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Cigarette Smoking Kinetic Model Reveals Profound Effects among Passive Smokers Compared with Cigarette Smokers

[1]Ahed J. Alkhatib, [2]Ahmad M. Boran, [3]Mohd G. Sghaireen and [1]Osama A. Alkouri
[1]Jordan University of Science and Technology, 22110 Irbid, Jordan
[2]The Royal University of Medical Sciences, Jordan
[3]Aljouf University, KSA, Saudia Arabia

Abstract: Smoking is associated with various adverse health effects. Among the chemicals associated with smoking are carbon monoxide and carboxyhemoglobin. The present study was conducted to investigate levels of carbon monoxide and carboxyhemoglobin among a sample of Jordanian students at cafes in Irbid City, Jordan. Both carbon monoxide and carboxyhemoglobin were measured before initiating smoking and after 1 h. The study included 236 participants of whom 102 passive smokers and 134 cigarette smokers. Carbon monoxide and carboxyhaemoglobin were measured in breathing by a device designed to measure both of carbon monoxide and carboxyhaemoglobin called carbon monoxide monitor (piCO+™ Smokerlyzer). Study findings showed that before initiating smoking, passive smokers had CO levels of 2.8 ppm and carboxyhemoglobin 2.9% while cigarette smokers had CO levels of 35.4 ppm and carboxyhemoglobin 35.7%. After 1 h of smoking, passive smokers had more increased levels of carbon monoxide 10.1 ppm and carboxyhemoglobin 9.6%. Among cigarette smokers after 1 h smoking, carbon monoxide levels became 38.2 ppm and carboxyhemoglobin 39%. Taken together, the results showed that carbon monoxide and blood carboxyhaemoglobin resulting from smoking exceed the normal levels into dangerous levels. Passive smokers are exposed to real smoking dangers and more affected by ambient smoking exposure than cigarette smokers.

Key words: Passive smokers, cigarette smokers, carbon monoxide, carboxyhemoglobin, Jordan

INTRODUCTION

Tobacco smoke generates carbon monoxide for smokers and non-smokers. It can also generates other chemicals with which environmental carbon monoxide may interact. It has been pointed that acute and chronic carbon monoxide exposure from tobacco smoke can affect the cardiopulmonary system (McNicoll, 2002).

It has been shown that cigarette smoking has 2 phases, a tar and a gas phase. The tar, particulate phase is the residual material that is being trapped when the smoke stream is passed through the cambridge glass-fiber filter that retains 99.9% of all particulate material with a size >0.1 μm (Pryor and Stone, 1993). On the other hand, the gas phase presents the material that passes through the filter. It has been reported that about 1017 free radicals/g to be contained in the particulate (tar) phase of cigarette smoke while the gas phase contains 1015 free radicals/puff (Pryor and Stone, 1993). Furthermore, it has been indicated that radicals within the tar phase have long long-lived (hours to months) whereas the radicals associated with the gas phase have a shorter life span (seconds) (Pryor *et al*., 1998; Smith and Fischer, 2001).

Mainstream smoke is a cigarette smoke that is drawn through the tobacco into an active smoker's mouth while sidestream cigarette smoke is the smoke emitted from the burning ends of a cigarette. Mainstream cigarette smoke comprises 8% of tar and 92% of gaseous components (Pryor and Stone, 1993).

Environmental tobacco smoke is a combination from sidestream smoke (85%) and a small fraction of exhaled mainstream smoke (15%) from smokers (Taylor *et al*., 1992). Furthermore, sidestream cigarette smoke has a relatively higher concentration of the toxic gaseous component than mainstream cigarette smoke (Glantz and Parmley, 1991). According to Powell (1998), nicotine which is a component of the tar phase is considered the addictive substance of cigarette smoke.

From a chemical point of view, Carbon Monoxide (CO) consists of one carbon and one oxygen molecule. Furthermore, it has a small dipole moment and a partial triple bond. It has been indicated that the bond between carbon and oxygen in CO to be the strongest bond in chemistry, 1076.4 kJ mol^{-1} (Christian *et al*., 2007). CO is a colorless and odorless gas, with boiling point at minus 191.5°C and melting point at -205°C (Budavari, 1996).

Corresponding Author: Ahed J. Alkhatib, Jordan University of Science and Technology, 22110 Irbd, Jordan

Although, CO is a stable molecule, it can be reduced by transition metals. Coordinated CO is more reactive than the free gas. It is known that proteins that have transition metals at the active site can form coordinated complexes with CO that have the ability to interacts with their function as an example in red blood cells CO bind with the iron centre in the haemoglobin molecule (Piantadosi, 2002).

Von Burg (1999) reported that endogenous production of CO leads to blood Carboxyhaemoglobin (COHb) concentrations of 0.4-1%. In later studies, it has been reported that COHb formation is typically induced by endogenous CO production, exogenous CO exposure or both (Morse and Choi, 2002; Smithline *et al*., 2003). When there is burn and smoke inhalation injury, large amounts of CO are inhaled which induces more COHb formation (Maestrelli *et al*., 2001). It is worth to mention that measurement of COHb concentrations in the blood of thermally injured patients has been considered to be a biological marker of both severity of inhalation injury and efficacy of treatment strategies (Clark *et al*., 1986; Mehta *et al*., 2001).

In the present study, researchers studied a kinetic smoking model through 1 h by measuring carbon monoxide and carboxyhemoglobin.

