



Prediction of the Effects of Combustion-Generated Pollutant on Human Health: Mathematical Models and Numerical Solutions

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ABSTRACT

The rapid increase in technological innovations and utilizations have adversely affected the environment and consequently continued to constitute a threat to the future survival of human. To counter these assaults and the threats of further degradation of the environment and human health, the basic recommended approach for predicting the impact of the pollution and for the determination of the risk assessment strategies is through the use of mathematical models. Therefore, this work presents mathematical models for the prediction of the effects of combustion generated pollutants, such as Carbon-monoxide (CO) on human health. The developed coupled system of nonlinear partial differential equation for the ambient concentration of carbon mono-oxide in which the human subject was exposed to and the concentration of Carboxyhemoglobin (COHb) in the blood is solved numerically using Alternating-Direct Implicit (ADI) scheme. From the computations, the variables of the models show significant results in their variations and the standard error of the predicted results from the model range in between 0.5-0.85 for the different concentrations of ambient carbon monoxide. This established that the computed results show good agreement with available experimental data. Therefore, the model can be used as a means of controlling the effects of the pollutant on human health and the results will serve as a way of evaluating our technological injuries, effectively controlling our pollutants emissions and also as a tool for designing and developing better equipments and engines with lower carbon or pollutants emissions.

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INTRODUCTION

The increasing vehicular traffic and rapid industrialization of the society, environmental contamination of the air, water, soil, and food steadily became a threat to the very survival of the human race. Understandably, many signified industrial and governmental communities have recently committed large resources of money and human power to the problem of environmental pollution and pollution abatement by effective control measures focusing on achieving air quality standard in accordance with the Clean Air Act of 1967 [1]. However, resolving the problems, one must predict the ambient air concentrations that will result from any planned set of emissions. According to El-Harbawi et al. [2], mathematical models are extremely useful tools to predict the impacts of chemical process accidents.

Developing such models will drastically reduce the cost and the task associated with experimentations, as an alternative way of determining the effects of the pollutants.

Following the increase in vehicular traffic and rapid industrialization, CO is considered as a common atmospheric pollutant that directly affects human health [3]. The adverse effects of this pollutant have aroused the interest of many researchers to study the emission and the degree of the effects of the pollutant on human health. In earlier works researchers pointed out the effects of relatively small CO exposures, which are normally found in urban, industrial and household air while in the recent times, Gallagher and Mason [4] presented a study on carbon monoxide poisoning in two workers using an LPG forklift truck within a cold store. As part of their findings, the clinical assessment with mathematical exposure modelling may lead to successful retrospective diagnosis

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of CO poisoning and identify putative work activities. Moreover, the CO poisoning should be suspected whenever internal combustion engines are used within buildings and workers complain of relevant symptoms. From the biological investigations, correlation was found to exist between environmental pollution and Hepatitis, hence conclusions were reached that several disease processes involve factors of environmental pollution, such as, the direct effects of pollution on lungs involving lung diseases and indirect effects on all parts of the body. The adverse effect of air pollution on health was further corroborated by Ren and Tong [5]. Providing solutions or finding a way to abate the problems if not totally eliminated, has been the ultimate purpose of most concerning researchers. One way of establishing a solution is the fact that CO inhibits the blood's capacity to carry oxygen. Since, hemoglobin has as much as 200 times affinity for CO as readily as oxygen, when inhaled, it binds reversibly with blood hemoglobin to form carboxyhemoglobin, impairing the oxygen-transport capacity of the blood, as well as the oxygen's release to body tissues. Therefore, the percentage of COHb in the blood has been considered as a good index of health effects of CO on humans. As pointed out by El-Harbawi et al. [1], the development of mathematical models for the computation of percentage COHb in due blood has been used to predict the impacts of the pollutant on human health. Therefore, in the earlier work, Forbes et al. [6] proposed the formulae to compute COHb level in the blood as a function of exposure time by measuring the rate of CO uptake by humans under a wide range of conditions. Also, Forster et al. [7] derived an equation containing 14 parameters to predict the COHb level in the blood while researchers later developed a model for the relationship between blood COHb, rate of CO production and the respiratory CO exchange. In the model, a constant concentration of O₂Hb in the blood was assumed based on the linearized nature of the developed equation. However, Peterson and Stewart [8] pointed out that the constant value of O₂Hb in the Coburn *et al's* equation (CFK equation) led to a significant error in the computation of blood COHb. Therefore, they proposed an iterative procedure for accounting the variation of O₂Hb in the CFK equation. The nonlinear CFK equation was solved numerically by Bernard and Duker [9] equation using the fourth order Runge Kutta method while in the same year, Tyuma et al. [10] obtained an analytical solution of the CFK equation while assuming that hemoglobin is always saturated with O₂ or CO or both. Also, Collier and Goldsmith [11] adopted the method proposed by Roughton and Darling [12] to solve the CFK equation by taking into consideration the reduced haemoglobin. Although the CFK equation was developed for the prediction of the concentration of CO in the body, it has been widely used to predict blood COHb under different CO exposures [13], [14], [15],

[16]. More than three decades ago, Ott and Mage [17] and Venkatram and Louch [18] proposed linear models for the computation of the percentage COHb in the blood.

