OPEN ACCESS
Scholars International Journal of Obstetrics and Gynecology
Abbreviated Key Title: Sch Int J Obstet Gynec
ISSN 2616-8235 (Print) |ISSN 2617-3492 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com

Original Research Article

Biomonitoring of Fetal exposure to Carbon Monoxide in the First Trimester of Pregnancy in the Core Niger Delta

Abbey, M[1*], Amadi, S. C[1], Mba, A. G[1,2], Ocheche, U. S[1, 2], Kwosah, N. J[1], Altraide, B. O[1], Sapira-Ordu, L[1], Nonye-Enyidah, E[1], Akani, C. I[3]

[1]Rivers State University Teaching Hospital, Port Harcourt, Rivers State, Nigeria
[2]PAMO University of Medical Sciences, Port Harcourt, Rivers State, Nigeria
[3]University of Port Harcourt Teaching hospital, Port Harcourt, Rivers State, Nigeria

DOI: 10.36348/sijog.2022.v05i05.007 | **Received:** 14.04.2022 | **Accepted:** 25.05.2022 | **Published:** 29.05.2022

***Corresponding author:** Abbey, M
Rivers State University Teaching Hospital, Port Harcourt, Rivers State, Nigeria

Abstract

Background: Human fetal medical biomonitoring, including that of fetal carboxyhemoglobin (fCOHb) had never been performed in the Niger Delta area of Nigeria irrespective of the devastating environmental pollution in the region. ***Aim:*** The goals of the study were to establish the severity of fetal impact on maternal exposure to CO in the first trimester of pregnancy in the Niger Delta by quantifying the levels of fetal fCOHb and to assess the impact of maternal demographic, social and obstetric characteristics on it. ***Material and methods*:** A cross-sectional study which was 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. fCOHb 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 fCOHb concentration ± SD was 0.93±0.72%. There were statistically significant differences in its values in different age categories (t=2.742, p<0.019), educational levels (t=2.328, p<0.020), BMI (t=5.545, p<0.0001), gravidity (t=6.447, p<0.0001) and parity (t=2.833, p<0.038). Paradoxically, the differences in its levels in the groups namely, smoking habits and drinking status, were not statistically significant. Out of the total 490 participants, 331(67.60%) had fetuses with mild impact from CO exposure (fCOHb= 0.28 to 0.85%), 125 (25.51%) – moderate impact (fCOHb = >0.85 to 1.70%) and 34 (6.90%) had severe impact (fCOHb = >1.70% and above). There were statistically significant differences in the severity of fetal impact in association with different maternal characteristics namely age groups (X^2=26.687, p<0.003), maternal alcohol intake (X2=18.866, p<0.0001), gravidity (X2=37.819, p<0.0001), parity (X2=12.098, p< 0.047) and BMI (x2=19.409; p<0.013) but there was no pattern in the directions of the differences except for BMI which showed significant positive correlation (r=0.214, p=0.0001) due mainly to maternal weight (r=0.181, p<0.0001). There was a paradoxical finding of 3 smokers having only mild impact. ***Conclusion*:** The mean value of fCOHb concentration ± SD 0.93±0.72% and the 3 degrees of fetal impact on exposure to CO were significantly affected by demographic, social and obstetric characteristics but there was no pattern in their effect except for BMI where a positive correlation was established.

Keywords: Biomonitoring, Fetal exposure, Carbon Monoxide, First Trimester, Pregnancy, Core Niger Delta.

BACKGROUND

The Niger Delta area in Nigeria is situated in the Gulf of Guinea between longitude 50E to 80E and latitude 40N to 60N (Figure 1). 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, Ondo and Rivers 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 ecosystem.