MATERIALS AND METHODS

This is a cross sectional study. A questionnaire was used as a tool to collect the data from study participants. Study participants included 236 participants of whom 102 passive and 134 cigarette smokers. Carbon monoxide and carboxyhaemoglobin were measured in breathing by a device designed to measure both of carbon monoxide and carboxyhaemoglobin called carbon monoxide monitor (piCO+™ Smokerlyzer). Researchers visited several coffee shops in Irbid City to collect data from participants.

RESULTS

As shown in Table 1, the study included 236 participants of which 102 passive smokers and 134 cigarette smokers.

As seen in Table 2, the mean level of CO among passive smokers was 2.8 ppm and was among cigarette smokers 35.4 ppm. This variation in level of CO among passive and cigarette smokers was statistically significant (p = 0.000). Carboxyhemoglobin percentage increased significantly (p = 0.000) from 2.9% among passive smokers to 35.7% among cigarette smokers.

As shown in Table 3, after 1 h of smoking, the level of carbon monoxide became in passive smokers (10.1 ppm) and among cigarette smokers (38.2 ppm) and this variation was statistically significant (p = 0.000). On the other hand, the level of carboxyhemoglobin became after 1 h 9.6% among passive smokers and 39% among cigarette smokers. The variation in carboxyhemoglobin level among passive and cigarette smokers was statistically significant (p = 0.000).

As shown in Table 4, before starting smoking of passive smokers, 9 smokers had carbon monoxide levels >10 ppm and this number increased up to 37 passive smoker. On the other hand of cigarettes smokers, 123 smokers had their monoxide level >10 ppm and this number increased to 130 smokers with carbon monoxide levels over 10 ppm.

As shown in Table 5, before starting smoking of passive smokers, there were 9 passive smokers with high levels of carboxy hemoglobin >3% and this number increased o 35 passive smokers with carboxyhemoglobin >3%. The results also showed that of 134 cigarette smokers, 122 had elevated levels of carboxyhemoglobin

Table 1: Frequency of smokers

Type of smoking	No. of cases	Percentage
Passive	102	43
Cigarette	134	57
Total	236	100

Table 2: The level of 1st readings of CO and CO-HB

	CO		CO-HB	
Variables	Concentration (ppm)	p-value	Percentage	p-value
Type of smoking				
Passive	2.8	0.000	2.9	0.000
Cigarette	35.4		35.7	

Table 3: The level of 2nd readings of CO and CO-HB

	CO		CO-HB	
Variables	Concentration (ppm)	p-value	Percentage	p-value
Type of smoking				
Passive	10.1	0.000	9.6	0.000
Cigarette	38.2		39.0	

Table 4: The frequency and distribution of smokers during measurements of carbon monoxide

	1st reading of CO		2nd reading of CO	
Smoking types	Danger (10 ppm, n)	Normal (≤10 ppm)	Danger (>10 ppm, n)	Normal (≤10 ppm, n)
Passive	9	93	37	65
Cigarette	123	11	130	4

Table 5: The frequency and distribution of smokers during measurements of carboxyhemoglobin

	1st reading of Co-HB		2nd reading of Co-HB	
Type of smoking	Normal (≤3%, n)	Danger (>3%, n)	Normal (≤3%, n)	Danger (>3%, n)
Passive	93	9	67	35
Cigarette	12	122	4	130

>3% and this number was further increased to 130 cigarette smokers with high levels of carboxyhemoglobins >3%.

DISCUSSION

Breath carbon monoxide levels and its equivalent carboxyhaemoglobin percentage in blood were studied among participants in Irbid cafes. No previous studies had been conducted to address this problem in these areas. Furthermore, little information is available in regard to dynamic changes of carbon monoxide and carboxyhaemoglobin levels during smoking.

Initial readings showed that passive smokers had levels of both CO and COHb within physiological range (<3 ppm and 3%, respectively). While the levels among cigarette smokers were significantly high for both CO and COHB (CO, 35.4 ppm; COHb, 35.7%; p = 0.000). These findings give an evidence that cigarette smokers are under continuous risk. After 1 h, measurement of CO reached a high level (10.1 ppm) and among cigarette smokers, CO levels increased to 38.2 ppm. The difference among passive and cigarette smokers is still statistically significant (p = 0.000). The same observation is seen for COHb measurements where passive smokers increased high levels of COHb 9.6% and cigarette smokers percentage of COHb increased to 39%. The results showed that the passive smokers are more affected by carbon monoxide, than the other groups of smokers and that may due to the ability of their lungs to acquire more carbon monoxide from breath. This is also true for carboxyhaemoglobin. Carboxyhaemoglobin which is considered the real biological indicator for the real exposure to carbon monoxide, seems to be completely resulted from conversion of carbonmonoxide. However, the data of the present study are in agreement with other reported studies in literature.

As an example Von Burg (1999) reported that endogenous production of CO leads to blood, Carboxyhaemoglobin (COHb). The finding are also in line with studies conducted by Morse and Choi (2002) and Smithline *et al.* (2003). Large amounts of CO and COHB after 1 h of initiating smoking can be explained according to study of Maestrelli *et al.* (2001) who reported that the presence of burn and smoke inhalation injury, large amounts of CO are inhaled which induces more COHb formation.

The data of the present study showed how within 1 h, 9 passive smokers who had >10 ppm, increased to 37 persons under the same conditions while 123 cigarette smokers with CO >10 ppm, increased to 130 participants under the same conditions. Some participants after 1 h, smoking still have normal measurements of both CO and COHb which may indicate to the outdoor status and other conditions.

CONCLUSION

The results showed that breath carbon monoxide and blood carboxy haemoglobin resulting from smoking exceed the normal levels into dangerous levels. Passive smokers are exposed to real smoking dangers and more affected by ambient smoking exposure than cigarette smokers.

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