

## Human Biomonitoring of Maternal exposure to Carbon Monoxide in the First Trimester of Pregnancy in the Core Niger Delta

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### Abstract

**Background:** In the Niger Delta area of Nigeria, human medical biomonitoring, including that of maternal Carboxyhemoglobin (MCOHb) had never been performed irrespective of the devastating environmental pollution in the region. **Aim:** The goals of the study were to quantify the impact of maternal exposure to CO in the first trimester of pregnancy in the core Niger Delta by measuring MCOHb concentrations and to assess the effect of maternal demographic and obstetric characteristics on the impact. **Material and methods:** The study was of cross-sectional design carried out at the Rivers State University Teaching Hospital (RSUTH) in Nigeria. 490 consecutive pregnant women in the first trimester were recruited from the antenatal clinic from January 2021 to January 2022. Gestational age was estimated with the aid of an ultrasound scan. Demographic, social and obstetric characteristics were taken. MCOHb concentrations were measured with the aid of a smokerlyser. Data was analyzed, using SPSS version 25.0 (Armonk, NY) software. Ethical approval was obtained from the RSUTH Ethics Committee. **Results:** The mean value of MCOHb concentration was  $1.15 \pm 0.40\%$ . Out of the 490 patients that were assessed, 461 (94.08%) had mild impact from CO exposure (MCOHb = 0.78-1.5%), 18 (3.67%) – moderate impact (MCOHb = 1.75-2.23%) and 11 (2.24%) had severe impact (MCOHb = 2.39% and above). Moderate and severe impact were most prominent in women of 25-29 and 35-39 years of age respectively at which they occurred in 11 out of 145 (7.59%) and 7 out of 103 women (6.80%) respectively and the differences at various age groups were statistically significant [ $X^2=23.119$ ,  $p<0.010$ , 95%CI (0.038,0.046)]. The differences in the severity of maternal impact among women with different BMI classes were statistically significant [ $X^2=56.707$ ,  $p<0.001$ , 95%CI (0.001,0.001)] with those with class III BMI most likely to have severe impact [4(22.22%) out of 18 patients]. There was inverse relationship between parity and the severity of the impact of CO exposure but the differences at various parity groups were not statistically significant [ $X^2=10.580$ ,  $p<0.012$ , 95%CI (0.101,0.113)]. There was also a paradoxical finding of 3 smokers having only mild impact. **Conclusion:** The mean value of MCOHb was  $1.15 \pm 0.40$ . Mild, moderate and severe impact from maternal CO exposure was established with the moderate and severe impacts more prominent at maternal ages of 25-39 years, at higher BMI and at lower parity.

**Keywords:** Human biomonitoring, Maternal exposure, Carbon Monoxide, First trimester, Pregnancy, Niger Delta.

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## BACKGROUND

The Niger Delta area in Nigeria is situated in the Gulf of Guinea between longitude 50E to 80E and latitudes 40N to 60N. It is a home to more than 20 million people drawn from nine states namely Rivers, Bayelsa, Akwa Ibom, Cross River, Delta, Abia, Edo, Imo and Ondo States, with the first two States called the 'Core Niger Delta'. The region produces over 90% of

Nigeria's foreign earnings through oil and gas exploration and production activities. It therefore plays host to most of the upstream and downstream oil related and non-oil related industries that release tons of pollutants including CO into the ecosystems.

The environmental terrain in the Delta has led to series of scientific research, the most notable ones of which were the World Bank study of the region and the

Environmental Assessment of Ogoniland in the core Niger Delta which delivered a catalogue of devastation due to oil pollution in the region [1, 2]. Ogoniland which is one of the heavily polluted regions in Rivers State was tagged 'a region of environmental disaster' [2]. Irrespective of the high levels of pollution in the Niger Delta, the guidelines on environmental protection against the pollutants, including CO were not fully implemented.

Generally, sources of CO pollution in the Delta were tobacco fumes, generators, firewood, kerosene, bush and refuse burning, fire outbreaks, barbecues, burning of fossil fuels in old vehicles, crude oil and gas industry (three refineries, oil wells, flow stations and gas flaring, crude oil and condensate spills, vapours from crude and refined oil storage, processing and transportation facilities, petrochemical plants and gas liquefaction plants) [3].

