Research Article

Toxic metals levels in cord and maternal blood and possible association with low birth weight of babies

Osaretin James Agbonlahor[1], Abiodun Mathias Emokpae[2], Andrew Ehiozee Evbuomwan[3]

[1]Department of Medical Laboratory Services, Stella Obasanjo Women and Children Hospital, Benin, Nigeria, [2]Department of Medical Laboratory Science, School of Basic Medical Sciences, University of Benin, Benin, Nigeria, [3]Department of Obstetrics and Gynecology, Stella Obasanjo Women and Children Hospital, Benin, Nigeria

Correspondence to: Abiodun Mathias Emokpae, E-mail: mathias.emokpae@uniben.edu

Received: February 19, 2016; **Accepted:** November 13, 2017

ABSTRACT

Background: The birth weight of neonates is a reflection of the maternal health, nutritional status, and environmental exposure to toxic metals. Very few studies have evaluated prenatal exposure to toxic metals and its possible association with small for date's babies in Nigeria. **Objective:** The objective of this study was to evaluate the levels of lead, cadmium, and mercury in maternal and cord blood among pregnant women at delivery and to ascertain whether there is any relationship between toxic metals levels and neonatal birth weight. **Materials and Methods:** A total of 300 subjects were recruited, which included 200 healthy pregnant women who were attending antenatal clinic and 100 age-matched non-pregnant women (controls). The concentrations of toxic metals in the cord and maternal blood were measured using atomic absorption spectrophotometer. **Results:** The maternal and cord blood levels of lead, cadmium, and mercury were significantly higher ($P < 0.001$) in small for date's babies than in babies with normal birth weight. There was a significant positive correlation between cord blood lead ($r = 0.088$, $P = 0.02$) and cadmium ($r = 0.119$, $P = 0.03$) with neonatal birth weight. There was, however, no significant association ($r = 0.009$; $P = 0.89$) between mercury and neonatal birth weight. **Conclusion:** The levels of the measured toxic metals were significantly higher in maternal and cord blood of those who had small for date babies than those who had babies with normal birth weight. This study indicated that increased levels of toxic metals may be associated with small for date's babies.

KEY WORDS: Maternal and Cord Blood; Toxic Metals; Low Birth Weight

INTRODUCTION

There is an increasingly global concern about the weight of babies at birth, as low birth weight is an important factors determining infant mortality and morbidity.[1] The World Health Organization defined low birth weight as weight at birth of <2500 g.[2] Most of the determinants of neonatal birth weight have been linked to maternal nutritional status and environmental exposure to toxic metals.[3] Toxic metals are known to cause adverse environmental and health hazards such as spontaneous abortion, reduced birth weight and length, premature delivery, congenital malformation, gestational hypertension or impaired neurodevelopment, and altered thyroid hormone status of newborn.[4] In pregnant women, these metals reached the fetus by transplacental transfer. Studies have shown that lead (Pb), cadmium (Cd), and mercury (Hg) levels in maternal and cord blood are associated with neonatal birth weight.[5] However, data on the effect of maternal and neonatal exposure to these toxic metals *in utero* on neonatal birth weight in our setting are very scarce, as few studies had considered prenatal exposure to toxic metals in Nigeria.[6,7] Obi *et al*.[7] examined the levels of Cd in pregnant women and its association with neonatal birth weight. The authors

Access this article online	
Website: http://www.ijmsph.com	Quick Response code
DOI: 10.5455/ijmsph.2018.0209413112017	

reported an increased level of Cd in women who had babies with low birth weight compared to those with normal birth weight. The other study[6] observed a positive association between methyl-mercury and neonatal birth weight. However, to the best of our knowledge, none had examined the levels of maternal and prenatal exposure *in utero* to multiple toxic metals (Pb, Cd, and Hg) and their possible association with low birth weight of babies in Nigeria. Reports elsewhere on the association between toxic metals and low birth weight of babies have not been consistent. Whereas some observed association between toxic metals and small for dates babies,[3,7-9] others failed to find any association.[10-12] This study was designed to determine the levels of Pb, Cd, and Hg in maternal and cord blood among pregnant women at delivery and to determine whether there is relationship between the levels of toxic metals and neonatal birth weight in Nigeria. The study may provide insight into the level of environmental exposure to toxic metals in the general population and their possible health hazards on pregnant women who are non-occupationally exposed and the newborn babies.

