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(RESEARCH ARTICLE)

Persistence exposure to toxic oil and gas flaring pollutants-mediated insulin resistance and hyperinsulinemia among populations in the Niger Delta

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Abstract

Environmental pollutants of oil and gas flaring are fast emerging major threats to the global health communities and environmental authorities; and also, an important source of morbidity and mortality. From purposively selected 94 volunteers exposed to toxic oil and gas flared pollutants in Ogoni land, Niger Delta, Nigeria, serum samples were used to evaluate possible association between non-communicable diseases (NCDs) and insulin resistance using C-peptide and Insulin kits. Volunteer's age ranged 18 to 50 years, and were exclusively females resident consistently, in the various communities, in the order of 10 years and above. When compared to control, the outcomes of this study indicated high levels of insulin resistance, insulin, beta cell function, blood glucose of 78%, 76.2%, 71.6%, 52.5%with corresponding decrease in connecting peptide value of 43.2% was associated within creases in various physio-metabolic risk factors including low density lipoprotein, triglyceride, total cholesterol, high density lipoprotein and body mass index of 64.9%, 62.4%, 60.6%, 52.5% and 52% respectively in the exposed population to oil and gas flaring pollutants. Meanwhile, the prevalence of cardio-metabolic risk factor ratio (TG/HDL-C) was higher for the exposed population (63.6 %.) than the control (36.4%); while the prevalence of hyperinsulinemia/insulin resistance showed age and time dependent increase of exposure. This study validates both insulin resistance and toxic pollutions of oil and gas flaring as sensitive indicators of multiple metabolic abnormalities. Further, the inherent capacity of toxic oil and gas flared pollutants to elicit detectable pathologies in various tissues and organ induced-insulin resistance, might plausibly be the mechanist-link, in metabolic derangements, in the development and progression of a number of chronic non-communicable diseases, including diabetes, dyslipidaemia, hypertension, cardiovascular diseases, cancers, respiratory diseases, obesity and complications and disabilities and even death.

Keywords: Insulin Resistance; Hyperinsulinemia; Non-Communicable Disease; Ogoni; Inorganic Toxic Pollutants; Environmental Pollution-Centered Hypothesis

1. Introduction

In recent years, increased risk for environmental toxicity to combined, cumulative or prolonged exposures to various petroleum environmental pollutants, especially from poisonous air, soil and water toxins, has appeared as a major worldwide health and environmental concerns. It is recognized that toxic pollution in the air, soil and water is among the leading non-communicable diseases (NCDs) risk factor globally and are responsible for an estimated 16% of all NCDs mortality [1]. Accordingly, the greatest increases in NCDs mortality are seen in low-income and middle-income countries, now experiencing epidemics of obesity, diabetes, cardiovascular disease, and cancer [1,2]. Of the three quarters of all NCDs deaths, 82% of the 16 million people who died prematurely, or before reaching 70 years of age, occur in low- and middle-income countries [3].

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In the Niger Delta, there is increasing tendency of its populations exposed to toxic oil and gas exploration and exploitation activities as well as second-hand smoke or black soot becoming more vulnerable to developing non-communicable diseases (NCDs) [2,4,5,6,7,8] with its complications [9] and rare syndromes [10]. However such studies [2,5,6]couldn't attribute the cause of NCDs, either to change of life-style due to impact of westernization and rapid urbanization or due to genetic predispositions. Aside, the three independently significant and varying NCDs: overweight and obesity, raised blood pressure, raised blood glucose with their associated risk factors (pre-diabetes and pre-hypertension) was suggested to be intricately linked to each other [2,5,7]. Previous study reported that the pathogenesis and progression in the risk of developing metabolic deformability, such as type 2 diabetes, in the Niger Delta, was associated with target organ damage, like left ventricular hypertrophy [11]. However, the mechanisms linking NCDs with one another remain incompletely understood.