Carbon monoxide (CO) is an inorganic colourless, odourless and non-irritating gas produced from the incomplete combustion of carbonaceous compounds. It is a primary pollutant as it is emitted from a source directly into the atmosphere. It enters into the body primarily through inhalation (though there is also nominal endogenous production of the gas) and diffuses across the alveolar membrane with nearly the same ease as oxygen (O<sub>2</sub>). It dissolves in blood, but is quickly bound to haemoglobin (Hb) to form COHb, which is measured as the percentage of haemoglobin so bound.

The binding of carbon monoxide to haemoglobin occurs with nearly the same speed and ease as with which oxygen binds to haemoglobin, although the bond for carbon monoxide is about 245 times as strong as that for oxygen [4, 5]. Thus, carbon monoxide competes equivocally with oxygen for haemoglobin binding sites but, unlike oxygen, which is quickly and easily dissociated from its haemoglobin bond, carbon monoxide remains bound for a much longer time. Therefore, COHb continues to increase with continued exposure, leaving progressively less haemoglobin available for carrying oxygen. The result is arterial hypoxaemia. Furthermore, COHb increases the binding strength of oxygen to haemoglobin, thus making release of oxygen into tissue more difficult. The latter is called a leftward shift in the oxyhaemoglobin dissociation curve, proportional to the COHb level.

The most important variables in the formation of COHb are the concentration and duration of carbon monoxide in inhaled air and the rate of alveolar ventilation [6]. Alveolar ventilations, largely determined by body energy expenditure (exercise), can vary over a wide range and is thus the major physiological determinant of the rate of COHb formation and elimination. Based on the laboratory studies of reduction in exercise capacity in both healthy

individuals and volunteers with cardiovascular disease, it was determined that COHb levels should not exceed 2%.

Carbon monoxide will also reduce the diffusion of oxygen into tissue via myoglobin by formation of carboxymyoglobin [7, 8]. It is probable that such effects become important only for high levels of carbon monoxide exposure [7]. CO also binds to other proteins (cytochrome P-450 and cytochrome oxidase) but the dosimetry is unclear and the functional significance appears to be limited to high levels of carbon monoxide exposure [9].

CO inhalation was the most common cause of poisoning in the industrialised world and in many countries, CO was the leading cause of the fatal poisoning reported [10]. It can cause multi-organ dysfunction and frequently necessitating admission to intensive care units. In pregnancy, maternal impact of CO pollution ranges from flu-like symptoms, headaches at carboxyhaemoglobin (COHb) levels of 5-20%, gastrointestinal, neurological and psychiatric symptoms to maternal death at COHb levels of >66% [11]. Maternal exposure to the gas during the period of ontogenesis is associated with abnormalities of organs and systems including the central nervous system [11, 13], skeleton and face [12] and the heart [13, 14]. Other toxic effects include intrauterine growth restriction [14], preterm labour [15], intrauterine fetal death [16, 17], and sudden infant death [18].

Irrespective of the presence of millions of sources of CO pollution in the Core Niger Delta, in contrast to what was obtainable in the developed countries of Europe, North America and Asia, organized daily environmental monitoring of its ambient and indoors concentrations and human medical biomonitoring of the gas in suspected acute or chronic poisoning were not practiced. What were available were data from sporadic assessments conducted by researchers in the Universities in the region and some data from environmental impact assessment conducted by multinational companies in their immediate area of operation. Human biomonitoring (HBM) can be defined as "the method for assessing human exposure to chemicals or their effects by measuring the chemicals, their metabolites or reaction products in human specimens." So, the ambient and indoor air concentrations of CO in the Delta ranged from 0 ppm to 191 µg/m<sup>3</sup> but in places within 60-200 metres from crude oil flow stations, the concentrations range from 100 to 5320 µg/m<sup>3</sup> [18].

Therefore, irrespective of the knowledge of the sources of CO production in the Niger Delta and its impact on mother and fetus, there was no data or register on the prevalence of its poisoning and its clinical presentations [19]. There was no data on maternal or paternal inhalation and exhalation of CO

and consequently, no knowledge of maternal and fetal carboxy-hemoglobin and associated health impact, based on their concentrations.

**Aim:** The primary aim of the study therefore was to establish the severity of maternal exposure to CO in the first trimester of pregnancy in the core Niger Delta by quantifying maternal carboxyhemoglobin levels. The secondary goal was to assess the extent of modification of the detrimental impact of CO by maternal demographic and obstetric characteristics, namely age, BMI and parity.

## METHODOLOGY

The study was of cross-sectional design carried out at the Rivers State University Teaching Hospital in Rivers State, which is one of the States in the core Niger Delta area of Nigeria. The study population included all pregnant women attending the antenatal clinics in the first trimester of pregnancy up till 14 weeks from January 2021 to January 2022. Consecutive attendants were counselled about the research project and verbal consents were obtained.