Many of the above reviewed developed models are strongly based on empirical laws and as such, do not appear to be derived from the basic physical principles. In developing the mathematical models that are based on physical principles, a great deal of complexities arises both in the solution and the involving parameters consequently, simplifying assumptions or idealization are made in order to obviate the complexities or difficulties. Singh et al., [3] developed mathematical models (nonlinear partial differential equation) for the computation of COHb in human blood as a function of exposure. However, the assumptions that pulmonary capillary as a two-dimensional channel calls for relaxation for more accurate solution. It should therefore, be noted that, in all the conceptual and developmental ways to effectively solve pollution problems, no proper account has been given to modeling CO uptake by the blood by taking account, the molecular diffusion, convection and facilitated diffusion with a good idealization of pulmonary capillary as a vessel (cylindrical coordinates). Therefore this forms the basis of this paper. The objective of this work is to develop mathematical models and provide numerical solution in order to predict the effects of the commonest combustion-generated pollutant, Carbon-monoxide, which has direct effects on human on Human health. This will serve as a way of evaluating our technological injuries, effectively controlling our pollutants emissions and also as a tool for designing and developing better equipment and engines with lower emissions.

Theoretical background

The effects of various pollutants on human health are shown in Fig. 1 below. In the pool of these health-damaging pollutants, an air pollutant called Carbonmonoxide is considered as a common pollutant that directly affects human health. In fact, is the one pollutant which produces change in human physiology that can be directly related to the concentration to which the subject was exposed. Considering the source of this pollutant, it is actually a product of incomplete combustion of hydrocarbon-based fuel, which becomes toxic when inhaled by man due to its strong affinity for hemoglobin (Hb), the oxygen carrier of the blood. Since it has 200-250 times affinity for hemoglobin and when inhaled, it binds reversibly with blood hemoglobin to form carboxyhemoglobin, impairing the oxygen-transport capacity of the blood, as well as the oxygen's release to body tissues, causing loss of consciousness, fatal asphyxiation, brain damage and all other kinds of health effects as shown in Table 1. Death occurs in human exposed to concentration of around 1000 ppm, corresponding to blood levels of 60% COHb. Reasonably

correlations have been found between daily mortality levels and CO. In addition, heart function has been shown to be altered by elevated COHb, as evidenced by the electrocardiograms of exposed healthy adults.

TABLE 1. Effect of carboxyhemoglobin in human blood [19]

Percentage of carboxyhemoglobin (COHb) in the blood	Effect
0.3-0.7	Physiologic norm for nonsmokers
2.5-3.0	Cardiac function decrements in impaired individuals, blood flow alterations; and, after extended exposure, changes in red blood cell concentration.
4.0-6.0	Visual impairments, vigilance decrements, reduced maximal work capacity
3.0-8.0	Routine values in smokers. Smokers develop more red blood cells than nonsmokers to compensate for this, as do people who live at high elevations, to compensate for the lower atmospheric pressure.
10.0-20.0	Slight headache, lassitude, breathlessness from exertion, dilation of blood cells in the skin, abnormal vision, potential damage to fetuses.
20.0-30.0	Severe headaches, abnormal manual dexterity.
30.0-40.0	Weak muscles, nausea, vomiting, dimness of vision, severe headaches, irritability, and impaired judgment.
50.0-60.0	Fainting, convulsions, coma
60.0-70.0	Coma, depressed cardiac activity and respiration, sometimes fatal
>70.0	Fatal

Development of the governing equations

As reported by Coburn and Forman [20], the blood COHb in the body is determined by the exchange of CO between the pulmonary capillary blood and the ambient air, endogenous production of CO, dilution of CO in the body tissue and metabolic consumption of CO, while the ambient air is transported to alveoli by ventilation. The blood then absorbs the CO from alveoli when it passes through the pulmonary capillaries. The

mechanisms involved in the transport of CO in the pulmonary capillary are the molecular diffusion, convection and the facilitated diffusion of CO, due to the presence of hemoglobin as carrier [3].

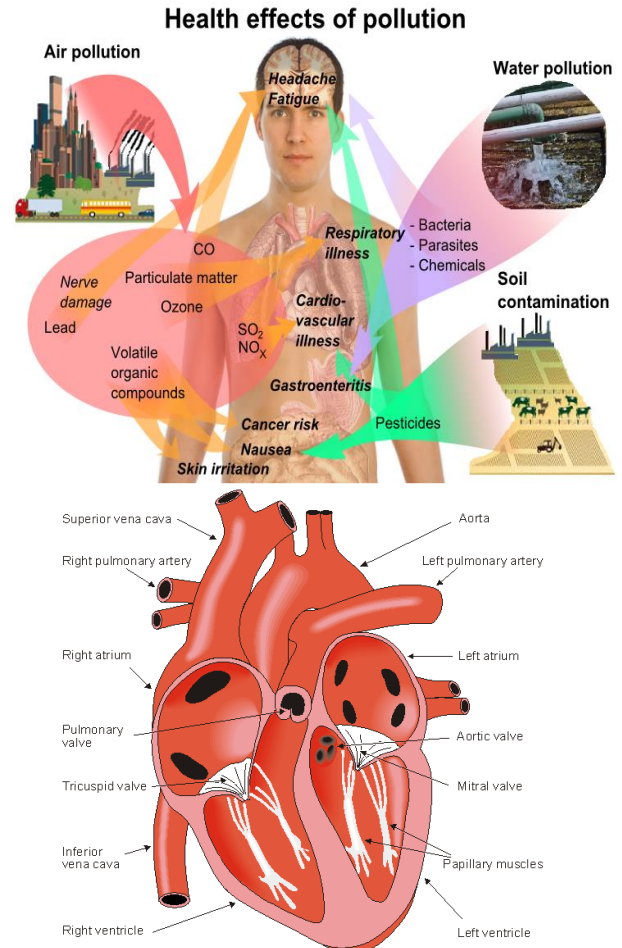


Figure 1. Overview of main health effects on humans from some common types of pollution

Model assumptions

The model is based on the following assumptions:

- The diffusion coefficient of hemoglobin and COHb are assumed to be the same. This is because the molecular mass of CO is much smaller compared to that of hemoglobin.
- The flow of blood through the pulmonary capillary is with a constant speed V in the axial direction.
- The diffusion of the species in the axial direction is neglected as this is justified by the scale analysis.
- The blood is considered to be fully saturated with O_2 in the pulmonary capillary because it is almost fully saturated during most of the time. The reaction for the replacement of O_2 from

- O₂Hb by CO is predominant reaction of CO with hemoglobin;
- v. The concentration of the reduced hemoglobin in the blood is negligible and the sum of the concentration of O₂Hb and COHb will be equal to the total hemoglobin content in the blood

Model formulation

In developing the mathematical model, we considered the pulmonary capillary as a two-dimensional cylinder, of Diameter ‘2R’ surrounded by the alveolar air as shown in Fig. 2.

