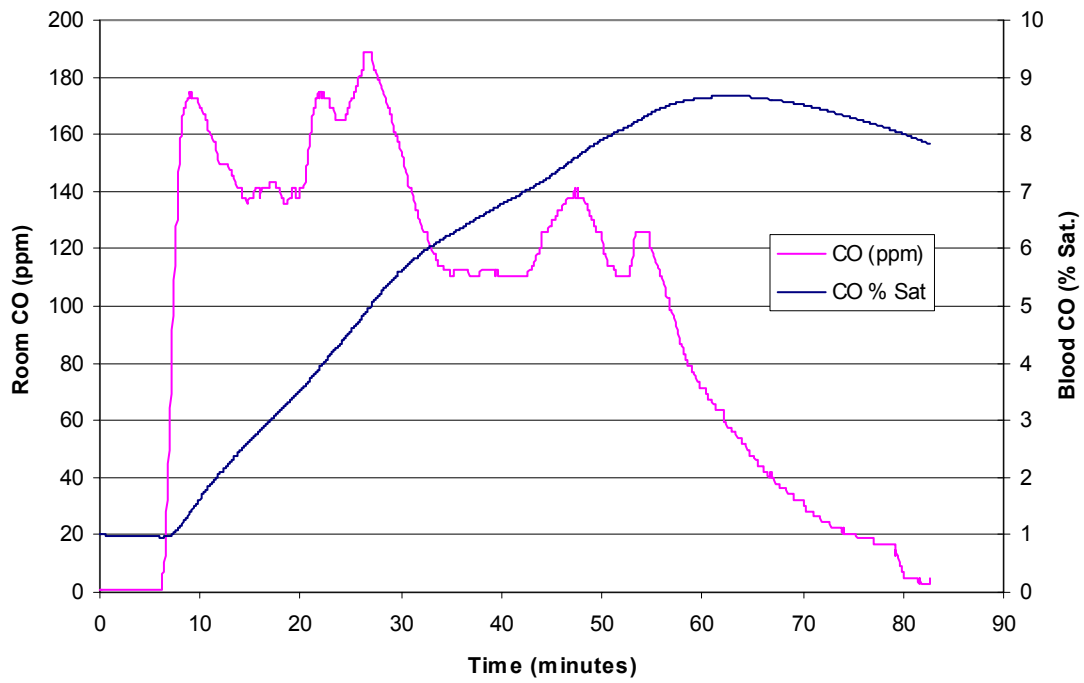


Figure 2: Results for a rocket stove.

Figure 3 shows the results for the Uganda stove with chimney. We see that the chimney is very effective at reducing the room CO level, with the peak room CO level being below 60 ppm and the average being about 30 over the course of 1 hour. This would meet CO standards in the developed world. We see that the blood CO level stays below 2%, which is well below the level seen in smokers who are not exposed to other external source of CO. In fact, until the stove starts, the blood CO level is decreasing from its assumed initial value of 1%, since 1% saturation is above the equilibrium level for a non-smoker.