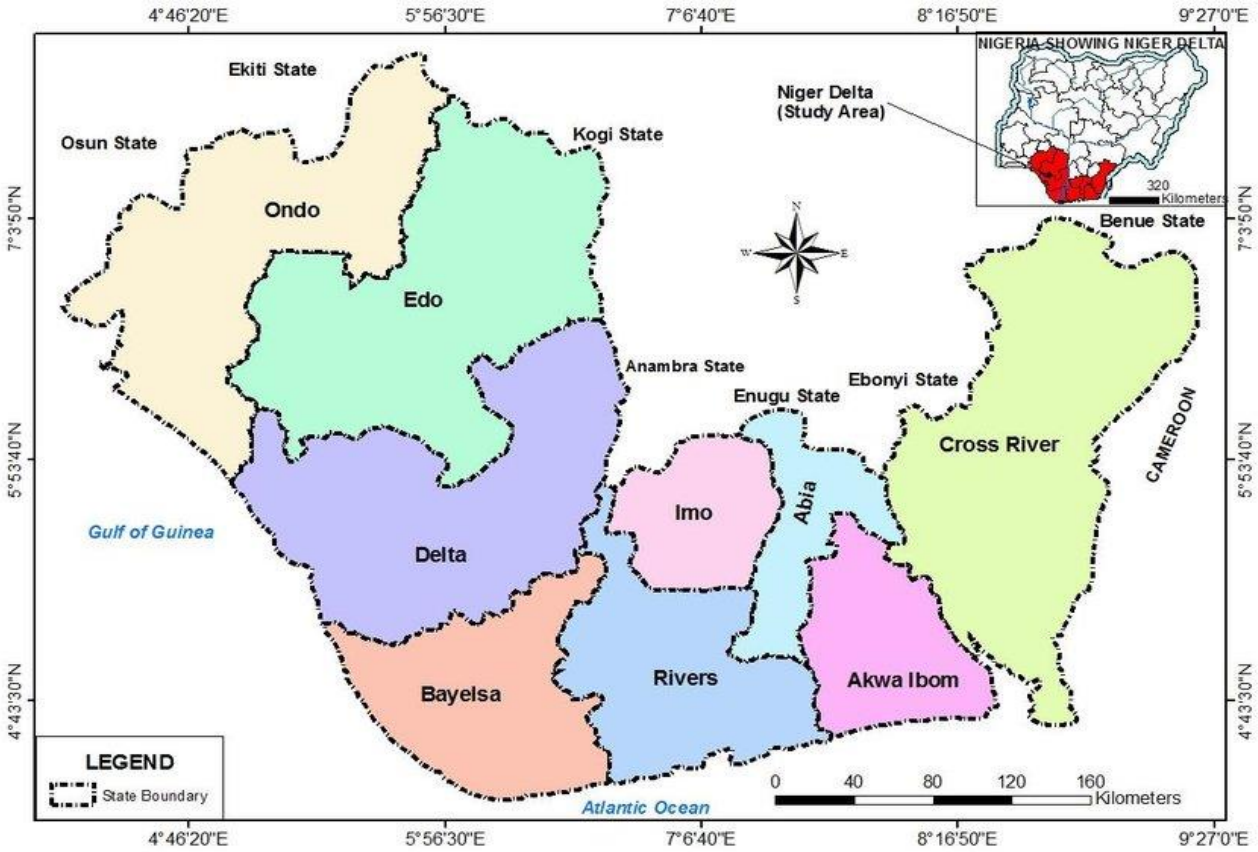

Figure 1: The Niger Delta

The environmental terrain in the Delta 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 area in Rivers State was tagged 'a region of environmental disaster [2].

Sources of CO pollution in the Delta were tobacco fumes, generator, 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. CO inhalation is the most common cause of poisoning in the industrialised world. It can cause multi-organ dysfunction and frequently necessitating admission to intensive care units.

In pregnancy it presents with signs and symptoms of chronic poisoning, namely, headaches, impaired physical performance, sensation of weakness, dizziness, sleepiness, visual difficulties, palpitations, nausea, vomiting at MCOHb concentrations of 5-20%. Acute poisoning presents with tachypnoea, respiratory insufficiency, tachycardia, hypotension, arrhythmia fever, confusion, disorientation, alteration in consciousness, convulsions, vomiting at MCOHb concentrations of 30-50%. Life-threatening poisoning occur at C0Hb levels of 50-66% with psychiatric problem, alteration in consciousness, apathy, apraxia, disorientation, muscular hypertonia, intestinal problems, urinary or faecal incontinence while lethal poisoning occurs at MCOHb levels >66 [4].

The toxic effects of carbon monoxide vary according to the gestational age at which the poisoning occurs. Chronic exposure to CO during the first two trimesters of pregnancy can produce significant intrauterine growth restriction [5, 6], presumably due to chronic hypoxia. CO poisoning potentiates oxygen deficiency, and intrauterine growth restriction can be very severe. Other toxic effects include preterm labour [7], intrauterine fetal death [8, 9] and sudden infant

death [10]. Fetal death can occur in the absence of severe maternal symptoms. During acute severe intoxication the fetus may die of anoxia because maternal haemoglobin is saturated with carbon monoxide. The death by anoxia occurs well before the levels of fetal carboxyhaemoglobin have had time to rise [11]. Fetal CO poisoning can be diagnosed retrospectively by measuring fCOHb concentrations post-mortem and it remains unchanged for several days after death.