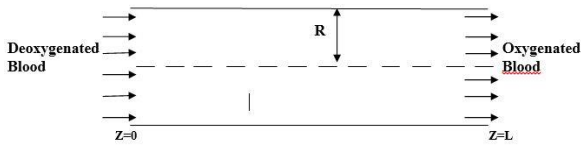
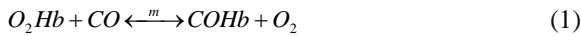


Figure 2: A two-dimensional cylindrical representation of pulmonary capillary.

The venous/deoxygenated blood enters the pulmonary capillary at $z = 0$ and leaves it as oxygenated blood at $z = L$. The diffused carbon monoxide into the blood from the ambient air combines with the hemoglobin inside the red blood cells to form COHb due to the higher affinity of hemoglobin for CO over O₂ at a ratio of 250 to 1 [21]. The net rate (S) at which CO combine in the red cells is mainly due to the replacement reaction and it is given by Gibson and Roughton [22].



The transient state mass balance for the species CO, COHb and O₂Hb leads to the following set of partial differential equations.

$$\left(\frac{\partial c_{CO}}{\partial t}\right) + v\left(\frac{\partial c_{CO}}{\partial z}\right) = D_{CO} \left[\frac{\partial^2 c_{CO}}{\partial r^2} + \frac{1}{r} \frac{\partial c_{CO}}{\partial r} \right] - S \quad (2a)$$

$$\left(\frac{\partial c_{COHb}}{\partial t}\right) + v\left(\frac{\partial c_{COHb}}{\partial z}\right) = D_{COHb} \left[\frac{\partial^2 c_{COHb}}{\partial r^2} + \frac{1}{r} \frac{\partial c_{COHb}}{\partial r} \right] + S \quad (2b)$$

$$c_{COHb} + c_{O_2Hb} = c_H \quad (2c)$$

From the chemical kinetics of carbon monoxide with hemoglobin, Gibon and Roughton [22] developed an expression for the net rate of combination of CO with O₂Hb as

$$S = m \frac{c_{CO}c_{O_2Hb}}{c_{O_2}} - mc_{COHb} \quad (2d)$$

From equation 2c,

$$c_{COHb} = c_H - c_{O_2Hb} \quad (2e)$$

Substituting eq.(2e) in eq. (2d), we have;

$$S = m \frac{c_{CO}(c_H - c_{O_2Hb})}{c_{O_2}} - mc_{COHb} \quad (3)$$

Substituting equation (3) into equations 2a and 2b, we arrived at;

$$\left(\frac{\partial c_{CO}}{\partial t}\right) + v\left(\frac{\partial c_{CO}}{\partial z}\right) = D_{CO} \left[\frac{\partial^2 c_{CO}}{\partial r^2} + \frac{1}{r} \frac{\partial c_{CO}}{\partial r} \right] - m' \frac{c_{CO}(c_H - c_{O_2Hb})}{c_{O_2}} + mc_{COHb} \quad (4a)$$

$$\left(\frac{\partial c_{COHb}}{\partial t}\right) + v\left(\frac{\partial c_{COHb}}{\partial z}\right) = D_{COHb} \left[\frac{\partial^2 c_{COHb}}{\partial r^2} + \frac{1}{r} \frac{\partial c_{COHb}}{\partial r} \right] + m' \frac{c_{CO}(c_H - c_{O_2Hb})}{c_{O_2}} - mc_{COHb} \quad (4b)$$

Equations 4a and 4b are subject to the following initial and boundary conditions.

$$t = 0, \begin{cases} c_{CO} = c_{CO}^o & 0 \leq r \leq R, \quad 0 \leq z \leq L \\ c_{COHb} = c_{COHb}^o \end{cases} \quad (5a)$$

$$r = 0, \frac{\partial c_{CO}}{\partial r} = 0, \tau > 0 \quad 0 \leq z \leq L \quad (5b)$$

$$z = 0, c_{CO} = c_{CO_{in}}, \tau > 0 \quad 0 \leq r \leq R \quad (5c)$$

$$r = R, \begin{cases} \frac{\partial c_{COHb}}{\partial r} = 0 & \tau > 0 \quad 0 \leq z \leq L \\ c_{CO} = \alpha c_{O_2} \end{cases} \quad (5d)$$