Emerging evidence attributed increases in the risk of developing NCDs in the Niger Delta to chronic exposure to potentially toxic petroleum chemically unrelated inorganic pollutants, present in the air, water and soil such as lead, cadmium and vanadium, through exacerbation of physiological changes of a broad and long-lasting impact adversely affect the health of humans [12-14,16-20]. Available literature simply illustrated correlation of constant exposure to petroleum environmental pollutant risk factors along with biological risk factors for NCDs [,2,4 ,5,6,21]. It has been hypothesized that toxic pollution through differential perturbations of membrane diffusion potential [2,22,23] aggravation of inflammatory response and oxidative stress [24,25] exert adverse effects such as tissue injury and organ dysfunction induced-increases in insulin resistance [2,5,6]. Increased caloric intake and sedentary habit have been suggested that cannot account as the principal environmental factors for the development and progression of insulin resistance [2,5,6]

Having this in mind, the present study was undertaken to assess insulin resistance and its metabolic risk factors with corresponding risk factors for NCDs, among 94 volunteers continually exposed to toxic pollution from the air, soil and water, such as lead, cadmium and vanadium respectively[12,13]. Insulin resistance or diminished insulin sensitivity, defined as a reduced biological action of insulin, is a well-known unifying pathophysiological denominator or sensitive indicator of multiple metabolic abnormalities frequently associated with cluster of NCDs risk factors including elevated plasma glucose, lipid regulation problems (elevated triglycerides increased small low-density lipoproteins and decreased high-density lipoproteins), hypertension obesity and organ damage, like left ventricular hypertrophy, atherosclerosis and chronic kidney disease[26-29]. It is argued below that prolonged exposure to toxic pollution in the air, water and soil, can stimulate long-term chronic inflammation, which may play important role in development and progression of NCDs-linked insulin resistance through multiple pathways regulating metabolism. To the best of our knowledge, this is the first study reporting on the mechanisms of toxic oil and gas pollution association with insulin resistance with its metabolic risk factors in the population of Ogoni as well as in that of Niger Delta.

2. Material and methods

2.1 Study area

This study was carried out on volunteers resident in Khana and Gokana Local Government Areas respectively in Ogoni land of Rivers State in the Niger Delta, which is home to the highest levels of air, water and soil pollution contaminated with heavy metals such lead, cadmium and vanadium, from oil and gas flaring or petroleum exploration activities [12,13].On the other hand, volunteers that serve as control were drawn from non-petroleum exploitation and exploration areas of Nigeria -Ogoja and Boki Local Government Areas of Cross River State [12,13]. The study was carried out between the years 2019 - 2020.

2.2 Population of the Study

Oral consent was given before the interviews began. Those who gave consent were requested to visit the hospital for blood testing. A total of 384 women within the ages of 18 and 50 years were recruited for the study which 184 met the inclusion criteria. Of the 184 volunteers, 94 with median age 35.60 years (SEM±0.86) living with the potentially toxic oil and gas flaring chemically unrelated compounds in the Ogoni land [12,13]serve as test group. The other 90, median age 30.98 years (SEM± 0.99), live in the non-petroleum exploration and exploitation environments and serve as control [12,13].The volunteers recruited were apparently healthy women who consented in writing or thumb-printed to participate in the study without any known ailment. All participants were interviewed by using a pre-tested, pre-designed standard questionnaire as previously described [12,13]. Medical officers, nurses and laboratory scientists in the hospitals within the region as well as community leaders were consulted for the studies in the various communities. The purpose of the study was well defined and permission was sort from the community heads for sensitization to

encourage full and freely participation in the study. All participants must have been residents in the communities consistently for at least ten years and above.

2.3 Analytical Procedures

Blood samples for the analysis of pancreas functions parameters were as described previously [12,13]. Systolic and diastolic blood pressure, body mass index, triglycerides, cholesterol, LDL-cholesterol, fasting blood glucose (FBG) were measured using standard methods[2,5,6,8].Pancreas functions parameters- β-cell function and insulin resistance from basal glucose and insulin or C-peptide concentrations were analyzed using C-peptide and Insulin kits obtained from CALBIOTECH USA, in strict compliance with the instructions provided by the manufacturer. Insulin resistance was calculated by using Homeostasis Model Assessment (HOMA-IR) approach-insulin (u/UmL) x blood glucose (mg/dL)/405.

2.3.1 Ethics declarations

Ethical clearance for this study was obtained from the Government Health Research Ethical Committees of Rivers State, and Cross River State (REC No. CRCMOHREC2020113) and the Institutional Ethical Research Committee, University of Port Harcourt (UPH/CEREMAD/REC/MM67/019), and the study were carried out in strict compliance to the guidelines of the National Committee for Research Ethics in Science and Technology.

2.4 Statistical analysis

Analysis was carried out using Statistical Package for Social Science (SPSS) version 22.0. Data analysis were expressed as mean ± SEM. Data were analysed using Independent Samples Test. Percentages for independent variables were calculated; $p<0.05$ was considered statistically significant.