Maternal exposure to CO during organogenesis is associated with formation of significant congenital abnormalities. They are as follows: Neurological defects - telencephalic dysgenesis (heterotopia, pachygyria, schisencephalia) [12], behavioural difficulties during infancy [13], skeletal effects namely hand and foot malformations, hip dysplasia and sublaxation, agenesis of a limb, inferior maxillary atresia with glossoptosis [14], and Cleft palate. Maternal exposure during the fetal period is associated with anoxic encephalopathy [15], while in the 3rd month of pregnancy, cardiac defects, including ventricular septal defects (VSD), right-sided cardiomegaly (caused by myosites hyperplasia) may occur [6, 16].

Unfortunately, in contrast to what happens in the developed world of Europe, part of Asia and North America, environmental and human biomonitoring are not practiced in Nigeria [3]. Therefore, there was paucity of information on the levels of fCOHb at which fetal complications occur. There was no data on the prevalence and clinical presentations of CO pollution in the Delta [17]. There was also no register of its poisoning. There were guidelines on environmental protection against pollutants in the region but unfortunately, they were not adhered to. In the case of CO, there was no knowledge of standardized data on its ambient and indoor concentrations. However, there were sporadic data on environmental biomonitoring in areas of interest, conducted by multinational companies and university research Fellows.

Aim

The primary aim of the study therefore was to establish the severity of fetal impact on maternal exposure to CO in the first trimester of pregnancy in the Niger Delta by quantifying the levels of fetal fCOHb. The secondary goal was to assess the impact of maternal demographic, social and obstetric characteristics on the fetal impact.

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 women 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 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 fetal Carboxy-Haemoglobin (fCOHb)

A hand-held instrument called Smokerlyzer has been used to measure the concentration of CO in expired air especially in smokers [18]. It displays CO ppm, %COHb and %fCOHb. Clinical research has demonstrated that a useful relationship between carbon monoxide and carboxyhaemoglobin is obtained after a short period of breath-holding. The device only directly measures the first parameter while the second and the third are calculations based on clinical evidence. COppm - %FCOHb calculation was taken from: Gomez C. et al (2005) [19].

Although women in the core Niger Delta almost do not smoke, they are 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 are affected. The smokerlyzer was therefore used to measure the concentrations of CO exhaled by the women and indirectly, the concentrations of fCOHb were given by the machine.

Table 1: Derivation of the %COHb and %fCOHb from CO ppm on the basis of clinical evidence 18]

a

COppm	%COHb
30	5.43
29	5.27
28	5.11
27	4.95
26	4.79
25	4.63
24	4.47
23	4.31
22	4.15
21	3.99
20	3.83
19	3.67
18	3.51
17	3.35
16	3.19
15	3.03
14	2.87
13	2.71
12	2.55
11	2.39
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

b

COppm	%FCOHb
20+	5.66
19	5.38
18	5.09
17	4.81
16	4.53
15	4.25
14	3.96
13	3.68
12	3.40
11	3.11
10	2.83
09	2.55
08	2.26
07	1.98
06	1.70
05	1.42
04	1.13
03	0.85
02	0.57
01	0.28

The severity of fetal exposure to CO was assessed, using the data from table 1a and 1b which came with the smokerlyzer. %fCOHb in table 1b was used as the 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 air of poor quality or passive smoking.

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

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 characteristic. Therefore, the sample size was calculated using the sample size formula for a cross-sectional study with a categorical outcome.

$n = Z\alpha/22\ P\ (1-P) / d2$

Where

$Z\alpha/22$ = 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,

$N = 1.962x0.5(1-0.5)/0.052 = 3.8416x0.5x0.5/0.0025 = 384.16$

The required number of patients for the study was therefore 384.16. Giving allowance for attrition rate of 10%, the final power for the study was 10/100×384+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 then 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 (X2), t-test 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. Verbal consents were obtained from all women 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 11-13^{+6} weeks of pregnancy. The mean age of study population ± SD = 31.57±4.49 years; median age = 32 years and age range = 21 – 50years. Majority of the patients were in the age category 25-34 years – 348 (71.02%) out of the total 490 patients, 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 1). Details of the other parameters were as shown in table and Figure 1.

