

Indoor air pollution and fatal daily household habits: two cases report in Kigali, Rwanda

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ABSTRACT

BACKGROUND: Around 3 billion people around the world, especially in low-income countries, cook indoors using polluting open fires or simple stoves fueled by kerosene, biomass and coal, which produce indoor air pollution (IAP). According to WHO, the population of sub-Saharan Africa, South and East Asia and the Western Pacific experience the highest rates of health problems from exposure to indoor pollutants. According to the World Bank sustainable energy database, only one third of the Rwandan population have access to electricity.

CASE PRESENTATION: Two case reports with both a fatal acute and chronic outcome of IAP are presented. Because of the lack of electricity, especially in poor environments, IAP is a major risk factor for increased mortality. The health impact of IAP is exemplarily discussed on two case reports how acute high exposure to carbon monoxide for instance can lead to fatal poisoning and death of the whole family.

CONCLUSION: The indoor air poisoning can affect people who are often unaware of the existence of a toxic gas, caused by combustion of biomass in their homes. Therefore community-based preventive intervention trials are needed to educate and alert the people.

Keywords: Indoor air pollution; Rwanda; East Africa; carbon monoxide; gas poisoning; WHO; World Bank sustainable energy database.

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INTRODUCTION

An estimated 3 billion people around the world, especially in low-income countries, cook indoors using polluting open fires or simple stoves fueled by kerosene, biomass and coal, which produce indoor air pollution (IAP) [1]. According to WHO, people in sub-Saharan Africa, South and East Asia and the Western Pacific experience the highest rates of health problems from exposure to indoor pollutants.

The health problems they face are non-communicable diseases including stroke, ischemic heart disease, chronic obstructive lung disease and lung cancer as well as carbon monoxide gas poisoning which lead to premature death [2]. The indoor use of these fuels leads to a higher level of indoor air pollutants than international ambient air quality standards allowed. Women and their children at low socioeconomic level are predominantly affected because of being normally responsible for food preparation and cooking on the daily basis and infants being usually close to

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their mothers [3]. This problem is exemplarily discussed on two case reports regarding acute and chronic health impacts due to IAP in Kigali, Rwanda.

Case 1

The unidentified man was found dead near the roadside in Kigali, Rwanda and taken to the Kigali Police Hospital Mortuary for further investigation. No other information was available. The external investigation showed a 50 – 60 year old undernourished male with a body length of 170 cm and a body weight of 45 kg (BMI: 15,6 kg/m²). He was fully clothed. There were no signs of trauma or injection marks. No conjunctival and oral mucosa bleeding noted

The autopsy findings revealed severe anthracosis in both lungs, hili, paratracheal and peribronchial lymph nodes. He suffered from a chronic adhesive pleuritis with extended scarification of the pleural cavity and a chronic bronchitis (Figure 1).

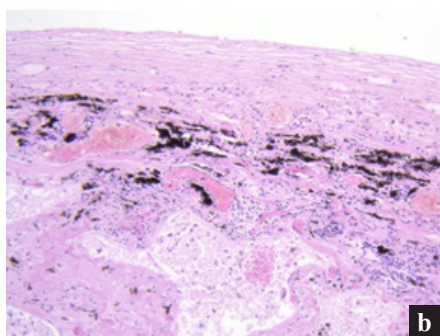
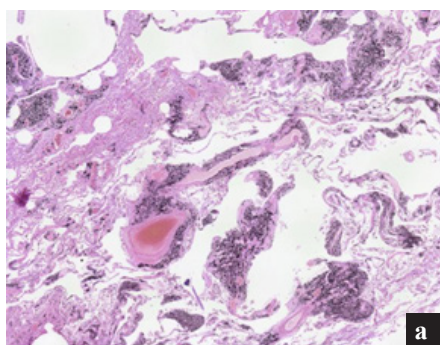


Figure 1a, 1b: Severe anthracosis and pleura fibrosis of the lung (HE, 50x)

The autopsy found chronic pulmonary emphysema pulmonary fibrosis, purulent bronchitis and bronchopneumonia with consecutive right ventricular insufficiency and chronic blood stasis in the liver and spleen.

No further macroscopic internal disease or cerebral alteration were diagnosed (Figure 2).