3. Results and discussion

This study confirms insulin resistance with its metabolic risk factors as sensitive indicators or molecular markers associated with toxic oil and gas chemically unrelated inorganic environmental pollutants (such lead, cadmium and vanadium [12-15] - induced multiple metabolic abnormalities. Inorganic toxic pollutants lead, cadmium and vanadium have been measured in biological samples, such as human blood[12-14], water and soil[12-15,30].The toxic pollutants might have cause decreased in insulin sensitivity due to the disruption of various molecular pathways cause insulin resistance in agreement with previous reports [31,32,33]. A probable underlying cause of insulin resistance with its metabolic risk factors could plausibly be attributed to toxic oil and gas flared pollutants through aggravation of inflammatory response, oxidative stress, or differential perturbations of biological membrane [22-25] among others, exert adverse impact on metabolic actives leading to insulin resistance. The implication is that reduction or inhibition of inflammation by controlling and prevention of toxic environmental pollution may play important role in improvements of insulin resistance and human metabolic functions

Table 1 describes typical mean values and percent difference calculated for both biological and biochemical parameters among 184 apparently healthy women aged 18 - 50 years who participated in the study. Evidently, the mean values of insulin resistance, insulin, beta cell function, blood glucose and connecting peptide of 78%, 76.2%, 71.6%, 52.5% and 43.2% of the exposed populations with respect to control was associated with various physio-metabolic risk factors including low density lipoprotein, triglyceride, total cholesterol, high density lipoprotein and body mass index of 64.9%, 62.4%, 60.6%, 52.5% and 52% respectively. The results of this study rather than being merely confirmatory reinforced the concept that insulin resistance enhances exposure to oil and gas flared pollutants to the risk of developing chronic metabolic dysfunctions. The toxic pollutants thus constitute the metabolic derangement initiating hyperinsulinemia in the exposed population. Interestingly, the exposed populations are however, unaware of being predisposed to developing insulin resistance with its metabolic risk factors and the associated metabolic abnormalities in agreement with previous studies [2,4,5,6]. These findings are suggestive that persistence/chronic exposure to potentially toxic oil and gas flaring chemically unrelated pollutants leads to increases in the probability of the development of insulin resistance with its metabolic risk factors, could be the plausible mechanist-link in the pathogenesis of many chronic physiological metabolic dysfunctions, including all of NCD mortality.

Table 1 Mean age, biological and biochemical parameters of the study population

Parameters	Study population (n=184)		Percentage difference	P-value
	Control (n=90)	Test(n=94)		
Age(years)	30.98±.99 (18-50)	35.60±.86 (19-50)	13.87	0.001
Body Mass Index (kg/m2)	23.72±.48 (14.4-38.3)	25.88±.50 (15.79-40.37	8.81	0.002
Systolic Blood Pressure (mmHg)	117.51±1.45 (89-170)	118.46±2.15 (85-210)	1.48	0.719
Diastolic Blood Pressure (mmHg)	74.66±1.03 (52-106)	71.59±1.48 (43-118)	-4.19	0.92
Total Cholesterol (mg/dl)	71.53±1.86 (28.79-100.34)	109.98±2.39 (51.25-187.06)	42.36	0.001
Triglycerides (mg/dl)	35.88±.90 (17.11-58.19)	59.42±1.45 (28.52-96.98)	49.40	0.001
High density lipoprotein(mg/dl)	28.63±.80 (14.41-48.98)	31.67±.85 (16.01-54.43)	10.08	0.001
Low density lipoprotein(mg/dl)	35.72±1.84 (1.8-64.14)	65.93±2.06 (12.68-147.41)	59.43	0.001
Blood sugar (mg/dl)	104.07±3.24 (61.0-196.0)	115.09±4.85 (80.0-432.0)	10.58	0.132
Insulin (µIU/ml)	8.60±1.05 (0.04-53.72)	27.48±2.46 (0.55-110.13)	219.53	0.001
Connecting Peptide (ng/ml)	0 .54±.01 (0.39-1.16)	0 .41±.00 (0.38-0.97)	-24.07	0.001
Insulin resistance	2.20	7.80	254.54	
Beta cell function	75.38	189.91	151.93	

Data presented as mean±SEM, $p<0.05$ considered significant (range in parenthesis)