Table 2: Demographic, obstetric and general characteristics of the patients, n=490

Demographic obstetric and general characteristics		Frequency (n)	Percentage (%)
Maternal age, Years	20 – 24 years	24	4.9
	25 – 29 years	133	27.1
	30 – 34 years	215	43.9
	35 – 39 years	103	21.0
	40 – 44 years	11	2.2
	≥45 years	4	0.8
Education	secondary	73	14.90
	tertiary	417	85.10
Smoking	no	487	99.39
	yes	3	0.61
Alcohol	No	372	75.9
	Yes	118	24.1
Gravida	G1	246	50.2
	G2	108	22.0
	G3	88	18.0
	>G3	48	9.8
Parity	Para 0	230	46.9
	Para 1	128	26.1
	Para 2 – 4	128	26.1
	≥ Para 5	4	0.8
BMI	18.5–24.9 (Normal weight)	101	20.60
	25.0–29.9 (Overweight)	212	43.30
	30.0–34.9 (Class I Obesity)	107	21.84
	35.0–39.9 (Class II Obesity)	51	10.61
	≥40.0) (Class III Obesity)	18	3.67
Marital Status	Married	490	100
	Not married	0	0

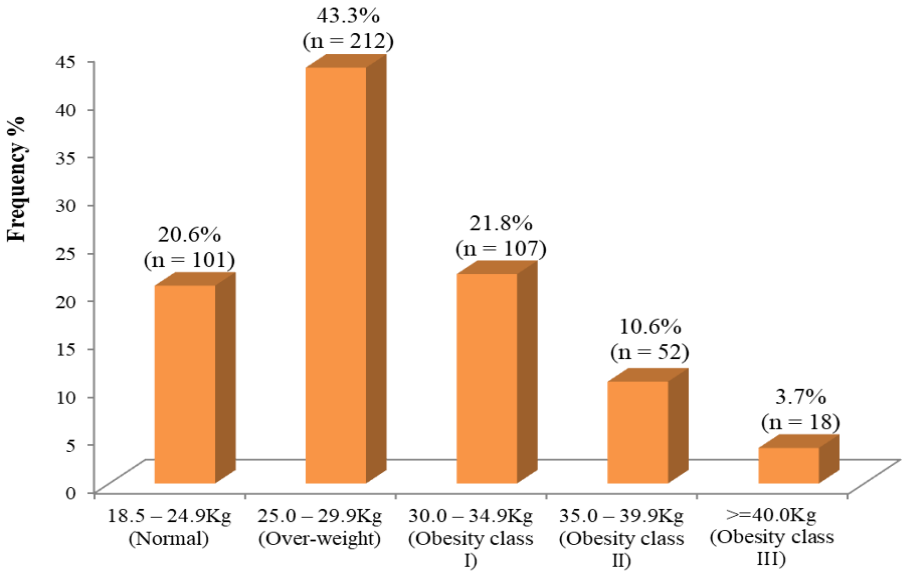

Figure 2: BMI classification of patients

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

The impact of fetal exposure to CO was measured by the mean value of fCOHb concentration on its own and in association with different maternal characteristics and by the classification of its concentrations into 3 degrees of severity [15]. The modifications of the impact by groups of maternal variables were also assessed.

Mean values of fetal COHb concentration on its own and in association with maternal characteristics

The mean value of fCOHb concentration out of the 490 participants in the study ± SD = 0.93±0.72%; Median = 0.85%; Range = 0.28 – 5.38%.

The mean values of fCOHb concentrations in association with the demographic, social and obstetric characteristics of the patients were as shown in table 3. The table showed statistically significant differences in the levels of fCOHb in association with the following maternal characteristics: age, educational levels, BMI, gravidity and parity but the differences in its levels were not statistically significant among smokers and those that take alcohol.

Table 3: The mean values of fCOHb in association with different maternal characteristics

Variables	Fetal COHb (%) Mean ± SD	t	p-value
Age category			
20 – 24 years	0.72±0.32		
25 – 29 years	0.91±0.60		
30 – 34 years	0.89±0.61	*2.742* [A]	*0.019**
35 – 39 years	1.12±1.07		
40 – 44 years	0.53±0.13		
≥45 years	0.61±0.05		
Educational level			
Secondary	1.11±1.00	*2.328*	*0.020**
Tertiary	0.89±0.65		
Intake of alcohol			
Yes	1.01±0.70	*1.433*	*0.153*
No	0.90±0.73		
Smoking history			
Yes	0.57±0.00	*-0.860*	*0.390*
No	0.93±0.72		
BMI			
18.50 – 24.9 (Normal)	0.86±0.53		
25.0 – 29.9 (Over-weight)	0.90±0.72		