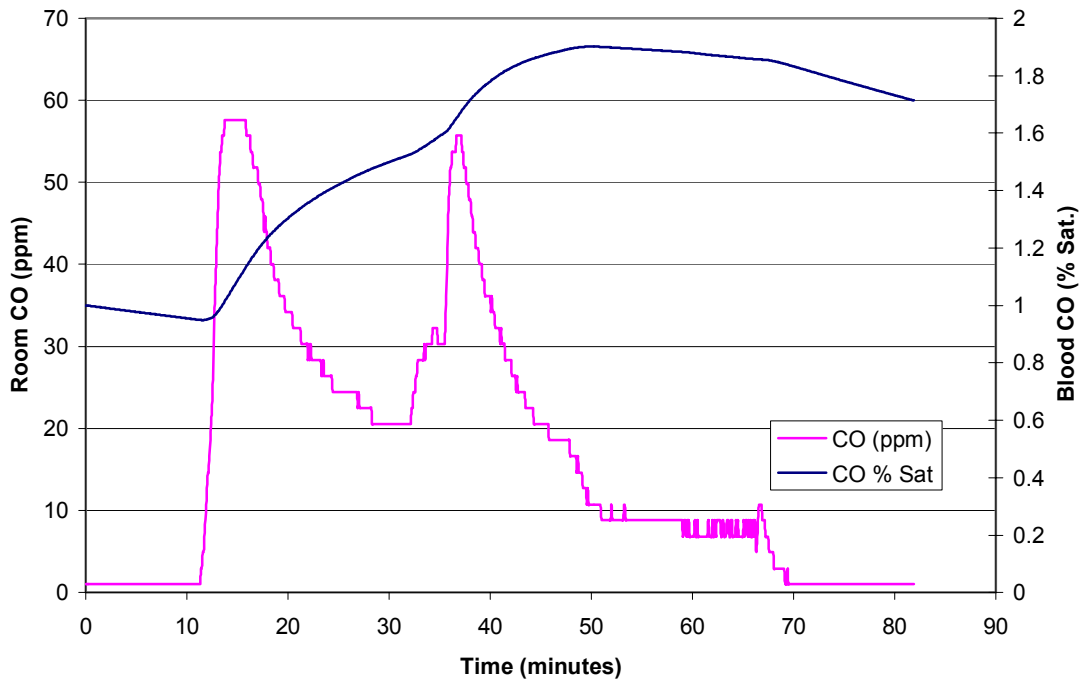


Figure 3: Results for the Uganda 2-pot stove with chimney.

Figure 4 shows the results for the Mali charcoal stove. As expected for a charcoal stove the room CO levels are very high, averaging about 1000 ppm for 70 minutes, with a peak of about 1350 ppm. The blood CO levels are also very high, about 47 % saturation. According to Table 1 this would put the user in a coma. Of course, such a stove would not normally be used in such a poorly ventilated area, or if it were, the cook would presumably not stay in the room continuously.

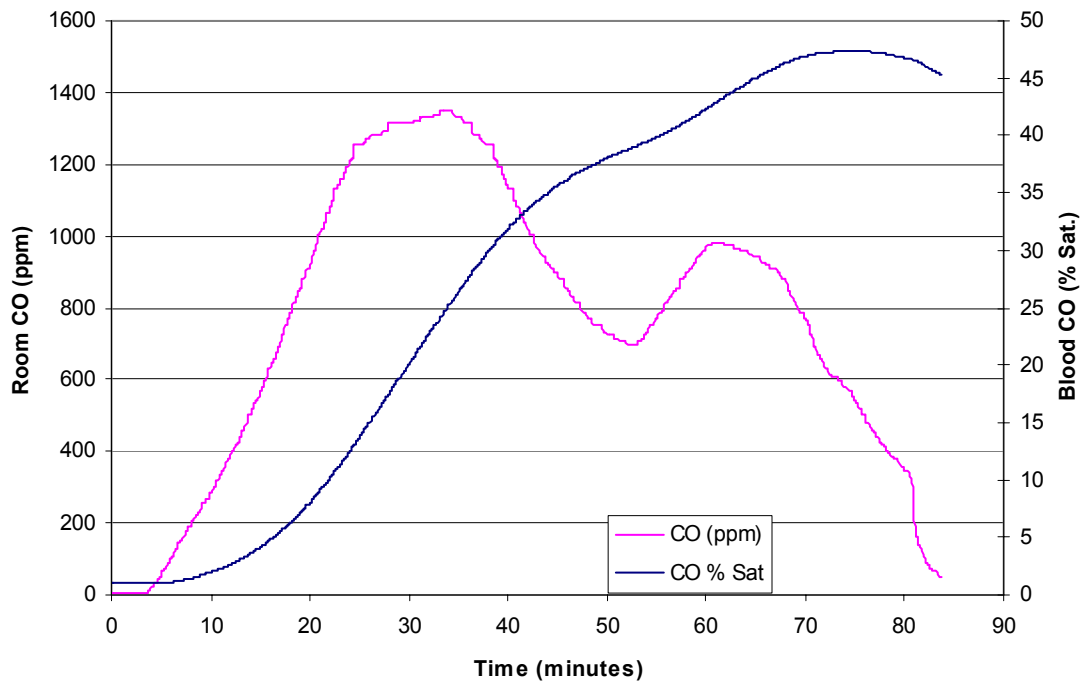


Figure 4: Results for the Mali charcoal stove.

Influence of Variables

The influence of some of the primary unknowns can be examined. Figure 5 shows the peak blood CO concentration for the rocket stove under the conditions listed above, except that the air pressure (barometric pressure) is varied. The highest barometric pressure in Fig. 5 is normal pressure at sea level. The lowest pressure is that would exist at about 13,000 feet (4000 m) the highest altitude at which significant numbers of humans live.

The figure shows that CO decreases at higher altitudes, which is caused by the fact that for a given mole fraction of CO, the actual pressure of the CO drops at higher altitude. The implicit assumption in this is that the stove will perform the same at all altitudes, which is probably not a good assumption. The model can take altitude into account quite easily, and altitude by itself doesn't have much of an effect on CO in the blood.