Using the following dimensionless parameters in equations 4a-5d

$$\beta_{CO} = \frac{m' c_H T}{c_{O_2}}, \quad \lambda_{CO} = \frac{m' \alpha CO_{pc} T}{c_{O_2}}$$

$$\beta_{COHb} = \frac{m c_H T}{\alpha CO_{pc}}, \quad \lambda_{COHb} = mT,$$

$$C_{CO} = \frac{c_{CO}}{c_H}, \quad C_{COHb} = \frac{c_{COHb}}{c_H}$$

$$C_{O_2Hb} = \frac{c_{O_2Hb}}{c_H}, \quad D'_{CO} = \frac{D_{CO} T}{(R')^2},$$

$$D'_{COHb} = \frac{D_{COHb} T}{(R')^2}, \quad V = \frac{vT}{L},$$

$$R = \frac{r}{L}, \quad Z = \frac{z}{L}, \quad \tau = \frac{t}{T} \quad (6)$$

We arrived at;

$$\left(\frac{\partial C_{CO}}{\partial \tau}\right) + V\left(\frac{\partial C_{CO}}{\partial Z}\right) = D'_{CO} \left[\frac{\partial^2 C_{CO}}{\partial R^2} + \frac{1}{R} \frac{\partial C_{CO}}{\partial R} \right] - \beta_{CO} C_{CO} (1 - C_{COHb}) + \beta_{COHb} C_{COHb} \quad (7a)$$

$$\left(\frac{\partial C_{COHb}}{\partial \tau}\right) + V\left(\frac{\partial C_{COHb}}{\partial Z}\right) = D'_{COHb} \left[\frac{\partial^2 C_{COHb}}{\partial R^2} + \frac{1}{R} \frac{\partial C_{COHb}}{\partial R} \right] + \lambda_{COHb} C_{CO} (1 - C_{COHb}) - \lambda_{COHb} C_{COHb} \quad (7b)$$

With the initial and boundary conditions

$$\tau = 0, \begin{cases} C_{CO} = C_{CO}^o \\ C_{COHb} = C_{COHb}^o \end{cases} \quad (8a)$$

$$0 \leq R \leq 1, \quad 0 \leq Z \leq 1$$

$$R = 0, \frac{\partial C_{CO}}{\partial R} = 0, \tau > 0$$

$$0 \leq Z \leq 1 \quad (8b)$$

$$Z = 0, C_{CO} = C_{lven}, \tau > 0$$

$$0 \leq R \leq 1 \quad (8c)$$

$$R = 1, \begin{cases} \frac{\partial C_{COHb}}{\partial R} = 0 \\ C_{CO} = \alpha CO_{labv} \end{cases} \quad (8d)$$

$$\tau > 0 \quad 0 \leq Z \leq 1$$

$$Z = 1, C_{CO} = C_{lven}, \tau > 0$$

$$0 \leq R \leq 1 \quad (8e)$$

The numerical scheme procedures

In developing a numerical scheme for the models developed above, application of simple explicit scheme

leads to reduction Fourier number from 0.5 to 0.25. This consequently shows reduction in the stability of the scheme. The application of Crank-Nicolson scheme which is unconditionally stable when applied to two-dimensional problems with periodic boundary conditions is limited in this problem since the resulting system of linear algebraic equations is no longer tridiagonal because of the five unknowns $C_{i,j}^{n+1}, C_{i+1,j}^{n+1}, C_{i-1,j}^{n+1}, C_{i,j-1}^{n+1}$, and $C_{i,j+1}^{n+1}$. This problem is not only limited to Crank-Nicolson, the same is true for all the implicit schemes. In order to overcome these difficulties, we apply Alternating-Direct Implicit scheme which was previous developed [21, 22] and unconditionally stable for all values Fourier number in any two-dimensional problem. The advantage of the ADI method is that the equations that have to be solved in each step have a simpler structure and can be solved efficiently with the tridiagonal matrix algorithm (TDMA). Also, this implicit scheme ensures a more efficient formulation and calculation than other implicit methods for multidimensional problems.

The procedures for the application of this scheme in this work are shown as follows.

First step, n $n + 1/2$ for equation (r – direction)

$$\frac{[C_{CO}]_{i,j}^{n+1/2} - [C_{CO}]_{i,j}^n}{(\Delta\tau/2)} + V \left(\frac{[C_{CO}]_{i,j+1}^n - [C_{CO}]_{i,j}^n}{\Delta Z} \right)$$

$$= D_{CO} \left\{ \begin{aligned} &\theta \frac{[C_{CO}]_{i+1,j}^{n+1/2} - 2[C_{CO}]_{i,j}^{n+1/2} + [C_{CO}]_{i,j-1}^{n+1/2}}{(\Delta R)^2} + \theta \frac{[C_{CO}]_{i+1,j}^{n+1/2} - [C_{CO}]_{i,j}^{n+1/2}}{R_i \Delta R} \\ &+ (1-\theta) \frac{[C_{CO}]_{i+1,j}^n - 2[C_{CO}]_{i,j}^n + [C_{CO}]_{i-1,j}^n}{(\Delta R)^2} + (1-\theta) \frac{[C_{CO}]_{i+1,j}^n - [C_{CO}]_{i,j}^n}{R_i \Delta R} \end{aligned} \right\} \quad (9a)$$

$$- \beta_{CO} \theta [C_{CO}]_{i,j}^{n+1/2} \{1 - [C_{COHb}]_{i,j}^n\} - (1-\theta) \beta_{CO} [C_{CO}]_{i,j}^n \{1 - [C_{COHb}]_{i,j}^n\} + \beta_{COHb} [C_{COHb}]_{i,j}^n$$