The exclusion criteria were pregnant women with physical disabilities such as deafness and dumbness, critically ill patients, as well as those with a history of ongoing mental illness/retardation (because of the difficulties associated with taking history from the patients), uncertain date of last menstrual period with no ultrasonographic estimation of gestational age between 11-14 weeks of gestational age. Gestational age was estimated with the aid of dating scans in the first trimester of pregnancy. Demographic, social and obstetric characteristics including age, education, drinking and smoking status, BMI and parity were taken.

### Measurement of maternal exhaled carbon monoxide

A hand-held instrument called Smokerlyzer has been used to measure the concentration of CO in expired air especially in smokers [20]. It displays CO in part per million (ppm), concentrations of maternal carboxyhaemoglobin (MCOHb) which is synonymous with the concentration of oxygen that is displaced by CO (%COHb) and fetal carboxyhaemoglobin (fCOHb) which, is synonymous with the percentage of oxygen that is displaced by CO in fetal circulation (%FCOHb)

but in the present study, we were interested in the concentrations of MCOHb.

Clinical research has demonstrated that a useful relationship between carbon monoxide and carboxyhaemoglobin is obtained after a short period of breath-holding. Smokerlyzer only directly measures the exhaled CO concentration while MCOHb and fCOHb are calculations based on clinical evidence. CO ppm-%COHb calculation was taken from Jarvis M *et al.*, (1986) [21].  $COHb = 0.63 + 0.16(EC50)$ , where (EC50) is the concentration of CO in ppm that is expired after inhalation as measured with a smokerlyzer (breath test) [20]. COppm-%FCOHb calculation was taken from Gomez C *et al.*, (2005) [22].

Although women in the core Niger Delta almost do not smoke, they were perpetually exposed to CO because of the presence of several sources of the gas in the Delta. We therefore hypothesised that they were likely to be affected by the gas just as women who smoke were. The smokerlyzer was therefore used to measure the concentration of exhaled CO in the women and indirectly, the concentrations of MCOHb and FCOHb were given by the machine. The severity of maternal exposure to CO was assessed, using the data from table 1a and 1b which came with the smokerlyzer. %COHb in table 1a for maternal COHb concentration was used as reference ranges for comparison.

### Green zone

This is where a mother really needs to be! It means she does not exhale more than 3 ppm of CO in her breath and that corresponds to less than 2% carbon monoxide (CO) in her blood. Most people have a small amount of CO in their breath, this is due to the air quality around them.

### Gray zone

Having a reading in this zone would indicate a light smoker or a non-smoker breathing in poor air quality or passive smoke.

### Red zone

Having a reading in this zone indicates that the person may well be a regular smoker with higher levels of CO in the blood; in the present study, we have extrapolated that to significant environmental pollution since almost all the study population does not smoke.

**Table 1: Derivation of the %COHb and %fCOHb from CO ppm on the basis of clinical evidence [20]**

a		b	
COppm	%COHb	COppm	%fCOHb
30	5.43	20+	5.66
29	5.27	19	5.38
28	5.11	18	5.09
27	4.95	17	4.81
26	4.79	16	4.53
25	4.63	15	4.25
24	4.47	14	3.96
23	4.31	13	3.68
22	4.15	12	3.40
21	3.99	11	3.11
20	3.83	10	2.83
19	3.67	09	2.55
18	3.51	08	2.26
17	3.35	07	1.98
16	3.19	06	1.70
15	3.03	05	1.42
14	2.87	04	1.13
13	2.71	03	0.85
12	2.55	02	0.57
11	2.39	01	0.28
10	2.23		
09	2.07		
08	1.91		
07	1.75		
06	1.59		
05	1.43		
04	1.27		
03	1.11		
02	0.95		
01	0.79		

### Determination of the sample size

The outcome measures in the study were the incidence of different measures of severity of exposure to CO and the modification of the impact by maternal age, parity, BMI and smoking status. Therefore, the sample size was calculated using the sample size formula for a cross-sectional study with a categorical outcome.

$$n = Z_{\alpha/2}^2 P (1-P) / d^2$$

Where

$Z_{\alpha/2}$  = Standard normal deviate at 95% confidence interval = 1.96.

P - Expected proportion in population based on previous studies. Since there were no figures in the past for the assessed parameters in the study, 50% was used in the calculation of the sample size.

d = Absolute error or precision = 0.05.