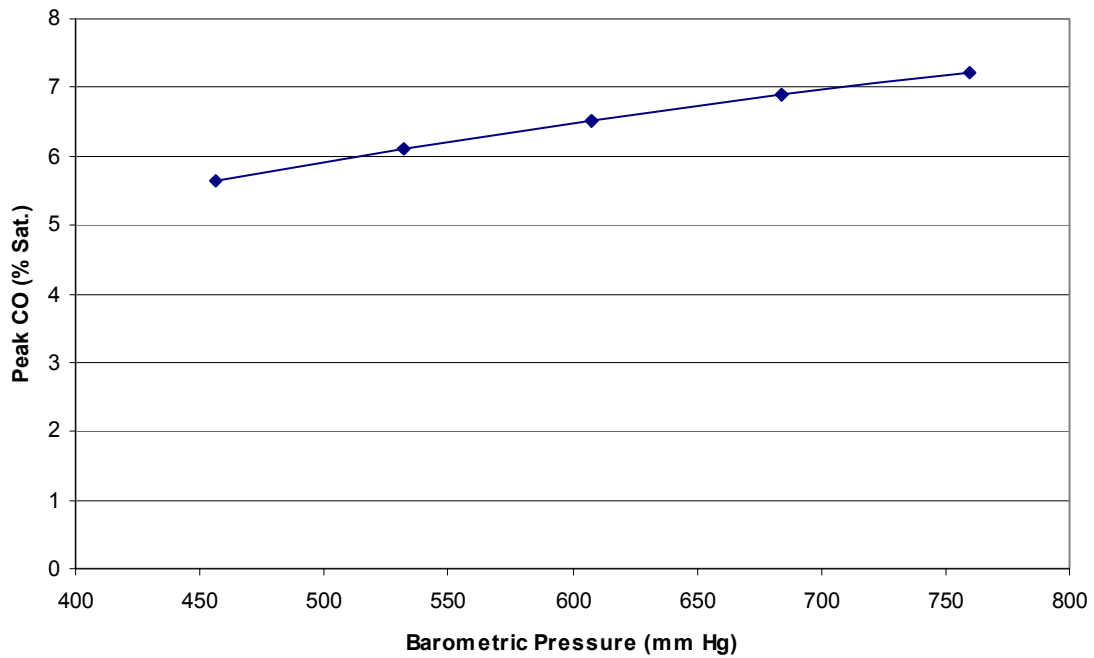


Figure 5: The effects of air pressure (barometric pressure on peak blood CO levels for a typical rocket stove.

Figure 6 shows the effects of blood Hb concentration on the peak blood CO level, again for the rocket stove under the same conditions as above. As previously mentioned, about 14 g per 100 ml is typical for a healthy adult female (Ref. 1). The peak CO level is a relatively weak function of the hemoglobin concentration until very low levels of hemoglobin are reached. This supports the idea that one does not need to know the exact composition of a person's blood to make an estimate of how CO will affect them. While Fig. 6 below does not show this factor, having a lower hemoglobin level is beneficial in one respect; it takes a smaller quantity of CO to achieve a high concentration in the blood, but once the air CO drops, the blood CO concentration drops more rapidly with a lower hemoglobin concentration.