30.0 – 34. (Obesity class I)	0.84±0.50	*5.545*	*0.0001**
35.0 – 39.9(Obesity class II)	1.14±0.94		
≥40Kg (Obesity class III)	1.56±1.38		
Gravidity			
G1	0.88±0.64		
G2	0.78±0.37	*6.447*^{A}	*0.0001**
G3	1.06±0.59		
>G3	1.25±1.43		
Parity			
Para 0	0.89±0.66		
Para 1	0.87±0.79	*2.833*^{A}	*0.038**
Para 2 – 4	1.05±0.74		
≥ Para 5	0.28±0.00		

*Statistically significant ($p<0.05$) SD – Standard deviation A - Anova

The 3 degrees of severity of the impact of fetal exposure to CO

The smokerlyzer, during the exhalation test, measured exhaled CO in ppm. fCOHb concentration in % of the displaced oxygen from maternal oxyhaemoglobin was deduced from the exhaled CO, using Gomes C *et al*., calculation [15]. The severity of the impact was then classified into 3 degrees, based on the levels of fCOHb; they were as follows: mild, moderate and severe with fCOHb = 0.78 to 1.59%, 1.75 to 2.23% and 2.39% and above respectively. Since there were gaps in the % of fCOHb between the 3 degrees of severity of impact, their values were modified into those shown in table 4 and figure with a view of enhancing easy comparison.

Out of the total 490 participants, more than half 331(67.60%) had fetuses with mild impact from CO exposure (fCOHb= 0.28 to 0.85%), 125 (25.51%) – moderate impact (fCOHb = >0.85 to 1.70%) and 34 (6.90%) had severe impact (fCOHb = >1.70% and above).

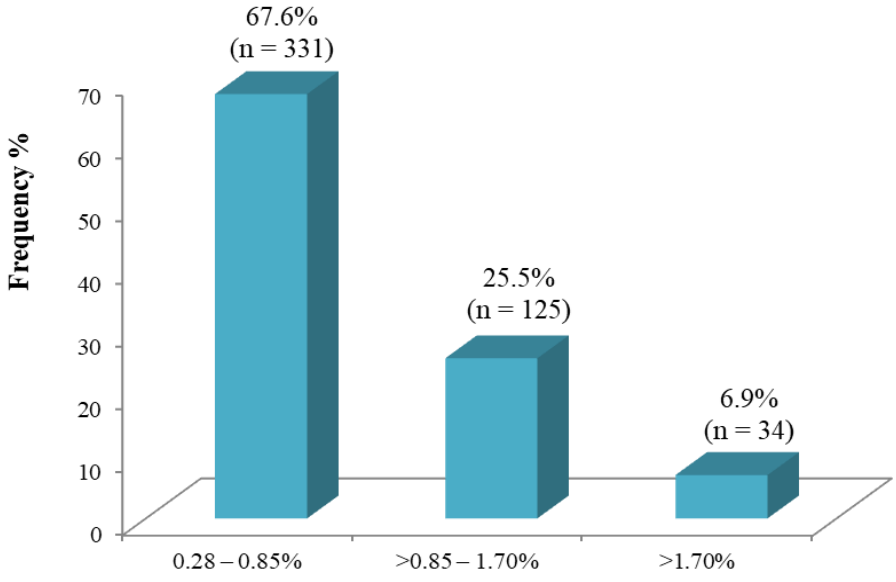

Figure 3 degrees of the severity of exposure to Carbon monoxide

Table 4: Classification of the participants into the 3 degrees of severity of exposure to Carbon monoxide, n = 490

Severity of imapct	fCOHb. (%)	Frequency	Percentages %
Mild	0.28 to 0.85	331	67.60
3	>0.85 to 1.70	125	25.51
Severe	>1.70	34	6.90

The effects of demographic and social characteristics of the patients on the severity of fetal exposure to CO

The severity of fetal impact on maternal exposure to CO was assessed with respect to age categories of the participants, educational levels, intake of alcohol, smoking history and BMI. The results were as shown in Table 5.

Table 5: The effects of demographic and social characteristics of the patient on the severity of fetal exposure to CO