In this study, the percent concentration ratio for the cardio-metabolic risk factors (TG/HDL-C) for the exposed populations (63.6%) was higher with respect to control (36.4%) and showed that insulin resistance, however, may not merely be a metabolic abnormality, but also a complex and multifaceted syndrome that can affect other tissues and organs in consonance with previous studies elsewhere. Beside, the prevalence of insulin resistance was 22% for the control and 78% for the exposed population aged 30.98±.99 and 35.60±.86 years respectively (Table 1). Table 2 describes the relationship between age, insulin and connecting peptides while Table 3 depicts the relationship between duration of exposure to environmental pollution with insulin and connecting peptides for the studied populations. It revealed preponderance of insulin resistance/hyperinsulinemia (which frequently coexist), with increase in age and duration of exposure to toxic pollution, while the reverse was the case for connecting peptides. The results demonstrate that the abnormalities of insulin resistance play a mechanistic role in oil and gas flaring pollution induced-NCDs, and indicated that both insulin resistance and oil and gas flared pollutants are involved in time-age dependent organ and tissue specific phenomenon-pathogenesis of NCDs.

Table 2 Relationship between age, insulin and connecting peptide of the population

Parameters		Age (years)			
		<25	**25-34**	**35-44**	**45-50**
Insulin (µIU/ml)	Control	5.93±1.93 (.19-53.72)	10.99±1.83 (.49-36.36)	9.82±2.56 (.04-47.63)	6.86±1.14 (2.24-109.38)
	Test	12.68±58 (10.45-16.19)	23.35±3.80 (6.84-110.13)	34.06±4.66 (.55-102.83)	29.65±5.47 (7.63-109.38)
	% difference	113.82	112.46	246.84	332.21
	P value	0.310	0.009	0.001	0.001
Connecting Peptide (ng/ml)	Control	0.57±.04 (.39-1.16)	0.51±.01 (.43-.85)	0.53±.03 (.42-.94)	0.52±.02 (.47-.70)
	Test	0.50±.04 (0.40-.83)	0.40±.01 (0.38-.97)	0.39±.00 (0.39-.42)	0.39±.00 (0.39-.42)
	% dif.	-12.28	-21.56	-26.41	-25
	P value	0.126	0.001	0.001	0.006

Data presented as mean±SEM, p<0.05 considered significant (range in parenthesis)

Table 3 Relationship of duration of exposure to environmental pollution with insulin and connecting peptide

Parameters		Duration of exposure (years)				
		<11	**11-20**	**21-30**	**31-40**	**41-50**
Insulin (µIU/ml)	Control	9.75±2.19 (1.16-53.72)	6.56±1.99 (.19-47.63)	10.18±2.34 (.49-30.27)	8.23±1.80 (.57-16.18)	7.32±2.34 (0.04-20.64)
	Test	23.57±3.44 (8.69-68.27)	37.49±8.20 (7.94-102.83)	23.73±5.06 (6.84-110.13)	30.82±5.25 (0.55-96.91)	24.09±6.16 (6.32-109.38)
	% difference	141.74	471.49	133.10	274	229.09
	P value	0.012	001	.024	.001	.038
Connecting Peptide (ng/ml)	Control	0.57±.03 (0.39-1.16)	0.57±.03 (0.39-1.10)	0.48±.01 (0.39-.69)	0.46±.01 (0.42-.50)	0.52±.03 (0.44-.70)
	Test	0.41±.01 (0.38-.62)	0.44±.04 (0.39-.96)	0.41±.02 (0.38-.83)	0.39±.01 (0.39-.42)	0.39±.01 (0.39-.41)
	% difference	-28.07	-22.80	-14.58	-15.21	-25.00
	P value	.000	.005	.095	.130	.015

Data presented as mean±SEM, p<0.05 considered significant (range in parenthesis)

4. Conclusion

Based on these results, chronic exposure to toxic oil and gas flaring pollution increases the risk of developing insulin resistance, which plausibly could be the underlying mechanisms in the pathogenesis and progression of metabolic disorders in NCDs.

In essence, accumulation of toxic pollution from the air, soil and water induced-persistence insulin resistance accounts for the pathogenesis and progression of the various metabolic abnormalities, which is of great concern. From this study,

the need to take urgent action on both environmental pollution and diseases of long duration, slow progression, that do not resolve spontaneously and that rarely achieve complete cure, is clearer than ever.

Provision of clean environment, by tackling of oil and gas exploration and exploitation activities as well as second-hand smoke or black soot by the Government, though could be one of the most important ways of preventing and controlling human metabolic diseases or reducing deaths from non-communicable diseases (NCDs), it is also as an important mean to provide a huge economic and social development boost for the country.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare they have no conflict of interests.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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