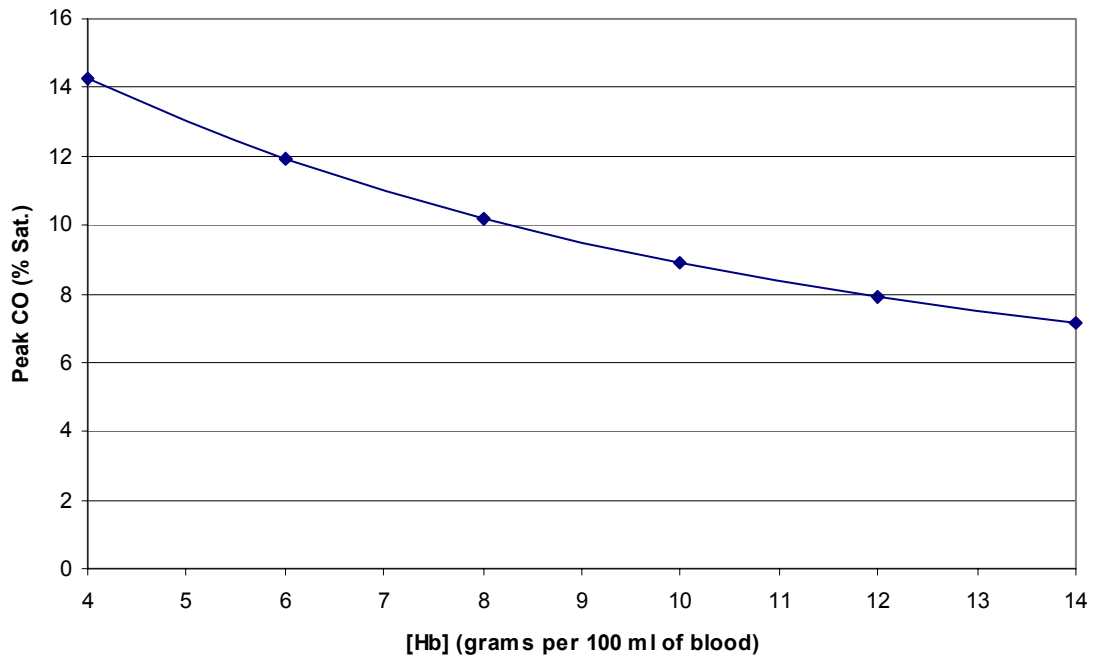


Figure 6: Effect of blood hemoglobin concentration on peak CO level for a rocket stove.

Figure 7 shows the effects of alveolar ventilation rate, which is based on breathing rate, which is based on activity level and probably altitude to some extent. As expected, the higher the breathing rate the faster the CO increases in the blood, and the higher will be the blood CO level during the cooking phase. The alveolar ventilation rate will have little influence on equilibrium or saturation CO level. However in the roughly 1 hour duration of a cooking event, the blood CO does not reach equilibrium.

Once the cooking event is finished and the room CO level decreases, a higher breathing rate will reduce the blood CO faster than a lower breathing level.

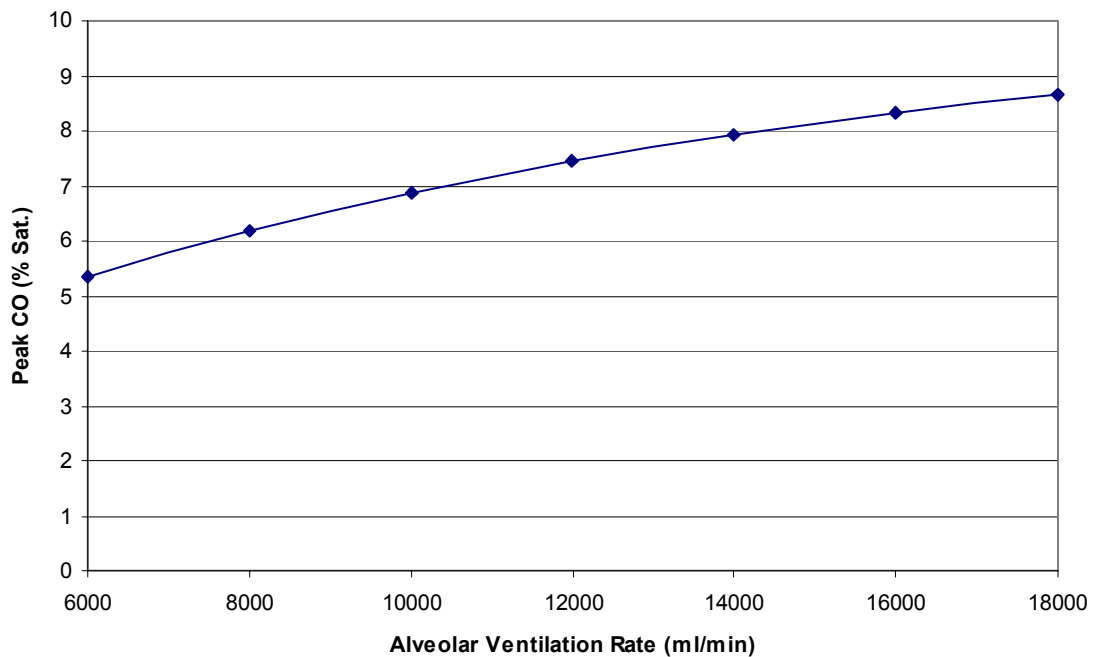


Figure 7: The effect of alveolar ventilation rate on blood peak CO level.

With regard to the effect of room ventilation, it is known that ventilating a room has a large effect on the pollutants within the room. To study this effect, a ventilation factor was applied to the base case, which was with the room fully closed. Since the ventilation was such a large factor, and since the rocket stove was on the border of being very clean even without ventilation, the worst stove, the Mali charcoal stove was used for this part of the study.