	Severity of impact (Levels of fCOHb)					
Variables	**Mild 0.28 – 0.85% n (%)**	**Moderate >0.85 – 1.70% n (%)**	**Severe >1.70% n (%))**	**Total n (%)**	**Chi Square**	**p-value**
Age category						
20 – 24 years	21 (87.5)	3 (12.5)	0 (0.0)	24 (100.0)		
25 – 29 years	91 (68.4)	26 (19.5)	16 (12.0)	133 (100.0))		
30 – 34 years	142 (66.0)	65 (30.2)	8 (3.7)	215 (100.0))	26.687^{F}	0.003*
35 – 39 years	62 (60.2)	31 (30.1)	10 (9.)	103 (100.0)		
40 – 44 years	11 (100.0)	0 (0.0)	0 (0.0)	11 (100.0))		
≥45 years	4 (100.0)	0 (0.0)	0 (0.0)	4 (100.0)		
Educational level						
Secondary	48 (65.8)	18 (24.7)	7 (9.6)	73 (100.0)	0.934	0.627
Tertiary	283 (67.9)	107 (25.7)	27 (6.5)	417 (100.0)		
Intake of alcohol						
Yes	64 (54.2)	48 (40.7)	6 (5.1)	118 (100.0)	18.866	0.0001
No	267 (71.8)	77 (20.7)	28 (7.5)	372 (100.0)		
Smoking history						
Yes	3 (100.0)	0 (0.0)	0 (0.0)	3 (100.0)	0.925^{F}	0.650
No	328 (67.4)	126 (25.7)	34 (7.0)	487 (100.0)		
BMI						
18.50 – 24.9 (Normal)	69 (68.3)	22 (21.8)	10 (9.9)	101 (100.0)		
25.0 – 29.9 (Overwt.)	143 (67.5)	60 (28.3)	9 (4.2)	212 (100.0)		
30.0 – 34.9(Obesity class I)	80 (74.8)	23 (21.5)	4 (3.7)	107 (100.0)	19.409^{F}	0.013*
35.0 –39.9 (Obesity class II)	31 (59.6)	14 (26.9)	7 (13.5)	52 (100.0)		
≥40 (Obesity class III)	8 (44.4)	6 (33.3)	4 (22.2)	18 (100.0)		

***Statistically significant (*$p<0.05$*)** ***F* – Fisher's exact**

The effect of BMI on fetal response to maternal exposure to CO was unique. Generally, the differences in the severity of fetal effect on exposure to CO were statistically significant (x2=19.409; p<0.013). Furthermore, a significant positive correlation was established between the severity of fetal impact and maternal BMI – the higher the BMI, the more the likelihood of having severe fetal impact (r=0.214, p=0.0001). The correlation was due mainly to maternal weight (r=0.181, p<0.0001) as shown in Table 6.

Table 6: Correlation between fetal COHb concentration and BMI

	Fetal COHb concentration (%)	
Variable	**Pearson Correlation co-efficient (r)**	**p-value**
Maternal weight at booking	0.181	0.0001*
Maternal height at booking	-0.066	0.149
BMI	0.214	0.0001*

Statistically significant ($p<0.05$*)

The impact of groups of obstetric characteristics on the severity of fetal exposure to CO

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

Table 7: The effects of obstetric characteristics on the impact of fetal exposure to CO

Level of fetal COHb						
Variables	**0.28 – 0.85% n (%)**	**>0.85 – 1.70% n (%)**	**>1.70% n (%))**	**Total n (%)**	**Chi Square**	**p-value**
Gravidity						
G1	174 (70.7)	54 (22.0)	18 (7.3)	246 (100.0)		
G2	79 (73.1)	29 (26.9)	0 (0.0)	108 (100.0)	37.819	0.0001*
G3	41 (46.6)	38 (43.2)	9 (10.2)	88 (100.0)		
>G3	37 (77.1)	4 (8.3)	7 (14.6)	48 (100.0)		
Parity						
Para 0	158 (68.7)	54 (23.5)	18 (7.8)	230 (100.0)		
Para 1	94 (73.4)	31 (24.2)	3 (2.3)	128 (100.0)	12.098 *F*	0.047*
Para 2 – 4	75 (58.6)	40 (31.2)	13 (10.2)	128 (100.0)		
≥ Para 5	4 (100.0)	0 (0.0)	0 (0.0)	4 (100.0)		

*Statistically significant (*p<0.05*) *F* – Fisher's exact

DISCUSSION

The mean age of the study population ± SD was 31.57±4.49 years. Majority of the patients were in the age categories 25-34 years – 348 (71.02%) out of the total 490 patients, followed by 103 (21.02%) at 35-39 years of age, indicating that most women had their children in the normal reproductive age limits.

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 fetal exposure to CO was measured by the mean value of fCOHb concentrations, by the 3 degrees of severity of fetal impact and by their associations with maternal, social and obstetric characteristics.