Therefore,

$$N = 1.96^2 \times 0.5(1-0.5) / 0.05^2 = 3.8416 \times 0.5 \times 0.5 / 0.0025 = 384.16$$

The required number of patients for the study was therefore 384.16. Giving allowance for attrition rate of 10%, the final sample size for the study was  $10/100 \times 384 + 384 = 422.56$ . Therefore, the number of patients to be recruited for the study was 423. We were however able to recruit 490 patients.

### Statistical Analysis

Data was collected on a special pretested proforma and then transferred into an excel file where they were cleaned and fed into SPSS version 25.0 (Armonk, NY) software for analysis. Simple proportions were used in the descriptive analysis. Quantitative data were summarized and presented as mean and standard deviation while qualitative data were presented as numbers and percentages. Comparison of related variables was conducted, using the Chi-square ( $X^2$ ) and the P-values. When the P-value was less than 0.05, the differences between the variables were said to be statistically significant. When an expected count was lower than 5 in a cell, Fisher Exact test was used.

### Ethical Consideration

The study was carried out in compliance with the international ethical guidelines for biomedical research involving human subjects. Ethical approval was obtained from the RSUTH Ethics Committee. Informed consents were obtained from all the women that were enrolled in the study. All the information that was collected from individual patients was available for clinical use and for the research purposes. Privacy rules were maintained and confidentiality was observed at all levels of dealing with patients' data.

## RESULTS

### Demographic, obstetric and general characteristics

Four hundred and ninety (490) pregnant women were recruited for the study from 11-14 weeks of pregnancy. The mean age of the participants in the study was  $31.5682 \pm 4.49$  years. Details of the other parameters namely education, alcohol consumption and cigarette smoking status, parity, BMI and marital status were as shown in Table 1.

**Table 2: Demographic, obstetric and general characteristics of the patients**

Demographic, obstetric and general characteristics		Frequency N =	Percentage. (%)
Maternal age, Years (n=490)	20-24	17	3.47
	25-29	145	29.59
	30-34	212	43.27
	35-39	103	21.02
	40-44	9	1.84
	45-49	4	0.82
Education (n = 490)	secondary	73	14.90
	tertiary	417	85.10
Alcohol (n=488)	no	370	75.82
	yes	118	24.18
	yes	118	24.18
Smoking (n=490)	no	487	99.39
	yes	3	0.61
Parity (n=483)	0	230	47.62
	1 to 2	223	46.17
	3 and above	30	6.21
BMI (n=472)	$\geq 40.0$ ) (Class III Obesity)	18	3.81
	18.5–24.9 (Normal weight)	80	16.95
	25.0–29.9 (Overweight)	218	46.19
	30.0–34.9 (Class I Obesity)	107	22.67
	35.0–39.9 (Class II Obesity)	49	10.38
Marital Status (n=490)	Married	490	100
	Not married	0	0

### Measures of severity of the impact of exposure to Carbon monoxide

The impact of maternal exposure to CO was measured by the mean value of MCOHb which was

$1.15 \pm 0.40\%$  and its actual concentrations as shown in Table 3.

**Table 3: Degrees of severity of exposure to Carbon monoxide, n = 490**

Form of CO with respect to the harboring medium	Degrees of severity	Frequency	Percentages %
Maternal Carboxy-Haemoglobin levels (MCOH).	Mild (0.78 to 1.59%)	461	94.08
	Moderate (1.75 to 2.23 %)	18	3.67
	Severe (2.39% and above)	11	2.2
Total		490	100

### The impact of demographic, obstetric and general characteristics of the patients on the severity of maternal exposure to CO (MCOHb)

#### The effects of maternal age on MCOHb concentrations

The modification of the severity of the impact of maternal exposure by her age was as shown in Table 4.

**Table 4: Relationship between maternal age and MCOHb concentrations**

Maternal Age group Years	No. of patients associated with different degrees of Severity (MCOHb). n%(row)/(col)			Total N(%col)	X <sup>2</sup>	P-value 95%CI
	Mild (0.78 to 1.59%)	Moderate (1.75 to 2.23%)	Severe (2.39 % and above)			
20-24	17(100)/ (3.69)	0(0)/0(0)	0(0)/ (0)	17(3.47)	23.119	0.010* (0.038,0.046)
25-29	134(92.41)/ (29.07)	11(7.59)/ (61.11)	0(0)/ (0)	145(29.59)		
30-34	204(96.23)/ (44.25)	4(1.89)/ (22.22)	4(1.89)/ (36.36)	212(43.27)		
35-39	93(90.29)/ (20.17)	3(2.91)/ (16.67)	7(6.80)/ (63.64)	103(21.02)		
40-44	9(100)/ (1.95)	0(0)/ (0)	0(0)/ (0)	9(1.84)		
45-49	4(100)/ (0.87)	0(0)/ (0)	0(0)/ (0)	4(0.82)		
<b>TOTAL</b>	461(94.08)	18(3.67)	11(2.24)	490(100)		