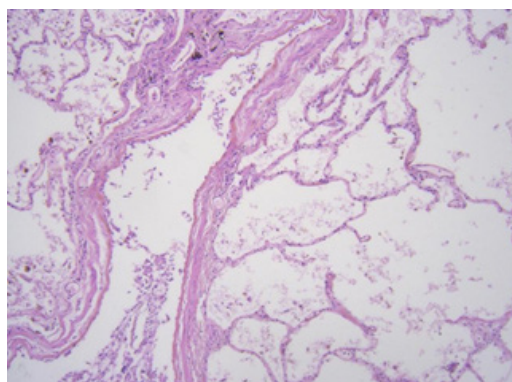


Figure 2: Severe emphysema of the lung

The cause of death was pulmonary insufficiency caused by severe chronic anthracosis, COPD and pulmonary fibrosis, complicated by purulent bronchitis and confluent bronchopneumonia.

The histology revealed severe anthracosis, pleura fibrosis, chronic emphysema and advanced confluent pneumonia in the Hematoxylin and Eosin coloration.

Case 2

A whole family was found dead in their house. The house was divided into two rooms. The father was found close to the house entrance while his wife and 7-months-old infant were found in the rear end of the house. The windows and the door were completely closed. Next to the mother and the child, a cooking station that had been prepared with fire wood was found.

The mother and the child were already dead. The father died shortly after being brought to the hospital.

The autopsy of the father showed no fatal pathologies, except slightly enlarged heart and a moderate coronary artery disease.

Heart-blood samples were taken from the mother and the child by opening the thorax. Salmon-pink-coloured muscle and light-red blood were revealed as a hint for a carbon monoxide poisoning.

The postmortem blood samples of all family members were tested in the toxicological laboratory in Hamburg, Germany.

The blood analysis revealed a 77% and 40% toxic CO-poisoning of the mother and of the child respectively. The result of COHb analysis of the father was <10%, because he had survived for several hours.

The cause of death was acute carbonmonoxide poisoning. The motives of death was defined as unnatural.

The cases were investigated during the forensic summer schools in Kigali, Rwanda, through a cooperation project between the Institute of Legal Medicine in Hamburg, Germany and the University of Rwanda.

DISCUSSION

Carbonmonoxide is a colourless, odourless and tasteless toxic gas and is the most common and widely distributed air pollutant, both in domestic and work environment. It accounts for numerous cases of carbonmonoxide poisoning every year. Carbon monoxide is produced by incomplete combustion of carbonaceous material such as charcoal and wood under high temperature and low oxygen supply [4]. Even at a combustion temperature of 1000°C the chemical balance of carbon and carbon dioxide ($C + CO_2 \leftrightarrow CO$, so-called “Boudouard equilibrium”) is almost completely on the side of the CO. The most original form of CO exposure is therefore the accumulation of the gas in charcoal or wood heated dormitories or the exit of CO from enclosed stoves and lamps in poorly or nonventilated rooms [5].

The gas prevents the binding of oxygen to the hemoglobin in the body. As a result, the blood can no longer transport the vital oxygen. The result is an oxygen deficiency of the tissue, which leads to headache, dizziness, tinnitus, blurred vision, vomiting, fatigue, muscle weakness and accelerated heartbeat in mild intoxication cases. Moderate and severe intoxications include shortness of breath and loss of consciousness with flattening of breathing. Mechanical Ventilation and oxygen supply can prevent death from this kind of intoxication. The result of prolonged respiratory disorders can be temporary or permanent brain damage [6, 7].

Because of the lack of electricity, especially in resource-limited environments, the indoor air pollution (IAP) is a major risk factor for increased mortality. According to the World Bank sustainable energy database, only one third of the Rwandan population has access to electricity (Figure 3).

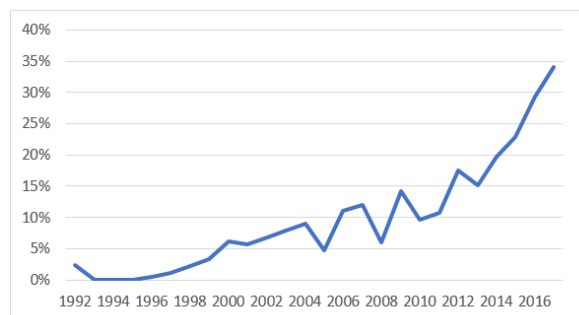


Figure 3: Access to electricity in Rwanda 1992 – 2017 [1]

Widely available, low-cost traditional energy sources such as coal and biomass (dung, wood, crop residues) are still the main source of energy for 60 to 90% of households in developing countries and are used for cooking, home heating and lightening [8]. It accounts for about 4% of the global burden of disease measured by disability-adjusted life years (DALYs) lost. In contrast to electricity, biomass is extremely polluting.