The mean value of fCOHb concentration out of the 490 participants in the study population ± SD was 0.93±0.72%. There were statistically significant differences in its values in different groups of maternal demographic, social and obstetric characteristics namely age categories (t=2.742, p<0.019), educational levels (t=2.328, p<0.020), BMI (t=5.545, p<0.0001), gravidity (t=6.447, p<0.0001) and parity (t=2.833, p<0.038). Paradoxically, the differences in its levels among other groups namely smoking (t= -0.860, p<0.390) and drinking habits (t=1.433, p<0.153) were not statistically significant but there was a negative correlations or reverse relationships between those that smoke and the mean value of fCOHb.

Out of the total 490 participants, more than half 331(67.60%) had fetuses with mild impact from CO exposure (fCOHb= 0.28 to 0.85%), 125 (25.51%) – moderate impact (fCOHb = >0.85 to 1.70%) and 34 (6.90%) had severe impact (fCOHb = >1.70% and above). The classification of the fetal impact on exposure to CO was adapted from the hitherto attained norms that came with the instrument Smokerlyzer Bedmont and was based on previous studies. [18, 19, 20]

The severity of fetal effect on maternal exposure to CO, at different gestational age groups 20-24 to ≥45 years varies from each other and the differences were statistically significant (X^2=26.687, p<0.003). The least affected were women in the age groups 40-44 and ≥45 years, none of whom had moderate nor severe impact. That was followed by women in the age category 20-24 - none had severe impact and only 3 (12.5%) had moderate impact. Moderate severity of impact was most prominent in women of 30-34 years of age at which 65 (30.2%) out of 215 women were affected and in women of 35-39 years of age at which 31(30.1%) out of 103 women were affected. Severe exposure was most prominent in women of 25-29 and 35-39 years-age groups at which it occurred in 16 (12.0%) out of 133 and 10(9%) out of 103 women respectively.

Regarding the relation between maternal alcohol intake and smoking habits and fetal response to CO exposure, the responses were paradoxical. Firstly, women who did not drink had more cases of severe impact [28 (7.5%) out of 372] than those who drink [6 (5.1%) out of 118] and the differences in fetal susceptibility generally to CO in both groups (drinkers and non-drinkers) were statistically significant (X2=18.866, p<0.0001).

Secondly, the only 3 smokers among the participants had mild impact while 34(7%) out of 487 non-smokers had severe impact but the differences were not statistically significant (X2=0.925, p=0.650).

Smoking exposes people to a high concentration of CO. [21] 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 [22]. Generally, without potential air pollution, the exhaled CO concentration would be expected to be in the range of 1–4 ppm in non-smokers and 2–18 ppm in smokers [23]. Their corresponding MCOHb and fCOHb should be proportional.

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. [24] 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. [25] 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 [21, 25-29].

No statistically significant differences were noted between the two educational groups, namely those that finished secondary and tertiary institution respectively (Table 5). Body mass index also affected fetal response to CO exposure. Severe fetal impact was most prominent in women with BMI ≥40 (Obesity class III) and 35.0 –39.9 (Obesity class II) at which they occurred in 4(22.2%) out of 18 and 7(13.5%) pot of 52 fetuses respectively. Moderate impact was most prominent in women with BMI ≥40 (Obesity class III). 25.0 – 29.9 (Overweight) and 35.0 –39.9 (Obesity class II) at which they occurred in 6(33.3%) out of 18, 60(28.3%) out of 212 and 14(26.9%) out of 52 participants respectively. Mild impact was most prominent in women with normal BMI, overweight and those with obesity class I (Table 5).

Generally, the differences in the severity of fetal effect on exposure to CO were statistically significant (x2=19.409; p<0.013). A significant positive correlation was established between the severity of fetal impact and maternal BMI – the higher the BMI, the more the likelihood of having severe fetal impact (r=0.214, p=0.0001). The correlation was due mainly to maternal weight (r=0.181, p<0.0001) as shown in Table 6.

Regarding the relationship between the severity of fetal impact and gravidity, severe fetal impact was most prominent in women who were Gravida 3 and in those who were >G3 and they occurred in 7(14.6%) out of 48 and 9(10.2%) out of 88 fetuses respectively. Moderate impact was most prominent in Gravida 3, G2 and G1, in descending order. Mild impact was also prominent in lower gravidity (Table 7) and the differences in the fetal impact with respect to different gravidities were statistically significant (X2=37.819, p<0.0001).

Maternal parity also had impact on the severity of fetal exposure to CO. Severe impact was prominent in Para 2-4 and 'Para o' and they occurred in 13(10.2%) out of 128 and 18 (7.8%) out of 230 women respectively. Moderate impact was also most prominent in Para 2-4 while mild impact was most prominent in ≥ Para 5. Generally, the differences between the fetal impact on exposure to CO in women of different parities were statistically significant (X2=12.098, p< 0.047) but no correlative pattern was established.