\*Statistically significant (p&lt;0.05)

**The effects of maternal smoking on MCOHb concentrations**

The severity of maternal exposure to CO was also associated with maternal smoking habit as shown

in table 4 and it was classified into mild, moderate and severe impact on the bases of different levels of maternal MCOHb.

**Table 5: Relationship between maternal smoking and MCOHb concentrations**

Smoking	No. of patients associated with different degrees of Severity (MCOHb). n%(row)/(col)			Total n(Col %)	X <sup>2</sup>	P-value 95%CI
	Mild (0.78 to 1.59%)	Moderate (1.75 to 2.23%)	Severe (2.39 % and above)			
no	458(94.05)/ (99.35)	18(3.70)/ (100.)	11(2.26)/ (100.)	487(99.39)	1.668	1.000 (1.000,1.000)
yes	3(100)/(0.65)	0(0)/0(0)	0(0)/(0)	3(0.61)		
<b>TOTAL</b>	461(94.08)	18(3.67)	11(2.24)	490(100)		

**The effects of maternal BMI on MCOHb concentrations**

The severity of exposure to Co was also assessed with respect to different classes of BMI. The results were as shown in table 6.

**Table 6: Relationships between maternal BMI and CO levels**

BMI Classification	No. of patients associated with different degrees of Severity (MCOHb). n%(row)/(col)			Total n (Col %)	X <sup>2</sup>	P-value 95%CI
	Mild (0.78 to 1.59%)	Moderate (1.75 to 2.23%)	Severe (2.39 % and above)			
18.5–24.9 (Normal weight)	74(92.50)/ (16.63)	6(7.50)/ (37.50)	0(0)/ (0)	80 (16.95)	56.707	0.001* (0.001,0.001)
25.0–29.9 (Overweight)	212(97.25)/ (47.64)	3(1.38)/ (18.75)	3(1.38)/ (27.27)	218 (46.19)		
30.0–34.9 (Class I Obesity)	103(96.26)/(23.15)	4(3.74)/ (25.00)	0(0)/ (0)	107 (22.67)		
35.0–39.9 (Class II Obesity)	42(85.71)/ (9.44)	3(6.12)/ (18.75)	4(8.16)/ (36.36)	49(10.38)		
≥40.0 (Class III Obesity)	14(77.78)/ (3.15)	0(0)/ (0)	4(22.22)/ (36.36)	18(3.81)		
<b>TOTAL</b>	445(94.28)	16(3.39)	11(2.33)	472(100)		

\*Statistically significant (p&lt;0.05)

### The effect of parity on MCOHb concentrations

The severity of maternal impact on exposure to Co was also assessed with respect to parity. The results were as shown in Table 7.

**Table 7: Relationships between Parity and MCOHb levels**

PARITY GRP	No. of patients associated with different degrees of Severity (MCOHb). n%(row)/(col)			Total (Col)	X <sup>2</sup>	P-value (95%CI)
	Mild (0.78 to 1.59%)	Moderate (1.75 to 2.23%)	Severe (2.39 % and above)			
0	214(93.04)/ (47.14)	12(5.22)/ (66.67)	4(1.74)/ (36.36)	230(47.62)	5.490	0.429 (0.419,0.439)
1 to 2	210(94.17)/ (46.26)	6(2.69)/ (33.33)	7(3.14)/ (63.64)	223(46.17)		
3 and above	30(100)/ (6.61)	0(0)/ (0)	0(0)/ (0)	30(6.21)		
<b>TOTAL</b>	454(94.00)	18(3.73)	11(2.28)	483(100)		

## DISCUSSION

The study was prompted by the popular believe that the core Niger Delta area of Nigeria was plagued by environmental pollution. The sustained impact of maternal exposure to CO was measured by the concentration of maternal Carboxyhemoglobin (MCOHb) in her blood. Maternal impact of CO exposure was classified into 3 degrees of severity (based on the levels of MCOHb), namely mild, moderate and severe. The classification was adapted from the hitherto attained norms that came with the instrument Smokerlyzer Bedmont and was based on previous studies [20-22].