Second step, n + 1/2 $n + 1$ for equation (z – direction)

$$\frac{[C_{CO}]_{i,j}^{n+1} - [C_{CO}]_{i,j}^{n+1/2}}{(\Delta\tau/2)} + V \left(\theta \frac{[C_{CO}]_{i,j+1}^{n+1} - [C_{CO}]_{i,j}^{n+1}}{\Delta Z} + (1-\theta) \frac{[C_{CO}]_{i,j+1}^{n+1/2} - [C_{CO}]_{i,j}^{n+1/2}}{\Delta Z} \right)$$

$$= D_{CO} \left\{ \begin{aligned} &\frac{[C_{CO}]_{i+1,j}^{n+1/2} - 2[C_{CO}]_{i,j}^{n+1/2} + [C_{CO}]_{i,j-1}^{n+1/2}}{(\Delta R)^2} + \frac{[C_{CO}]_{i+1,j}^{n+1/2} - [C_{CO}]_{i,j}^{n+1/2}}{R_i \Delta R} \end{aligned} \right\} \quad (9b)$$

$$- \beta_{CO} \theta [C_{CO}]_{i,j}^{n+1} \{1 - [C_{COHb}]_{i,j}^{n+1/2}\} - (1-\theta) \beta_{CO} [C_{CO}]_{i,j}^{n+1/2} \{1 - [C_{COHb}]_{i,j}^{n+1/2}\} + \beta_{COHb} [C_{COHb}]_{i,j}^{n+1/2}$$

First step, n $n + 1/2$ for equation (r – direction)

$$\begin{aligned} & \frac{[C_{COHb}]_{i,j}^{n+1/2} - [C_{COHb}]_{i,j}^n}{(\Delta\tau/2)} + V \left(\frac{[C_{COHb}]_{i,j+1}^n - [C_{COHb}]_{i,j}^n}{\Delta Z} \right) \\ &= D_{COHb} \left\{ \theta \frac{[C_{COHb}]_{i+1,j}^{n+1/2} - 2[C_{COHb}]_{i,j}^{n+1/2} + [C_{COHb}]_{i,j-1}^{n+1/2}}{(\Delta R)^2} + \theta \frac{[C_{COHb}]_{i+1,j}^{n+1/2} - [C_{COHb}]_{i,j}^{n+1/2}}{R_i \Delta R} \right. \\ & \quad \left. + (1-\theta) \frac{[C_{COHb}]_{i+1,j}^n - 2[C_{COHb}]_{i,j}^n + [C_{COHb}]_{i,j-1}^n}{(\Delta R)^2} + (1-\theta) \frac{[C_{COHb}]_{i+1,j}^n - [C_{COHb}]_{i,j}^n}{R_i \Delta R} \right\} \quad (9c) \\ & + \lambda_{CO} \theta [C_{CO}]_{i,j}^{n+1/2} \{1 - [C_{COHb}]_{i,j}^n\} - (1-\theta) \lambda_{CO} [C_{CO}]_{i,j}^n \{1 - [C_{COHb}]_{i,j}^n\} - \lambda_{COHb} [C_{COHb}]_{i,j}^n \end{aligned}$$

Second step, $n + 1/2$ $n + 1$ for equation (z – direction)

$$\begin{aligned} & \frac{[C_{COHb}]_{i,j}^{n+1} - [C_{COHb}]_{i,j}^{n+1/2}}{(\Delta\tau/2)} + V \left(\theta \frac{[C_{COHb}]_{i,j+1}^{n+1} - [C_{COHb}]_{i,j}^{n+1}}{\Delta Z} + (1-\theta) \frac{[C_{COHb}]_{i,j+1}^{n+1/2} - [C_{COHb}]_{i,j}^{n+1/2}}{\Delta Z} \right) \\ &= D_{COHb} \left\{ \frac{[C_{COHb}]_{i+1,j}^{n+1/2} - 2[C_{COHb}]_{i,j}^{n+1/2} + [C_{COHb}]_{i,j-1}^{n+1/2}}{(\Delta R)^2} + \frac{[C_{COHb}]_{i+1,j}^{n+1/2} - [C_{COHb}]_{i,j}^{n+1/2}}{R_i \Delta R} \right\} \quad (9d) \\ & + \lambda_{CO} \theta [C_{CO}]_{i,j}^{n+1} \{1 - [C_{COHb}]_{i,j}^{n+1/2}\} - (1-\theta) \lambda_{CO} [C_{CO}]_{i,j}^{n+1/2} \{1 - [C_{COHb}]_{i,j}^{n+1/2}\} - \lambda_{COHb} [C_{COHb}]_{i,j}^{n+1/2} \end{aligned}$$

On rearranging eq. 9a-9d, a more convenient form for calculation is obtained

$$\begin{aligned} & -D_{CO} \left\{ \frac{\theta}{(\Delta R)^2} + \frac{\theta}{R_i \Delta R} \right\} [C_{CO}]_{i+1,j}^{n+1/2} + \left\{ \frac{\Delta\tau}{2} + \frac{2\theta D_{CO}}{(\Delta R)^2} + \frac{\theta D_{CO}}{R_i \Delta R} + \beta_{CO} \theta \{1 - [C_{COHb}]_{i,j}^n\} \right\} [C_{CO}]_{i,j}^{n+1/2} + \frac{\theta D_{CO}}{R_i \Delta R} [C_{CO}]_{i-1,j}^{n+1/2} \\ &= \left\{ \frac{(1-\theta) D_{CO}}{(\Delta R)^2} + \frac{(1-\theta) D_{CO}}{R_i \Delta R} - \frac{V}{\Delta Z} \right\} [C_{CO}]_{i+1,j}^n + \left\{ \frac{\Delta\tau}{2} + \frac{V}{\Delta Z} - \frac{(1-\theta)}{(\Delta R)^2} D_{CO} - \frac{(1-\theta)}{R_i \Delta R} + (1-\theta) \beta_{CO} \{1 - [C_{COHb}]_{i,j}^n\} \right\} [C_{CO}]_{i,j}^n \quad (10a) \\ & + \frac{(1-\theta) D_{CO}}{(\Delta R)^2} [C_{CO}]_{i-1,j}^n + \beta_{COHb} [C_{CO}]_{i,j}^n \end{aligned}$$