MATERIALS AND METHODS

Study Population

Two hundred healthy pregnant women attending antenatal clinic were consecutively enrolled before the onset of confirmed labor for deliveries at Stella Obasanjo Hospital, Benin City. The control group comprised 100 age-matched non-pregnant women who were recruited from among staff and students of the hospital.

Ethical Consideration

Institutional ethical approval was obtained from Ethical Committee of the Edo State Hospitals Management Board and individual inform consent was obtained before the commencement of study. Demographic and clinical information were obtained using structured questionnaires.

Inclusion Criteria

Healthy pregnant women who aged 18 years and above expecting singleton, pregnant women who were on routine antenatal drugs, and pregnant women who had carried the pregnancy to term and delivered either by spontaneous vaginal delivery or cesarean section were included.

Exclusion Criteria

The pregnant women with chronic illness such as diabetes mellitus, hypertension, renal failure, heart diseases, and thyroid dysfunction were excluded. Those who had preterm deliveries, abruption placenta previa, intrauterine death, bad obstetric history, intrauterine rupture and congenital anomalies of the baby, polyhydramnios, clinical signs of infection, and benign tumors and malignancies were also excluded.

Specimen Collection

About 5 ml of venous blood was collected from the antecubital vein of the mother at the onset of labor and immediately after delivery; 5 ml of blood was collected from the umbilical vein of the respective maternal origin into anticoagulated container and labeled. The samples were kept refrigerated before toxic metals analysis.

Determination of Cadmium

The concentration of Cd in maternal and cord blood was analyzed with electrothermal atomic absorption spectrometer (Perkin Elmer analyst 800, Norwalk, USA) using method previously described.[13]

The matrix modifier solution was prepared by mixing 50 µL of 0.3 g/L of Mg $(NO_3)_2$ and 1 ml of 0.33 g/L of Pd $(NO_3)_2$, and 2 ml of 0.2% v/v nitric acid and 50 µL of 0.1% Triton X-100. Then, 1 in 7 dilution of whole blood was made with 0.2% HNO_3 and 0.1% Triton X-100 and cadmium standard working solution (1 µg/L) was made by dilution with deionized water.

The light source (hollow cathode lamp) specific for cadmium was inserted into the electrothermal atomic absorption spectrometry (ETAAS), the wavelength was adjusted to 228.8 nm. The instrument was standardized with the standard blank (1% HNO_3) and cadmium standard.

An aliquot of 20 µL of whole blood was injected directly into the graphite furnace. Equal volume of matrix modifier was injected into the graphite furnace.

The concentration of Cd (µg/L) was displayed on the screen after the runtime (4 min).

Determination of Lead

The concentrations of Pb in maternal and cord blood were analyzed with electrothermal atomic absorption spectrometer (Perkin Elmer analyst 800, Norwalk, USA) adopting the methods described below.[13] Lead standard working solution was diluted with deionized water. Matrix modifier solution 50 µL of 10 g/L of $NH_4H_2PO_4$ was mixed with 2 ml of 0.2% v/v nitric acid and 0.1% Triton X-100 s. 1 in 13 dilution of whole blood was made with 0.2% HNO_3 and 0.1% Triton X-100. The hollow cathode lamp specific for lead was inserted into the ETAAS. The wavelength was adjusted to 283.3 nm and the instrument was standardized and calibrated with standard blank and lead standard.

An aliquot of 20 µL of whole blood was injected directly into the graphite furnace.

Equal volume of matrix modifier solution was also injected into the graphite furnace. The concentration of lead in the sample was displayed on the screen after the runtime (3 min).

Determination of Mercury

Mercury was determined by inductively coupled plasma mass spectrometer (Agilent 7500, Norwalk, USA) using the methods described by Fong *et al*.[14] 100 µL of Hg standard was diluted with 5 ml of HNO_3 and 1.7 ml of deionized water and mixed thoroughly with 200 µL internal standard. 100 µL of whole blood was diluted with 5 ml of HNO_3 and 1.7 ml of deionized water and mixed thoroughly with 200 µL internal standard and 100 µL of gold. The instrument was standardized and calibrated with standard blank and mercury standards. Aliquot of 20 µL of the prepared sample was aspirated into the quartz spray chamber. The result data was displayed on the screen after the run.