In addition to harmful pollutants and irritant gases, there are also carbon monoxide (CO), nitrogen dioxide (NO₂), sulphur dioxide (SO₂), formaldehyde, and carcinogens such as benzopyrene and benzene found in the smoke [2, 3, 9-11]. In developing countries, the international ambient air quality standards may be exceeded by a factor of 10, 20 and up to 50, exceeding even high levels found outdoors in coal-burning cities in northern China [12].

The small pollutants in indoor environments are able to penetrate deep into the lungs and appear to have a great health damage potential [13]. This leads to acute and chronic exposure and different health issues.

Acute high exposure to carbon monoxide can lead to fatal poisoning. This poisoning can affect people who are often unaware of the existence of a toxic gas, caused by combustion of biomass in their homes (Figure 4).



Figure 4a, 4b: Cooking on solid fuels, Musanze, Rwanda. Photo credit to J. Byukusenge

Carbon monoxide is absorbed by the body through inhalation and is diffused across the alveolar membrane with almost the same ease as oxygen (O_2).

The effect of CO on the human body is threefold: First, CO displaces the oxygen molecules (O_2) easily because of the approximately 200-300-fold (in fetal hemoglobin even 600-fold [LIT 29]) affinity to Iron (II) binding sites of hemoglobin and thereby reduces the oxygen transport capacity of the blood by forming carboxyhemoglobin (COHb) [14].

Second, CO ligands cause an allosteric change in hemoglobin, which enhances the binding of the remaining O_2 molecules, thereby releasing less oxygen into the tissues (physiologically, this manifests as left-shift of the oxygen-binding curve) [15].

The third effect is based on the fact that CO also binds to iron (III) centers - but much weaker than, for example, the cyanide ion of hydrocyanic acid. Therefore, intracellular CO interferes with the function of ferricytochrome enzymes such as cytochrome c oxidase, resulting in prolonged persistence, which leads to cytotoxic hypoxia [16]. Because all these effects are tissue hypoxia, CO poisoning manifests itself first of all in the hypoxia-sensitive central nervous system, and later in the myocardium. This is because CO has only a 40-fold affinity to myoglobin compared to oxygen [16, 17].

All effects are important in acute toxicity in the medium dose range. Peracute courses with death within minutes seem to be dependent on the cardiovascular history of the patient. The permanent neurological damage described by some authors after CO intoxication is mainly attributed to the cytotoxic effect of CO [7]. Long exposure time and late start of treatment is therefore also a significant health risk even with less severe clinical symptoms.

Since poisoning is difficult to identify, CO is referred to as a “silent killer”: It is, as mentioned above, invisible and odorless disease and does not cause perceptible sensations or inhalation symptoms. Back in the years many deadly poisonings were interpreted as diabolical causes, because the causes were not recognizable and therefore the death was apparently a connection with occult acts (in which often coal fire and incense played a role) [5].

The most important guiding symptom of acute poisoning is the reduction of vigilance (Table 1). In addition to the clinical symptoms, the assessment of the severity of poisoning can be based on the COHb fraction [17, 18]. Both the absorption behavior of COHb and oxygen hemoglobin cannot be differentiated in conventional pulse oximeters, because of their similarity [19]. The transcutaneous oxygen saturation measurement therefore often

shows false-normal values after CO exposure [20]. A high concentration of COHb leads to a bright red discoloration of the blood. However, this specific sign manifests clinically reliably only postmortem in the form of distinct bright red lividity, while the conspicuous reddening of the skin mentioned in textbooks is actually rarely observed [6]. The COHb value can be determined with conventional blood gas analyzers. The preclinical CO meters and multi-wave length pulse oximeters (“Rainbow” Technique) are used [19].

Preclinical CO pulse oximetry enables rapid differential diagnosis and early stratification in the sighting of larger numbers of patients.

There is persistent evidence that chronic exposure to indoor pollutants cause severe diseases, including acute lower respiratory infections (ALRI) in children and chronic obstructive lung disease (COPD) in adults as well as severe anthracosis and lung cancer [3, 21, 22]. Other illnesses attributed to IAP include eye irritation, perinatal mortality, low birth weight, increased susceptibility to asthma and middle ear infections in children, tuberculosis, cataracts and cancers of the nasopharynx and larynx in adults [2, 3, 10, 23-25].