$$\begin{aligned} & \left\{ \frac{\theta V}{\Delta Z} \right\} [C_{CO}]_{i,j+1}^{n+1} + \left\{ \frac{\Delta\tau}{2} - \frac{V\theta}{(\Delta Z)} + \beta_{CO} \theta \{1 - [C_{COHb}]_{i,j}^{n+1/2}\} \right\} [C_{CO}]_{i,j}^{n+1} \\ &= \left\{ \frac{D_{CO}}{(\Delta R)^2} + \frac{D_{CO}}{R_i \Delta R} \right\} [C_{CO}]_{i+1,j}^{n+1/2} - \left\{ \frac{2}{(\Delta R)^2} D_{CO} + \frac{D_{CO}}{R_i \Delta R} + (1-\theta) \beta_{CO} \{1 - [C_{COHb}]_{i+1,j}^{n+1/2} - V \frac{(1-\theta)}{\Delta Z} - \frac{\Delta\tau}{2} \right\} [C_{CO}]_{i,j}^{n+1/2} \quad (10b) \end{aligned}$$

$$\begin{aligned} & -V \frac{(1-\theta)}{(\Delta Z)} [C_{CO}]_{i,j+1}^{n+1/2} + \frac{D_{CO}}{(\Delta R)^2} [C_{CO}]_{i-1,j}^{n+1/2} + \beta_{COHb} [C_{COHb}]_{i,j}^{n+1/2} \\ & -D_{COHb} \left\{ \frac{\theta}{(\Delta R)^2} + \frac{\theta}{R_i \Delta R} \right\} [C_{COHb}]_{i+1,j}^{n+1/2} + \left\{ \frac{\Delta\tau}{2} + \frac{2\theta D_{COHb}}{(\Delta R)^2} + \frac{\theta D_{COHb}}{R_i \Delta R} + \lambda_{CO} \theta \{1 - [C_{COHb}]_{i,j}^n\} \right\} [C_{COHb}]_{i,j}^{n+1/2} + \frac{\theta D_{COHb}}{R_i \Delta R} [C_{COHb}]_{i-1,j}^{n+1/2} \\ &= \left\{ \frac{(1-\theta) D_{COHb}}{(\Delta R)^2} + \frac{(1-\theta) D_{COHb}}{R_i \Delta R} - \frac{V}{\Delta Z} \right\} [C_{COHb}]_{i+1,j}^n + \left\{ \frac{\Delta\tau}{2} + \frac{V}{\Delta Z} - \frac{(1-\theta)}{(\Delta R)^2} D_{COHb} - \frac{(1-\theta)}{R_i \Delta R} + (1-\theta) \lambda_{COHb} \{1 - [C_{COHb}]_{i,j}^n\} \right\} [C_{COHb}]_{i,j}^n \quad (10c) \\ & + \frac{(1-\theta) D_{COHb}}{(\Delta R)^2} [C_{COHb}]_{i-1,j}^n + \lambda_{COHb} [C_{COHb}]_{i,j}^n \end{aligned}$$

$$\begin{aligned}
& \left\{ \frac{\theta V}{\Delta Z} \right\} [C_{COHb}]_{i,j+1}^{n+1} + \left\{ \frac{\Delta \tau}{2} - \frac{V\theta}{(\Delta Z)} + \lambda_{COHb} \theta \{1 - [C_{COHb}]_{i,j}^{n+\frac{1}{2}}\} \right\} [C_{COHb}]_{i,j}^{n+1} \\
& = \left\{ \frac{D_{COHb}}{(\Delta R)^2} + \frac{D_{COHb}}{R_i \Delta R} \right\} [C_{COHb}]_{i+1,j}^{n+\frac{1}{2}} - \left\{ \frac{2}{(\Delta R)^2} D_{COHb} + \frac{D_{COHb}}{R_i \Delta R} + (1-\theta) \lambda_{COHb} \{1 - [C_{COHb}]_{i+1,j}^{n+\frac{1}{2}}\} - V \frac{(1-\theta)}{\Delta Z} - \frac{\Delta \tau}{2} \right\} [C_{COHb}]_{i,j}^{n+\frac{1}{2}} \\
& - V \frac{(1-\theta)}{(\Delta Z)} [C_{COHb}]_{i,j+1}^{n+\frac{1}{2}} + \frac{D_{COHb} [C_{COHb}]_{i-1,j}^{n+\frac{1}{2}}}{(\Delta R)^2} + \lambda_{COHb} [C_{COHb}]_{i,j}^{n+\frac{1}{2}}
\end{aligned} \quad (10d)$$

Each of these equations 9a-9d leads to M-1 equation in M-1 unknowns at each half the-step, $\Delta t/2$. The systems each have a tridiagonal coefficient matrix. The simulation parameters are summarized in Table 2.

TABLE 2: Parameters for the simulation

S/N	Parameter Description	Symbol	Value	Source
1	Diffusion Coefficient of CO in the blood	D_{CO}	$3.0 \times 10^{-5} \text{cm}^2/\text{s}$	Murray and Wyman [23]
2	Diffusion Coefficient of COHb in the blood	D_{COHb}	$2.5 \times 10^{-7} \text{cm}^2/\text{s}$	Murray and Wyman [23]
3	Solubility of CO in the blood	α_{CO}	$1.07 \times 10^{-9} \text{mol/mlmmHg}$	Foster [7]
4	Solubility of O_2 in the blood	α_{O_2}	$1.4 \times 10^{-9} \text{mol/mlmmHg}$	Forster [7]
5	Association rate	m'	18.8/s	Holland [24]
6	Dissociation rate	m	0.062/s	Holland [24]
7	Radius of the pulmonary capillary	r	$4 \times 10^{-4} \text{cm}$	Guyton [25]
8	Total hemoglobin concentration	H	$9.302 \times 10^{-6} \text{mol/ml}$	Holland [24]
9	The partial pressure of O_2	P_{alv}	100 mmHg	Peterson and Stewart [8]
11	Transit time	T	0.75s	Roughton [12]
12	Volume of blood	V_b	5000 ml	Thew and Vaupel [26]