The mean age of the participants in the study was 31.5682± 4.49 years. Majority of the patients 357 (72.86%) were in the age category of 25-34 years; that was followed by 103 (21.02%) at 35-39 years of age, indicating that most women had their children in the normal reproductive age limits (Table 2).

The impact of maternal exposure to CO was measured by the mean value of MCOHb concentration which was 1.15±0.40% and its actual concentrations. The mean concentration of MCOHb in the present study falls within the range for mild impact on maternal exposure to CO and also below agreed level in adults which was 2% although no level is completely save in terms of clinical presentation. Based on the laboratory studies of reduction in exercise capacity in both healthy individuals and volunteers with cardiovascular disease, it was determined that COHb levels should not exceed 2%. The Coburn-Forster-Kane (CFK) equation is used to determine the levels of carbon monoxide to which a normal adult under resting conditions for various intervals can be exposed without exceeding a COHb level of 2% [9].

Out of the total 490 patients that were assessed, majority of them 461(94.08%) had mild impact from CO exposure (MCOHb= 0.78-1.5%), 18 (3.67%) – moderate impact (MCOHb = 1.75-2.23%)

and 11 (2.24%) had severe impact (MCOHb = 2.39% and above). The level of the severity of the impact of maternal exposure to CO at different gestational age groups 20-24 to 45-49 years were different from each other and the differences were statistically significant [ $X^2=23.119$ ,  $p<0.010$ , 95%CI (0.038,0.046)].

The least affected were women in the age groups 20-24, 40-44 and 45-49 years, none of who had moderate nor severe impact. Moderate severity of impact was more common in women of 25-29 years of age at which 11 out of 145 women (7.59%) were affected, 35-39 years of age - 3 out of 103 patients (2.91%) and lastly in women of 30-34 years of age at which 4 out of 212 women (1.89%) were affected. Severe exposure was most prominent at 35-39 and 30-34 years-age groups at which it occurred in 7 out of 103 (6.80%) and 4 out of 212 women (1.89%) respectively.

Out of the total 490 patients that were assessed, only 3 (0.61%) of them were smokers and they all had mild impact on exposure to CO as shown by the concentrations of CO in table 4. The differences between the number of patients who had mild, moderate and severe impact that smoke and those that did not smoke were not statistically significant in terms of maternal MCOHB concentrations [ $X^2=1.668$ ;  $p< 1.000$ , 95%CI (1.00 (1.000,1.000)]. Smoking exposes people to a high concentration of CO [24]. In the WHO report, the CO concentration in tobacco smoke was around 4.5% (45,000ppm), and smokers inhale air with a concentration of about 400–500 CO ppm during smoking [25]. Generally, without potential air pollution, the exhaled CO concentration would be expected in a range of 1- 4 ppm in non-smokers and 2–18 ppm in smokers [26]. Their corresponding MCOHb should be very high too.

Contrary to what was expected, the 3 smokers in the present study had mild impact on exposure o CO. Even though Jarvis et al. reported that exhaled CO measurement could distinguish smokers from non-smokers, they mentioned that a few smokers could not

be identified due to the fact that they did not inhale the smoke very deeply [27]. Long period since the last cigarette was taken could also be an explanation for the low exhaled CO in the smokers. The carboxyhemoglobin half-life for a healthy person breathing air is approximately 4 hours [28]. If a person stops smoking for a sufficiently long period, the exhaled CO concentration could be similar to non-smokers. Furthermore, smokers could lower their CO exposure by reducing the puff volume, the puffs smoked and the tendency and depth of inhaling [24, 28-32].

Regarding the relationship between maternal BMI and MCOHb concentrations, out of the total 472 patients that were assessed, 445(94.28%), 16(3.39%) and 11(2.33%) of them had MCOHb concentrations indicating mild (0.78 to 1.59%), moderate (1.75 to 2.23%) and severe impact (2.39% & above) respectively; the difference was statistically significant [ $X^2=56.707$ ,  $p<0.001$ , 95%CI (0.001,0.001)].

MCOHb concentrations of mild and moderate impact were least prominent in Class III Obesity with each found in 14(77.78%) and in 0(0%) respectively out of the 18 patients in that class. Severe impact was least prominent in women with normal BMI [0(0%) out of the 80 women in that class] and in class I obesity [0(0%) out of the 107 patients in that class]. The mild, moderate and severe categories of MCOHb concentrations were most prominent in women who were overweight [212(97.25%) out of the 218 patients in that class], in those who have normal weight [6(7.50%) out of 80 patients in that class] and in class III obesity [4(22.22%) out of 18] respectively. The highest maternal concentration of COHb was noticed in women with class III followed by women in the class II obesity [4(8.16) out of the 49 patients in that group.