Figure 8 below shows the effect of ventilation factor on the peak blood CO level for the Mali charcoal stove. The ventilation factor is defined as the average level of CO under that ventilation condition divided by the average level of CO under the no ventilation condition. In performing the calculation it was assumed that the reduction in pollutant concentration was constant. Thus, a ventilation factor of 0.3 (corresponding to a hole in the roof) means that with the hole in the roof the concentration to CO at any time was assumed to be 30% of what it was at that same time with the room fully closed.

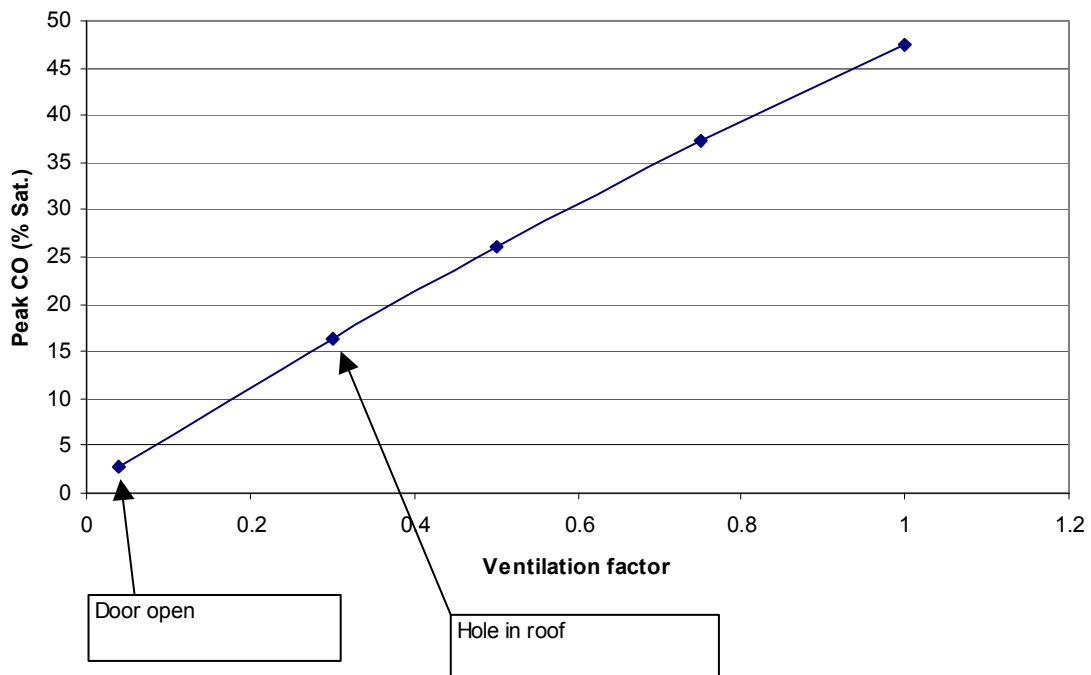


Figure 8: Effect of room ventilation on the peak blood CO level.

A ventilation factor of 0.04 corresponds to measured results with a door open, and a ventilation factor of 0.3 corresponds to a hole in the roof. A ventilation factor of 1 is for the fully closed condition. It is seen in Fig. 8 that ventilation makes a very large difference, and that with good ventilation, even a very polluting stove produces low CO levels in the blood.

Conclusions of the CO Study

- It is possible to theoretically model the uptake of CO into the body, given the CO levels in the air in the room.
- The results are a function of a number of body-related variables, hemoglobin concentration, alveolar ventilation rate, body mass, and initial blood CO concentration.
- The blood CO level can be related to health effects, at least approximately.
- The effects on the body of a number of variables in the stove design, and a number of variables in room ventilation may be studied, at least approximately. Altitude can also be taken into account in this model.

References for Part 1:

1. Predicting the carboxyhemoglobin levels resulting from carbon monoxide exposures, Jack E. Peterson and Richard D. Stewart, *Journal of Applied Physiology*, Vol. 39, No. 4, pp. 633-638, October, 1975.
2. Considerations of the physiology and variables that determine the blood carboxyhemoglobin concentration in man, R.F. Coburn, R.E. Forster, and P.B. Kane, *Journal of Clinical Investigations*, Vol. 44, pp. 1899-1910, 1965.
3. *Danger Signals to Human Health*, W.F. Sulilatu, Netherlands Organization for Applied Scientific Research, ca. 1983.
4. Private communication with Brock Andreatta, 3rd year medical student.
5. Private communication from Dean Still.