RESULTS AND DISCUSSION

Using the initial COHb of 0.25%, Fig. 3 shows the effects of atmospheric concentration of carbon monoxide on the percentage concentration of carboxylhemoglobin (COHb) in human subject exposed to a constant concentration flux of 100, 150, 200 and 500ppm ambient CO variation of the percentage of COHb. For the human subject exposed to a constant concentration flux of 50ppm ambient CO, it is shown that the COHb increases with continual exposure time to the ambient concentration. As the subject is exposed to the pollutant for the first 2hours, he has 2.8% of the COHb and judging from the table shown in the previous section, the subject will start to experience Cardiac function decrements, aggravation of cardiovascular disease, peripheral arteriosclerosis, blood flow alterations, and after extended exposure of 3-4hours, the subject would have accumulated up to 4.3% of the COHb in his body and he will start to experience visual impairments, vigilance decrements, reduced maximal work capacity, decreased exercise performance and manual dexterity. As the exposure continues, the effects seem to be intensified. For the human subject exposed to a constant concentration flux

of 95ppm ambient CO, as the subject is exposed to the pollutant for the first 1hr, he has 4.0% of the COHb and judging from the table, the subject will start to experience decrements of vigilance (i.e ability to detect small changes in one's environment that occur at unpredictable times), decreased exercise performance. And after a long exposure for the next 5hours, the subject would have accumulated up to 10.0% of the COHb in his body and he will start to experience slight headache, lassi breathlessness from exertion, dilation of blood cells in the skin, abnormal vision, and potential damage to fetuses. As the exposure continues, the effects seem to be intensified and severe damage to the subject health can occur.

For the human subject exposed to a constant concentration flux of 200ppm ambient CO, as the subject is exposed to the pollutant for the first 1hr, he has 4.0% of the COHb, from the table, the subject will start to experience decrements of vigilance (i.e ability to detect small changes in one's environment that occur at unpredictable times), decreased exercise performance. After a long exposure for the next 5hours, the subject would have accumulated up to 10.0% of the COHb in his body and he will start to experience slight headache, lassi

breathlessness from exertion, dilation of blood cells in the skin, abnormal vision, and potential damage to fetuses. As the exposure continues, the effects seem to be intensified and severe damage to the subject health can occur.

For the human subject exposed to a constant concentration flux of 500ppm ambient CO, as the subject is exposed to the pollutant for the first 1hr, he has 4.0% of the COHb, shown in the table, the subject will start to experience decrements of vigilance (i.e ability to detect small changes in one's environment that occur at unpredictable times), decreased exercise performance. And after a long exposure for the next 5hours, the subject would have accumulated upto 10.0% of the COHb in his body and he will start to experience slight headache, lassie breathlessness from exertion, dilation of blood cells in the skin, abnormal vision, and potential damage to fetuses. As the exposure continues, the effects seem to be intensified and severe damage to the subject health can result.

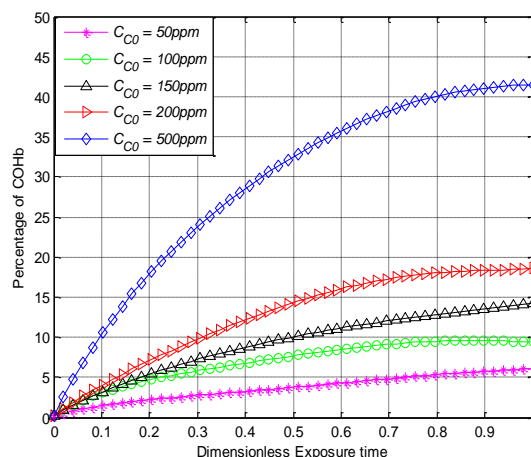


Figure 3. Effects of atmospheric concentration of carbon monoxide on the percentage concentration of percentage carboxylhemoglobin in human subject

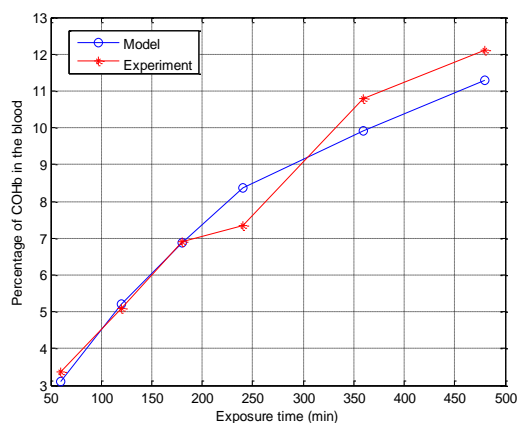


Figure 4. Comparison of Percentage of COHb in human subjects exposed concentration flux of 95ppm ambient CO

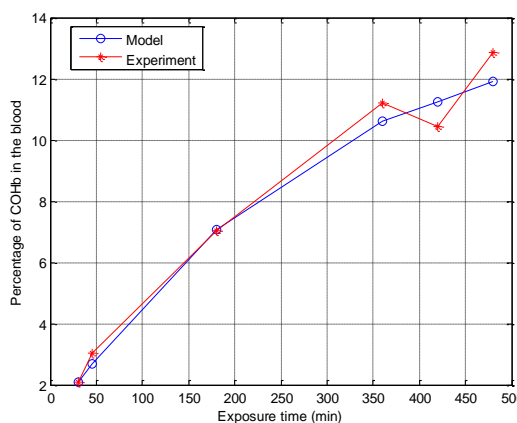


Figure 5. Comparison of Percentage of COHb in human subjects exposed to a constant concentration flux of 100ppm ambient CO.