Regarding the relationship between maternal parity and severity of maternal COHb concentrations, out of the total 483 patients that were assessed, 454(94.00), 18(3.73) and 11(2.28) of them had mild, moderate and severe impact and the differences between different groups of severity at different parity groups were not statistically significant [ $X^2=5.490$ ,  $p<0.429$ , 95%CI 0.429(0.419,0.439)].

MCOHb concentration of mild severity was least prominent in Para 0 [214(93.04%) out of 230], while those of moderate and severe categories were least found in para 3 and above - 0(0%) in each category out of the total 30 patients in that parity group. Mild, moderate and severe impact were most prominent at para 3 and above [30(100%) out of 30], Para 0 [12(5.22%) out of 230 patients] and Para 1-2 [7(3.14%) out of 223 patients] respectively.

## CONCLUSION

The mean value of MCOHb  $1.15\pm 0.40\%$  in the present study fell within the range for mild impact on

maternal exposure to CO and it was lower than that which was expected in an adult. Out of the total 490 patients that were assessed, majority of the women 461(94.08%) had mild impact from CO exposure, 18 (3.67%) – moderate impact and 11 (2.24%) had severe impact. The differences between the severity of the impact vary with maternal age and BMI and the differences were statistically significant with severe impact registered at 35-39 and 30-34 years of age and in women in class III obesity. There was inverse relationship between parity and the severity of the impact on CO exposure but the differences between various parity groups were not statistically significant. There was also a paradoxical finding of 3 smokers having only mild impact.

## RECOMMENDATIONS

There is need for the introduction of a unified Niger Delta air quality assessment and also a universal human biomonitoring of the impact of CO on mothers in the Niger Delta. That will go a long way identifying those regions that are worse affected and women that are most at risk of the exposure.

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## REFERENCES

1. World Bank Defining an Environmental Development Strategy for the Niger Delta. Vol. 1, Industry and Energy Operations, West Central Africa Department. 1995.
2. UNEP Environmental Assessment of Ogoniland. United Nations Environmental. 2011
3. Green, K. I., & Abbey, M. (2022). Sources of Carbon Monoxide (CO) Pollution in the Niger Delta area of Nigeria. *Saudi J Biomed Res*, 7(2), 107-113.
4. Joumard, R., Chiron, M., Vidon, R., Maurin, M., & Rouzioux, J. M. (1981). Mathematical models of the uptake of carbon monoxide on hemoglobin at low carbon monoxide levels. *Environmental Health Perspectives*, 41, 277-289.
5. Roughton, F. J. W. (1970). The equilibrium of carbon monoxide with human hemoglobin in whole blood. *Annals of the New York Academy of Sciences*, 174(1), 177-188.
6. McCARTNEY, M. L. (1990). Sensitivity analysis applied to Coburn-Forster-Kane models of carboxyhemoglobin formation. *American Industrial Hygiene Association Journal*, 51(3), 169-177.
7. Bruce, E. N., & Bruce, M. C. (2003). A multicompartiment model of carboxyhemoglobin and carboxymyoglobin responses to inhalation of