According to the World Health Report 2002, 2.7% of the total global burden of bad health measured as DALY’s is attributable to IAP (Figure 7). Indoor air pollution is on the fourth rank in developing countries with high mortality after underweight, unsafe sex and unsafe water, sanitations and hygiene [26]. Globally it is on the eighth rank among other risk factors. These risks are not distributed evenly across countries, or even within countries [26]. The health risks of indoor air pollution are strongly correlated with poverty as marked in the case reports above. There is a need in revealing these cases through forensic work to bear with this diagnosis in mind and to investigate the concentration of COHb in blood in forensic laboratories, because a CO poisoning cannot be diagnosed on a macroscopic and microscopic level.

Carbonmonoxide poisoning can be suspected by careful analysis of the circumstances and the scene of death, especially one or multiple persons in a closed room with an open fire or ash. The external examination, concerning bright red livor mortis

(indication for CO poisoning) on dark skinned people is not easy.

Nevertheless the typical bright-cherry-red colour of the livor mortis can be found on the palm, the nail bed (Figure 5),



Figure 5: Typical pink nail beds after carbon monoxide poisoning [27]

The mucosa of the mouth and the conjunctivae. Thus these signs are only indications, and not irrefutable proofs. During the autopsy the muscles are – as in textbooks described - salmon-coloured, and the blood is bright red. The suspected diagnosis can be precised with some special pretests; the commonly used is the is the “Formalin-Test”, blood mixed with formalin. In case of COHb, the fluid remains reddish (Figure 6) [27].



Figure 6: Formalin-Test: blood mixed with formalin; Brownish colour: cyanotic blood; Reddish colour: COHb = Carbon monoxide poisoning

In case of cyanotic blood the fluid becomes brownish. Another pretest is the “Heating Test”. By heating the blood in test tubes the COHb blood remains reddish, while cyanotic blood becomes brownish again.

These pretests are easy to perform and are indicators for the right diagnosis [28].

In conclusion, indoor air pollution is a major environmental risk to health. By reducing indoor air pollution levels, the burden of disease from stroke, heart diseases, lung cancer, acute and chronic respiratory diseases as well as accidental fatal gas

poisoning can be reduced in developing countries. Community-based preventive intervention trials are needed to educate and alert the population at risk. Good ventilation, use of improved cooking stoves, access to gas and electricity and an indoor, handy CO measuring device can reduce the exposure to smoke or CO gas.

Table 1: Symptoms of carbon monoxide poisoning

Severity	Symptoms	CO-Hb-fraction
Light	Shortness of breath during exercise, headache	>10%
	Dizziness, tinnitus, nausea and vomiting	>20%
	Tiredness, blurred vision	>30%
Moderate	Tachycardia, loss of consciousness, cardiac arrhythmias	>40%
	Coma, tachypnea	>50%
Severe	Seizures, Cheyne-Stokes respiration	>60%
	Respiratory and circulatory arrest	60 – 80%

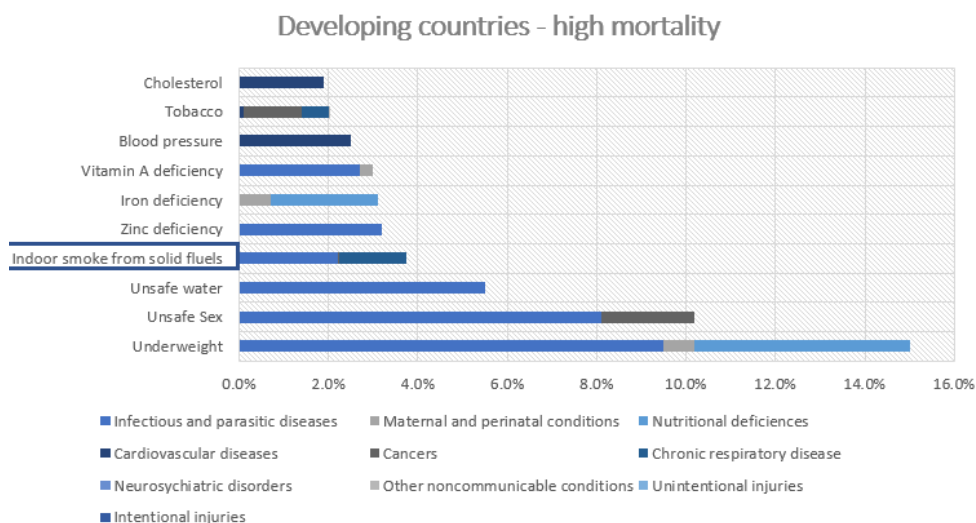


Figure 7: Burden of disease attributable to 10 selected risk factors, by the World Health Report 2002 [26]

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