Statistical Analysis

The data obtained were analyzed using Statistical Package for the Social Sciences program (SPSS) version 16.0 (Chicago, IL, USA).The values obtained in this study are presented as mean ± standard error of mean. Student's *t*-test and analysis of variance were used to compare means while correlation coefficient was calculated using linear regression analysis. $P < 0.05$ was considered as statistically significant.

RESULTS

The results of the investigations are presented on Tables 1-4.

Table 1 summarizes the levels of Pb, Cd, and Hg in both maternal and cord blood. The level of Pb in pregnant women who had babies with low birth weight was significantly higher (0.20 ± 0.016 vs. 0.13 ± 0.018; $P < 0.001$) than in those who had babies with low birth weight. Similarly, the levels of Cd (1.07 ± 0.128 vs. 0.61 ± 0.073; $P < 0.001$) and Hg (0.07 ± 0.014 vs. 0.06 ± 0.017; $P = 0.022$) were significantly higher in those who had babies with low birth weight than those who had babies with normal birth weight. The concentrations of Cd (0.31 ± 0.023 vs. 0.17 ± 0.016; $P < 0.001$) and Hg (0.025 ± 0.005 vs. 0.022 ± 0.002; $P = 0.010$) in cord blood of babies with low birth weight were higher than in those with normal birth weight. The level of Pb in cord blood of babies with low birth weight was higher but not statistically significant when compared to those with normal birth weight (0.071 ± 0.003 vs. 0.065 ± 0.004; $P = 0.305$).

Table 1: Comparison of the levels of measured toxic metals in maternal and cord blood of low and normal birth weight babies (mean±SEM)

Measured toxic metals	Birth weight		*P*
	LBW (*n*=24)	NBW (*n*=176)	
Lead (µg/L)s			
Maternal	0.20±0.016	0.13±0.018	0.007
Cord	0.071±0.003	0.065±0.004	0.305
Cadmium (µg/L)			
Maternal	1.07±0.128	0.61±0.073	0.000
Cord	0.31±0.023	0.17±0.016	0.000
Mercury (µg/L)			
Maternal	0.07±0.014	0.06±0.017	0.022
Cord	0.025±0.005	0.022±0.002	0.010

LBW: Low birth weight, NBW: Normal birth weight, SEM: Standard error of the mean

Table 2 summarizes that statistically significant positive correlation was observed between cord blood Pb ($r = 0.088$; $P = 0.02$) and Cd ($r = 0.119$; $P = 0.03$) with neonatal birth weight. While the correlation between cord blood Hg ($r = 0.009$; $P = 0.89$) and neonatal birth weight was however not significant.

Table 3 summarizes that Pb (0.178 ± 0.011 vs. 0.068 ± 0.002), Cd (0.767 ± 0.046 vs. 0.189 ± 0.010), and Hg (0.068 ± 0.003 vs. 0.027 ± 0.001) levels in maternal blood were significantly higher ($P < 0.001$) than the levels in cord blood. The values of maternal: Cord blood ratio ranges from 2.52 for Hg, 2.62 for Pb to 4.06 for Cd.

Table 4 summarizes the mean concentration of the measured parameters in pregnant women at delivery and non-pregnant women (controls). Pb (0.18 ± 0.01 vs. 0.29 ± 0.01) and Hg (0.07 ± 0.03 vs. 0.08 ± 0.004) levels in the pregnant women at delivery were significantly lower ($P < 0.001$) compared with controls. While Cd (0.77 ± 0.05 vs. 0.18 ± 0.01) level in the pregnant women was significantly higher ($P < 0.001$) compared to non-pregnant women (controls).