- carbon monoxide. *Journal of Applied Physiology*, 95(3), 1235-1247.
8. Bruce, E. N., Bruce, M. C., & Erupaka, K. (2008). Prediction of the rate of uptake of carbon monoxide from blood by extravascular tissues. *Respiratory physiology & neurobiology*, 161(2), 142-159.
  9. Raub, J. A., & Benignus, V. A. (2002). Carbon monoxide and the nervous system. *Neuroscience & Biobehavioral Reviews*, 26(8), 925-940.
  10. Raub, J. A., Mathieu-Nolf, M., Hampson, N. B., & Thom, S. R. (2000). Carbon monoxide poisoning—a public health perspective. *Toxicology*, 145(1), 1-14.
  11. Marzella, L., & Myers, R. A. M. (1985). Carbon monoxide poisoning. *Am Fam Physician*, 34, 186-194
  12. Bailey, L. T. J., Johnston, M. C., & Billet, J. (1995). Effects of carbon monoxide and hypoxia on cleft lip in A/J mice. *The Cleft palate-craniofacial journal*, 32(1), 14-19.
  13. Osborne, J. S., Adamek, S., & Hobbs, M. E. (1956). Some components of gas phase of cigarette smoke. *Analytical Chemistry*, 28(2), 211-215.
  14. Dalhamn, T., Edfors, M. L., & Rylander, R. (1968). Retention of cigarette smoke components in human lungs. *Archives of Environmental Health: An International Journal*, 17(5), 746-748.
  15. Silverman, R. K., & Montano, J. (1997). Hyperbaric oxygen treatment during pregnancy in acute carbon monoxide poisoning. A case report. *The Journal of Reproductive Medicine*, 42(5), 309-311.
  16. Cramer, C. R. (1982). Fetal death due to accidental maternal carbon monoxide poisoning. *Journal of Toxicology: Clinical Toxicology*, 19(3), 297-301.
  17. Farrow, J. R., Davis, G. J., Roy, T. M., McCloud, L. C., & Nichols, G. R. (1990). Fetal death due to nonlethal maternal carbon monoxide poisoning. *Journal of Forensic Science*, 35(6), 1448-1452.
  18. Hutter, C. D. D., & Blair, M. E. (1996). Carbon monoxide—does fetal exposure cause sudden infant death syndrome?. *Medical hypotheses*, 46(1), 1-4.
  19. Abbey, M., Adebari, O. O., Green, K. I., & Chinko, B. C. (2022). Carbon Monoxide (CO) Pollution in the Niger Delta area of Nigeria and Its Impact on Foeto-Maternal Health. *Sch Int J Obstet Gynec*, 5(2), 57-64.
  20. Smokerlyzer. Bedford [www.bedfont.com](http://www.bedfont.com). 1996.
  21. Jarvis, M. J., Belcher, M., Vesey, C., & Hutchison, D. C. (1986). Low cost carbon monoxide monitors in smoking assessment. *Thorax*, 41(11), 886-887.
  22. Gomez, C., Berlin, I., Marquis, P., & Delcroix, M. (2005). Expired air carbon monoxide concentration in mothers and their spouses above 5 ppm is associated with decreased fetal growth. *Preventive Medicine*, 40(1), 10-15.
  23. Coburn, R. F., Forster, R. E., & Kane, P. B. (1965). Considerations of the physiological variables that determine the blood carboxyhemoglobin concentration in man. *The Journal of clinical investigation*, 44(11), 1899-1910.
  24. Robinson, J. C., & Forbes, W. F. (1975). The role of carbon monoxide in cigarette smoking: I carbon monoxide yield from cigarettes. *Archives of Environmental Health: An International Journal*, 30(9), 425-434.
  25. Raub, J. (1999). Environmental Health Criteria 213: Carbon Monoxide; World Health Organization: Geneva, Switzerland, 2, 1-19.
  26. Maga, M., Janik, M. K., Wachsmann, A., Chrząstek-Janik, O., Koziej, M., Bajkowski, M., ... & Nizankowski, R. (2017). Influence of air pollution on exhaled carbon monoxide levels in smokers and non-smokers. A prospective cross-sectional study. *Environmental Research*, 152, 496-502.
  27. Pan, K. T., Leonardi, G. S., Ucci, M., & Croxford, B. (2021). Can exhaled carbon monoxide be used as a marker of exposure? A cross-sectional study in young adults. *International journal of environmental research and public health*, 18(22), 11893.
  28. Kao, L. W., & Nañagas, K. A. (2004). Carbon monoxide poisoning. *Emergency Medicine Clinics*, 22(4), 985-1018.
  29. Vogt, T. M., Selvin, S., & Hulley, S. B. (1979). Comparison of biochemical and questionnaire estimates of tobacco exposure. *Preventive Medicine*, 8(1), 23-33.
  30. Weinhold, L. L., & Stitzer, M. L. (1989). Effects of puff number and puff spacing on carbon monoxide exposure from commercial brand cigarettes. *Pharmacology Biochemistry and Behavior*, 33(4), 853-858.
  31. Strasser, A. A., Lerman, C., Sanborn, P. M., Pickworth, W. B., & Feldman, E. A. (2007). New lower nicotine cigarettes can produce compensatory smoking and increased carbon monoxide exposure. *Drug and alcohol dependence*, 86(2-3), 294-300.
  32. Muhammad-Kah, R., Liang, Q., Frost-Pineda, K., Mendes, P. E., Roethig, H. J., & Sarkar, M. (2011). Factors affecting exposure to nicotine and carbon monoxide in adult cigarette smokers. *Regulatory Toxicology and Pharmacology*, 61(1), 129-136.