Part 2: Particulates

Unlike carbon monoxide, particulates generally do not have immediately recognizable health effects. Therefore, particulate exposure does not need to be monitored instantaneously, and average values can be used to assess the health effects. Much of the natural particulate matter (PM) in the air that we breathe, such as dust and pollen, is large enough that they are trapped in the nose and throat. But the *fine* particles (less than 10 microns) generated by combustion are so small that they make it past the nose, deep into the lungs. Over time, these particles collect in the lungs, causing damage. This can result in distress such as Acute Lower Respiratory Infection (ALRI, common in children), chronic obstructive pulmonary disease (COPD, scarring of the lung tissue common in young women), lung cancer (from coal burning), and cardiopulmonary mortality, among others.

A study conducted on over a half million people in 151 U.S. cities found that “death rates in the areas most polluted with fine particulates were 17% higher than in the least polluted areas, as a result of a 31% higher rate of death from heart and lung disease, even when most cities complied with the U.S. federal standards for particulate pollution” (World Bank, 1998). The Health Effects Institute Review of studies of ambient PM₁₀ in 90 cities in the year 2000, show a consistent one half percent increase in mortality for every change of 10 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) measured for 24 hours before the day of death (HEI, 2001). The higher the concentration of particles, the greater the effect on the health of the population.

Ambient levels of PM10 mg/m ³	Increase in human death	Hospitalization for heart disease	Hospitalization for Pneumonia and COPD
10	0.5	1%	2%
50	2.5%	5%	10%
65	4%	6.5%	13%
100	5%	10%	20%
150	7.5%	15%	30%

Based on Information from The Health Effects Institute, 2000, BI/Clean Air Revival, Inc. 2001

Particulate Standards

The following chart shows international average particulate exposure limit recommendations. Generally, 150 $\mu\text{g}/\text{m}^3$ over a 24-hour period is the short-term limit, while 50 $\mu\text{g}/\text{m}^3$ should not be exceeded over the long-term.

Table 1. Reference Standards and Guidelines for Average Ambient Particulate Concentration
(micrograms per cubic meter)

Standard or guideline	Long-term (annual)			Short-term (24 hours)		
	PM ₁₀	BS	TSP	PM ₁₀	BS	TSP
EU limit values		80 ^a	150 ^a		250 ^c	300 ^d
EU guide values		40–60 ^a			100–150 ^c	
USEPA primary and secondary standards	50 ^e			150 ^e		
WHO guidelines ^h		40–60	60–90		100–150	150–230
WHO guidelines for Europe ^a	50		70 ⁱ	125	120	

Notes: PM10, particulate matter less than 10 microns in aerodynamic diameter; BS, black smoke (converted to $\mu\text{g}/\text{m}^3$ measure); TSP, total suspended particulates.

a. Median of daily mean values.

b. Arithmetic mean of daily mean values.

c. 99th percentile of all daily mean values throughout the year.

d. 95th percentile of all daily mean values throughout the year.

e. Daily mean values.

f. Arithmetic mean.

g. Guideline values for combined exposure to sulfur dioxide and particulates.

h. Not to be exceeded for more than one day per year.

i. Guideline for thoracic particles. According to International Organization for Standardization standard ISO-TP, thoracic particle measurements are roughly equivalent to the sampling characteristics for particulate matter with a 50% cutoff point at 10 microns diameter. Values are to be regarded as tentative at this time, being based on a single study that also involved sulfur dioxide exposure.

Sources: European Community 1992 (EU); United States, CFR (USEPA); WHO 1979 (WHO guidelines); WHO 1987 (WHO guidelines for Europe).

(World Bank Group, 1998)

Particle Size and Measurement

The size of the particle plays a role in the effects on health. The total suspended particulates (TSP) in the air do not necessarily affect respiratory health. Generally, only

particles smaller than 10 microns are respirable. Those below 2.5 microns are the most detrimental to health. A brief study of particle size distribution was conducted at Aprovecho's summer stoves camp in August of 2005. Average concentrations of PM_{2.5} and PM₁₀ during a cooking task were measured simultaneously by co-locating two Airmetrics Minivols. Results of five different stoves burning wood showed that particles smaller than 2.5 microns accounted for 95% of total particles 10 microns and below, with a standard deviation of 3%. Thus, almost all the particles produced by wood combustion are those of a size most detrimental to health.

There are several methods available for measuring particulates, falling into two general categories: gravimetric and optical.

