# A Biochemical Study on the impact effect of Carbon monoxide indoor intoxication on children and adults

*By*

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***B. Sc.in Biochemistry Ain Shams University,*** ١٩٧٩

***Master in Environmental Science, Ain Shams University,*** [illegible]

**A Thesis Submitted in Partial Fulfillment**
*Of*
**The Requirement for the Doctor of philosophy**
*In*
**Environmental Science**

**Department of Environmental Basic Science**
**Institute of Environmental Studies & Research**
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# ENGLISH SUMMARY

Acute carbon monoxide (CO) poisoning is one of the most frequent causes of intoxication and is the most common cause of household toxic death. The accidental carbon monoxide intoxication is usually most prevalent in low economic and rural areas during winter carbon monoxide is a product of combustion of organic matter under conditions of restricted oxygen supply, which prevents complete oxidation to carbon dioxide.

Common sources of carbon monoxide that may lead to indoor toxication include hous fires, furnaces or heaters, wood – burning stoves, and propan fueled equipment such as portable campin g stoves, ice resurfaces, forklifts, gasoline – powered tools such as high pressure washers, floor buffers, welders used in buildings or semienclosed spaces and cigarette smoking. Carbon monoxide is also introduced through emissions from variety of combustion sources and in the infiltration or ventilation air from outdoors.

Toxicity primarily is due to cellular hypoxia which is caused by impeded oxygen delivery. Carbon monoxide binds hemoglobin resulting in a state of relative anemia, as it binds hemoglobin ٢٣٠ – ٢٧٠ times more avidly than oxygen. Thus even small concentration, can result in significant levels of carboxy hemoglobin. Once the gas is inhaled, it is readily absorbed through the lungs and takes place of oxygen that is normally carried in the red blood cells. Tissues; most importantly the brain become starved for life sustaining oxygen.

Symptoms of mild poisoning poisoning include headaches, vertigo, and flu – like effects making the initial diagnosis may be difficult and are often misinterpreted, so it is known as the "silent killer". Larger

بسم الله الرحمن الرحيم

(قَالُواْ سُبْحَانَكَ لاَ عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ)

صدق الله العظيم

سورة البقرة الآية ٣٢

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| | |
|---|---|
| A.G | Anion Gap |
| ABGs | Arterial blood gases |
| ACh | Acetylcholine |
| ADP | Adenosine diphosphate |
| ALT | Alanine amino transferase |
| AMP | Adenosine monophosphate |
| AST | Aspartate amino transferase |
| ATP | Adenosine Triphosphate |
| BuCHE | Butyryl cholinesterase |
| CK | Creatine kinase |
| $Cl^-$ | Chlorine ions |
| CNS | Central Nervous System |
| CO | Carbon monoxide |
| $CO_2$ | Carbon dioxide |
| COHb | Carboxyhemoglobin |
| COX | Cytochrome – C Oxidase |
| CPK | Cretaine phospho kinase |
| CT | Computed Tromographic |
| DA | Dopamine |
| DOPA | Dihydroxy phenylalanine |
| EDTA | Ethylene diamine tetra acetic acid |
| G ٦ P.DH | Glucose ٦ – Phosphate dehydrogenase |
| GGT | Gamma glutamyl transpeptidase |
| GOD | Glucose oxidase |
| GSH | Reduced glutathione |
| Hb | Hemoglobin |
| $HCO^-_3$ | Carbonates |
| HCT | Hematocrite |
| HK | Hexokinase |
| HK | Hoxokinase |
| HO | Heme Oxygenase |
| ٥HT | ٥-Hydroxy tryptamine |
| IU | International unit |
| $K^+$ | Potassium ions |
| L | Liter |
| N | Normal |
| N.S | Non significant |
| $Na^+$ | Sodium ions |
| NAAQS | National Ambient Air Quality Standards |

| | |
|---|---|
| NADPH | Nicotinamide Adenine Dinucleotide Phosphate |
| NE | Norepinephrine |
| NO | Nitric Oxide |
| $O_2Hb$ | Oxyhemoglobin |
| OPT | Orthophaladehyde |
| OSSG | Oxidized glutathione |
| P | Plasma |
| PCC | Poison Control Center |
| PCHE | Pseudo Cholin Esterase |
| $PCO_2$ | Partial Pressure of Carbon dioxide |
| PCr | Phosphocreatine |
| $PO_2$ | Partial Pressure of Oxygen |
| Ppm | Part per million |
| ROS | Reactive oxygen species |
| S | Serotonin |
| SPSS | Statistical Package for social science |
| THB | Tetrahydro Bioptrein |
| U | Unit |
| UV | Ultra violet |
| WHO | World Health Organization |