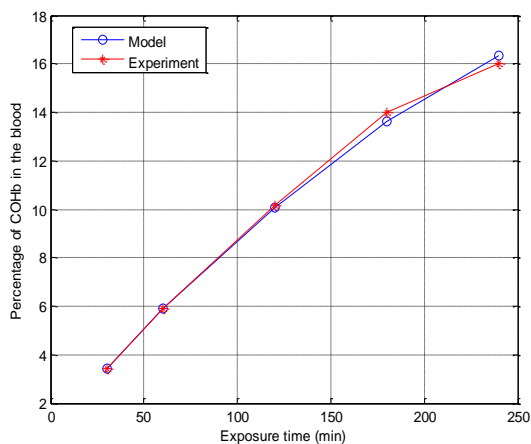


Figure 6. Comparison of Percentage of COHb in human subjects exposed concentration flux of 200ppm ambient CO

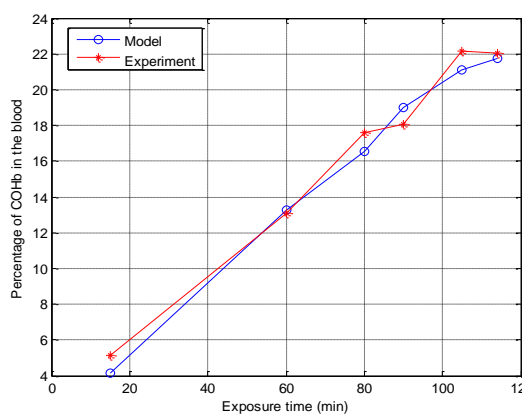


Figure 7. Comparison of Percentage of COHb in human subjects exposed to a constant concentration flux of 500ppm ambient CO.

Figs. 4-7 represent the comparison of COHb concentration in the blood as a function of exposure time computed from the model with that experimental performed (Peterson and Stewart [8]). The results predicted are in good agreement with those measured experimentally.

The standard error of the predicted results from the model range in between 0.5-0.85 for the difference concentrations of ambient carbon monoxide. The low value of the error reveals that the model can actually be used in place of the experiment to predict the level of COHb in the blood thereby predicting the effects of the pollutant on the subject.

CONCLUSION

In this work, mathematical models for the prediction of the effects of carbon-monoxide (CO) on human health have been developed. The developed mathematical models were solved numerically using Alternating-Direct Implicit (ADI) scheme. The concentration of COHb in the blood is computed as a function of the exposure time and the ambient CO from the solution of the differential equations. The computed COHb in the blood from the developed models was used to predict the effects of carbon-monoxide (CO) on human health. The predicted results show good agreement with those measured experimentally (Peterson and Stewart, [8]). The variations in the models parameter indicate significant variations in the results. This can be used as a means of controlling the effects of the pollutant on human health. Also the variables of the models show significant results in their variations. This can be used as a means of controlling the effects of the pollutant on human health. Lastly, these results will serve as a way of evaluating our technological injuries, effectively controlling our pollutants emissions and also as a tool for designing and developing better equipments and engines with lower carbon or pollutants emissions.

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Persian Abstract

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چکیده

امروزه اختراعات با نگاه فناورانه و به کار گیری آن‌ها در جهت مخالف مصالح محیط‌زیستی و به تبع آن ادامه تهدید برای بقای انسان‌های آینده، سریعاً در حال رشد است. برای مقابله با چنین تهدیداتی و پیشگیری از تغییرات نامطلوب در سلامت محیط زیست و انسان، راه اساسی، پیش‌بینی اثر آلودگی است و برای تعیین استراتژی ارزیابی ریسک استفاده از مدل‌های ریاضی پیشنهاد می‌شود. بنابراین، پژوهش حاضر مدل‌های ریاضیاتی را برای پیش‌بینی اثرات احتراق آلاینده‌های تولیدی نظیر مونوکسید کربن (CO) بر سلامت انسان به کار گرفته است. از سیستم ادغامی ارتقاء یافته‌ای از معادله دیفرانسیلی غیرخطی جزئی برای تعیین غلظت مونوکسید کربن محیط که فرد مورد آزمایش در معرض آن قرار داشت استفاده گردید و غلظت کربوکسی‌هموگلوبین (COHb) محلول در خون به صورت عددی با استفاده از Alternating-Direct Implicit (ADI) بدست آمد. بنابر محاسبات، متغیرها نتایج قابل توجهی در گستره تغییرات خود نشان داده‌اند و خطای استاندارد از نتایج پیش‌بینی شده از بازه مدل بین ۰/۵-۰/۸۵ برای غلظت‌های متفاوتی از مونوکسید کربن محیط مشاهده شد. این نتیجه بیانگر این مطلب است که نتایج حاصله با داده‌های تجربی موجود از هم‌بستگی خوبی برخوردارند. بنابراین، این مدل می‌تواند به عنوان ابزاری برای کنترل اثرات آلاینده بر سلامت انسان بکار گرفته شده و از نتایج آن به عنوان ابزاری برای ارزیابی آسیب‌های مرتبط با تکنولوژی خود، کنترل موثر انتشار آلاینده‌ها و نیز برای طراحی و ارتقاء بهتر تجهیزات و موتورها با انتشار کمتر گاز مونوکسید کربن و دیگر آلاینده‌ها استفاده نمود.