Gravimetric measurements are conducted by drawing air at a constant rate through a filter for a given period of time. The mass of the particles collected on the filter is then weighed and factored by the time and flow rate to determine the average concentration of particles in the air during the sampling period. Impactors or cyclones can be used at the inlet to the filter to separate out particles larger than a given size. Some available gravimetric systems include:

- The A.P. Buck personal monitor measures PM₄ and is small enough to be carried on a person in order to determine personal exposures, \$500
- Airmetrics Minivol measures PM_{2.5} and PM₁₀ at 5 Liters per minute, \$2,500

Optical measurements are conducted by passing smoke through a beam of light in a sensor chamber. The quantity of light reflected off the smoke particles is measured by a receiver which generates a voltage proportional to the light. More smoke reflects more light, resulting in a higher voltage output. Concentration data is recorded in real-time to a data-logger or computer, giving data about the average concentration as well as the peaks during the cooking process. Some optical PM measuring devices include:

- Radiance Nephelometer, a laboratory-standard instrument which requires a cool and dry smoke sample, \$7,000
- UCB Particulate monitor which uses a photoelectric smoke detector and data logger with software, \$500
- The Aprovecho Appropriate Technology Particulate meter is currently in development using a highly-sensitive smoke detector connected to a computer or data logger, \$250

Findings

In the poorly-ventilated test kitchen studies at Aprovecho, concentrations of Particles of 4 microns and smaller were measured with an A.P. Buck pump and filter system at breathing height (1.3 m high, 1.3 m aside from stove) within the kitchen. Different types of stoves run carefully in a laboratory setting were shown to emit the following pollution

levels during a cooking task (bringing 5 Liters of water to a boil then simmering for 30 minutes) in the poorly-ventilated test kitchen:

- From 15,000 – 30,000 $\mu\text{g}/\text{m}^3$ – *thick smoke, limited visibility in the kitchen* -- open fire and portable stoves
- About 10,000 $\mu\text{g}/\text{m}^3$ – *haze of smoke* -- rocket stove and charcoal stoves
- Under 5,000 $\mu\text{g}/\text{m}^3$ – *some smoke visible* -- wood burning stoves with electric fan
- Under 500 $\mu\text{g}/\text{m}^3$ – *smoke not visible* -- liquid gas fuels and stoves with chimneys

Note that these are average concentrations during the cooking task; peak values would be considerably higher. An open-air type kitchen would have lower concentrations, whereas a tightly closed adobe kitchen may have even higher concentrations. Additionally, these are results of carefully operated stoves, so actual in-field use will differ. Fortunately when the particulate concentration is uncomfortably high, a person may try to avoid the smoke by crouching lower to the floor or leaving the room.

The World Health Organization recommends a maximum 100-150 $\mu\text{g}/\text{m}^3$ 24 hour-mean particulate concentration exposure. The U.S. Public Standard for allowable PM_{10} is 150 $\mu\text{g}/\text{m}^3$ daily and 50 $\mu\text{g}/\text{m}^3$ annually. Since cooking for families is done every day, it is more appropriate to use the annual, long-term standard in this analysis. Based on this, it is possible to determine what stove and ventilation combinations may be safe to use in a real kitchen. For each of five stoves plus the open fire, the average particulate concentration during a cooking task was multiplied by three meals per day and averaged over a 24-hour period.

Ventilation

In order to quantify the effects of different levels of ventilation, a second study was conducted in the test kitchen at Aprovecho. A constant pollution source was used while the level of ventilation was varied. Carbon monoxide was measured with a HOBO datalogger and particulates 4 microns and below were measured with an A.P. Buck pump and filter gravimetric system (Still and MacCarty, 2005). Results showed that opening a 20 X 25 cm hole in the roof reduced both CO and PM levels by 70%, while simply opening a door alone reduced levels by 95%.