# ABSTRACT

Carbon monoxide (CO) is a significant toxic gas. It is called a “silent killer”. Acute CO poisoning is the most common type of fatal poisoning in many countries as it is one of the most frequent causes of intoxication and is the most common cause of household toxic death. This study aimed to carry out a comparative study of the effects of CO intoxication on Egyptian adults and children’s, some blood gases most important biochemical & hematological parameters, enzymes, neurotransmitters and hormone insulin. This work was conducted on cases admitted to Poison Control Center (PCC) Ain Shams University Hospitals with complaint diagnosed as CO poisoning during the winter of ٢٠٠٦ – ٢٠٠٧ and then classified according to age into ٢ groups: Gr I A.P (adult patients group) aging from ١٨ – ٤٠ years and Gr II CH.P (Children patients group) aging from ٢ - ١٠ years. Each group consisted of ٤٠ patients. Control cases were also classified according to their ages into ٢ groups GR III $NC_{١}$ (adult control group) and Gr IV $NC_{٢}$ (children control group). Each group consisted of ٢٠ normal persons.

There was a significant increase in blood pH, COHb, $K^{+}$, plasma glucose, insulin, urea, creatinine, ALT, AST, GGT and CPK in adults and children, while a significant decrease in $PCO_{٢}$, $PO_{٢}$, $HCO^{-٣}$, $O_{٢}Hb$, blood $Na^{+}$, plasma P.cholinesterase, serotonin, dopamine, and norepinephrine was recorded in both adults and children. A non significant change in hemoglobin, hematocrite and A.G was recorded in both groups. Chlorine ions were significantly decreased in children while it was insignificantly changed in adults.



**Key wards:**

Carbon monoxide poisoning – Blood gases – indoor intoxication – Hematology – neurotransmitters – Insulin.

# INTRODUCTION

Acute carbon monoxide (CO) poisoning is one of the most frequent causes of intoxication and is the most common cause of household toxic death (**Deshamps, *et al.,* ٢٠٠٣**).

Carbon monoxide poisoning occurs after the inhalation of carbon monoxide gas. Carbon monoxide is a product of combustion of organic matter under conditions of restricted oxygen supply, which prevents complete oxidation to carbon dioxide ($CO_٢$).

Common sources of carbon monoxide poisoning include housefires, automobile exhaust, water heaters, kerosene space heaters, and furnaces. Stoves used for cooking and heating during outdoor activities also produce significant amounts of carbon monoxide, kerosene stoves also produce carbon monoxide when burned in a small tent (**Thomassen, *et al.,* ٢٠٠٤**).

Each year particularly during the heating season (winter), thousands of people are poisoned by carbon monoxide, with potentially devastating outcomes.

Carbon monoxide is a significantly toxic gas, and carbon monoxide poisoning is the most common type of fatal poisoning in many countries (**Omaye, ٢٠٠٢**). Symptoms of mild poisoning include headaches, vertigo, and flu – like effects making the initial diagnosis can be difficult and are often misinterpreted, so it is known as "silent killer" (**Krenzelok *et al.*, ١٩٩٦**). Larger exposures can lead to significant toxicity of the central nervous system, heart and even death. Once the gas is inhaled, it is readily absorbed through the lungs it binds tightly with hemoglobin and takes the place of oxygen that is normally carried in the red blood cells. Tissues; most importantly the brain; became starved for life sustaining oxygen (**Smoots & Wood, ١٩٩٨**).

Toxicity primarily is due to cellular hypoxia which is caused by impeded oxygen delivery. Carbon monoxide binds hemoglobin resulting in a state of relative anemia, as it binds hemoglobin ٢٣٠ – ٢٧٠ times more avidly than oxygen. Thus even small concentration, can result in significant levels of carboxy hemoglobin (COHb) (**Piantodosi,** ١٩٩٦**; Klasner *et al.,*** ١٩٩٨**; Olsen,** ٢٠٠٥). Long-term sequelae often occur. Carbon monoxide can also have severe effects on the fetus of a pregnant woman.

Hemoglobin, myoglobin and mitochondrial cytochrome oxidase are thought to be the mechanisms by which carbon monoxide produces toxic effects (**Gorman, *et al.,*** ٢٠٠٣).

Domestic carbon monoxide poisoning can be prevented by early detection with the use of household carbon monoxide detectors.