CONCLUSION

The mean value of fCOHb concentration ± SD which was 0.93±0.72% Out of the total 490 participants, 331(67.60%) had fetuses with mild impact from CO exposure (fCOHb= 0.28 to 0.85%), 125 (25.51%) – moderate impact (fCOHb = >0.85 to 1.70%) and 34 (6.90%) had severe impact (fCOHb = >1.70% and above). There were statistically significant differences in the mean values of fCOHb and the incidence of different degrees of severity when they were considered in association with different age categories, BMI, gravidity, parity. Unfortunately, there was no pattern in the directions of the differences except for BMI which showed significant positive correlation (r=0.214, p=0.0001) due mainly to maternal weight (r=0.181, p<0.0001). There was a paradoxical finding of 3 smokers having only mild impact.

RECOMMENDATIONS

A Nigerian Government–assisted air quality assessment and a universal maternal and fetal biomonitoring of the impact of maternal exposure to CO in the Niger Delta were highly recommended. They would go a long way identifying those regions that were worse affected and the women and the fetuses that were most at risk of the exposure.

REFERENCES

1. World Bank Defining an Environmental Development Strategy for the Niger Delta. (1995). Vol. 1, Industry and Energy Operations, West Central Africa Department.
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. Marzella, L., Myers, R.A.M. (1986). Carbon monoxide poisoning. Am Fum Physician, 34; 186194
5. 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.
6. 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.
7. 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.
8. Cramer, C. R. (1982). Fetal death due to accidental maternal carbon monoxide poisoning. *Journal of Toxicology: Clinical Toxicology*, *19*(3), 297-301.
9. 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.
10. Hutter, C. D. D., & Blair, M. E. (1996). Carbon monoxide—does fetal exposure cause sudden infant death syndrome?. *Medical hypotheses*, *46*(1), 1-4.
11. Margulies, J. L. (1986). Acute carbon monoxide poisoning during pregnancy. *The American journal of emergency medicine*, *4*(6), 516-519.
12. Woody, R. C., & Brewster, M. A. (1990). Telencephalic dysgenesis associated with presumptive maternal carbon monoxide intoxication in the first trimester of pregnancy. *Journal of Toxicology: Clinical Toxicology*, *28*(4), 467-475.
13. Singh, J. (1986). Early behavioral alterations in mice following prenatal carbon monoxide exposure. *Neurotoxicology*, *7*(2), 475-481.
14. 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.
15. Tachi, N., & Aoyama, M. (1983). Effect of cigarette smoke and carbon monoxide inhalation by gravid rats on the conceptus weight. *Bulletin of Environmental Contamination and Toxicology*, *31*(1), 85-92.
16. Osborne, J. S., Adamek, S., & Hobbs, M. E. (1956). Some components of gas phase of cigarette smoke. *Analytical Chemistry*, *28*(2), 211-215.
17. Mkpe, A., Oloyede, O., Adebari, K. I. Green, Bruno, C. Chinko. (2022). Carbon Monoxide (CO) Pollution in the Niger Delta area of Nigeria and Its Impact on Feto-Maternal Health. *Sch Int J Obstet Gynec*, 5(2), 57-64.
18. Smokerlyzer. (1996). Bedfont www.bedfont.com.
19. Alderman, B. W., Baron, A. E., & Savitz, D. A. (1987). Maternal exposure to neighborhood carbon monoxide and risk of low infant birth weight. *Public Health Reports*, *102*(4), 410.
20. Jarvis, M. J., Belcher, M., Vesey, C., & Hutchison, D. C. (1986). Low cost carbon monoxide monitors in smoking assessment. *Thorax*, *41*(11), 886.
21. 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.
22. World Health Organization. (2009). Environmental Health Criteria 213—Carbon Monoxide. 1999. *Available from URL: whqlibdoc. Who. int. Diakses tanggal*, *19*.
23. Maga, M., Janik, M. K., Wachsmann, A., Chrząstek-Janik, O., Koziej, M., Bajkowski, M., ... & Niżankowski, 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.
24. 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.
25. Kao, L. W., & Nañagas, K. A. (2004). Carbon monoxide poisoning. *Emergency Medicine Clinics*, *22*(4), 985-1018.
26. Vogt, T. M., Selvin, S., & Hulley, S. B. (1979). Comparison of biochemical and questionnaire estimates of tobacco exposure. *Preventive Medicine*, *8*(1), 23-33.
27. 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.
28. 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.
29. 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.