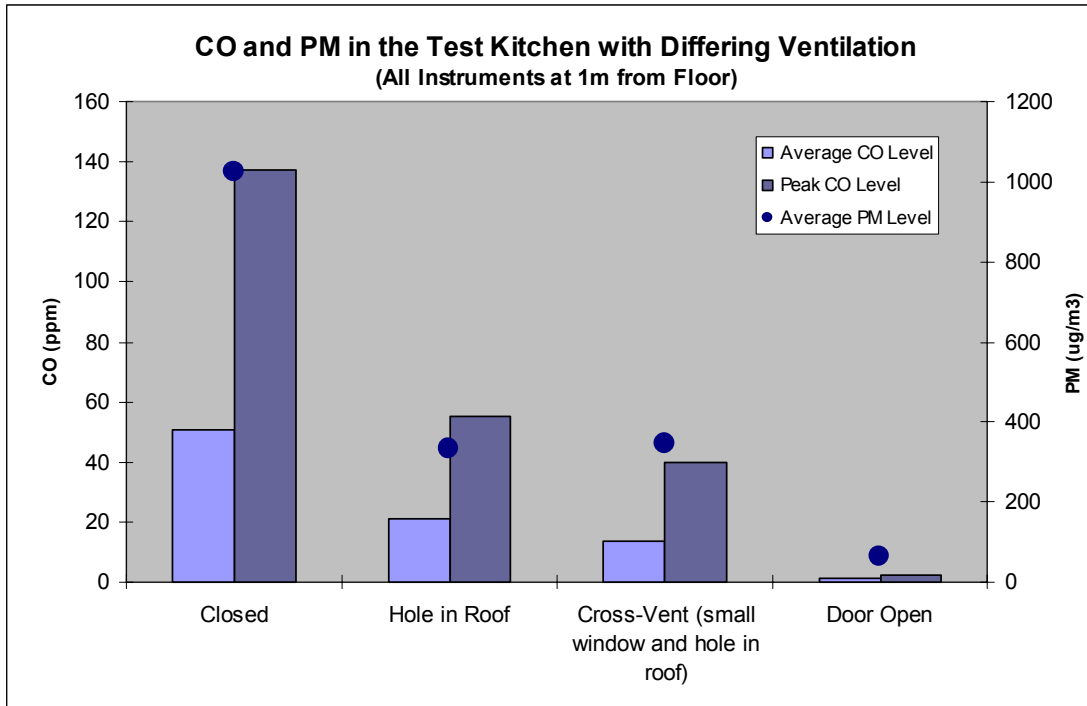


Figure 9: Effects of Ventilation in the Test Kitchen (Still and MacCarty, 2005)

These ventilation factors can be applied to the daily average particulate concentrations to determine if each stove can be run safely indoors. It is assumed that 3 meals per day are prepared similarly.

$$\text{Daily Average PM Level} = \frac{(1 - \text{Ventilation Factor}) * 3 * \text{average concentration} * \text{time duration}}{24 \text{ hours per day}}$$

(Eq. 9)

Results are as follows:

DAILY PARTICULATE LEVEL COMPARED TO RECOMMENDED LIMITS

Recommended Short-Term (24-hour) Limit	USEPA	150	WHO Europe	125	$\mu\text{g}/\text{m}^3$
Recommended Long-Term (Annual) Limit	USEPA	50	WHO Europe	50	$\mu\text{g}/\text{m}^3$

	Open Fire	Mali Charcoal	WFP Rocket Stove	Electric Fan/Wood Stove	Two-Pot Chimney Stove	Propane
Time to Cook 5 Liters min.	66	73	46	55	50	77
Avg Concentration During Cooking $\mu\text{g}/\text{m}^3$	14,972	8,437	8,974	2,152	479	51
Closed Kitchen -- 0% Ventilation						
Average Concentration in 24hr $\mu\text{g}/\text{m}^3$	2,059	1,274	865	247	50	8
Hole in Roof -- 70% Ventilation						
Average Concentration in 24hr $\mu\text{g}/\text{m}^3$	618	382	260	74	15	2
Door Open -- 95% Ventilation						
Average Concentration in 24hr $\mu\text{g}/\text{m}^3$	103	64	43	12	2	0

*Does not include ambient background neighborhood pollution
 Closed test kitchen has 3 air exchanges per hour

The dark boxes denote particulate levels above all maximum exposure health standards. The medium boxes are concentrations between daily and annual limits, and the light boxes show the stoves and ventilation combinations that would meet both WHO and U.S. Public annual standards.

From this information, it can be seen that:

- Carefully operated stoves with functional chimneys and propane stoves were safe to use inside a test kitchen, even without added ventilation.
- The carefully operated wood burning stove with an electric fan was clean enough to use inside the closed test kitchen (approximately three air exchanges per hour.)
- The carefully operated rocket stove required significantly increased amounts of ventilation to reduce CO and PM levels to reasonable levels.
- The carefully operated open fire and charcoal stove emitted too much pollution even when the door or hole in the roof were opened to be safely used indoors.

Conclusions of the Particulate Study

This study is not intended to predict in-field measurements of actual concentrations in kitchens around the world. It does present a general relationship between stove choice, ventilation level, and exposure to harmful pollution. Ventilation and stove choice are both found to affect theoretical levels of exposure in a test kitchen.

In this study when cooking occurred in the unventilated kitchen only gas-burning stoves or carefully operated stoves with chimneys would protect inhabitants from harmful levels of Indoor Air Pollution. Increasing ventilation was shown to be very helpful when using any type of biomass cooking stove. Cooking with a carefully operated electric fan stove or rocket stove also diminishes the measured levels of CO and PM in these studies.

References for Part 2:

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