DISCUSSION

This study evaluated the levels of toxic metals (Pb, Cd, and Hg) in cord and maternal blood and the concentrations correlated with neonatal birth weight. There were significantly higher levels of Pb ($P < 0.007$), Cd ($P < 0.001$), and Hg ($P < 0.022$) in blood of those women who had small for date's babies compared to those who had babies with normal birth weight. The levels of Pb ($P < 0.305$), Cd ($P < 0.001$),

Table 2: Relationship between measured toxic metals in cord blood and birth weight of babies

Toxic metals	*r*	*P*
Lead (ug/l)	0.088	0.02
Cadmium (ug/l)	0.119	0.03
Mercury (ug/l)	0.009	0.89

Table 3: Comparison of the levels of toxic metals in maternal and cord blood samples (mean±SEM)

Parameters	Lead (ug/l)	Cadmium (ug/l)	Mercury (ug/l)
Maternal	0.178±0.011	0.767±0.046	0.068±0.003
Cord	0.068±0.002	0.189±0.010	0.027±0.001
Maternal/cord ratio	2.62	4.06	2.52
Number of pairs	200	200	200
P	0.000	0.000	0.000

SEM: Standard error of the mean

Table 4: Comparison of the levels of toxic metals in pregnant women at delivery and non-pregnant women (control) (mean±SEM)

Parameters	Lead (ug/L)	Cadmium (ug/L)	Mercury (ug/L)
Pregnant women on delivery *n*=200	0.18±0.01	0.77±0.05	0.07±0.03
Control groups *n*=100	0.29±0.01	0.18±0.01	0.08±0.004
P	0.000	0.000	0.000

SEM: Standard error of the mean

and Hg ($P < 0.001$) in cord blood were higher in babies with low birth weight than in those with normal birth weight. The levels of Pb ($P < 0.022$) and Cd ($P < 0.03$) were significantly associated with birth weight of babies. The consequences of environmental toxicants on the fetus underscore the inability of the placental to completely protect the fetus from exposure. It could be due to the indirect effect of the toxicants on the mother and or the placenta.

To the best of our knowledge, no study has evaluated prenatal exposure to multiple toxic metals in Nigeria, and this is the first study in Benin City, Edo state; South-South Nigeria. The study evaluated maternal and cord blood concentrations of multiple toxic metals in pregnant women and the extent of transplacental transfer of these metals from the maternal circulation to the fetus.

The data presented in this study provided information on the levels of maternal exposure to the measured toxic metals and their possible association with low birth weight of babies in non-occupationally exposed subjects in our environment. It indicated that maternal-fetal transfer was a major source of early life exposure to toxic metals. The observed higher levels of toxic metals in women and cord blood of babies with low birth weight is consistent with those reported elsewhere.[5,15] Our finding however did not agree with some other studies.[16,17] These latter authors observed no significant difference between maternal and cord blood Pb concentrations in small for date's babies and those with normal birth weight. The possible reason for this inconsistency may be due to the low levels of Pb concentration observed in their studies compared to the present study. However, maternal lead exposure during pregnancy had been reported as a risk indicator for reduced fetal growth,[17] and Pb is an important environmental pollutant which may cause several adverse fetal outcomes including low birth weight.[18]

Various molecular, cellular, and intracellular mechanisms have been advanced to explain lead toxicity. These include generation of oxidative stress, ionic mechanism, and apoptosis.[15] Of these, oxidative stress has been observed to be more pronounced and much more severe.[19] Lead causes the generation of reactive oxygen species, which result in damage to various biomolecules such as DNA, proteins, enzymes, and membrane-based lipids and it also simultaneously impairs the antioxidant defense system.[19] Although the mechanism by which Pb reduces birth weight is not fully understood, it was proposed to have both local and systemic effects. By local effect, it decreases fetal skeletal growth after been deposited in fetal osteocytes and systemically, it has an affinity for fetal thyroid tissue causing decrease in thyroid hormones levels which in turn cause decreased in soft tissue organ growth, hence decrease in birth weight.[20]

The maternal and cord blood Cd levels in this study were significantly higher in cord of babies with low birth weight than those with normal birth weight. This observation is in agreement with other authors[2,7-9] but disagreed with some other studies.[10-12] The latter did not find any association between Cd levels and birth weight of babies. Although most of these studies were done only in maternal blood, the positive correlation observed between maternal and cord blood Cd levels in this study shows that fetal Cd levels is dependent on maternal concentration. The possible reasons these authors did not observe a significant association between Cd exposure levels and birth weight could be because of low Cd exposure in the population studied. Cadmium had previously been reported to be associated with low birth weight in babies elsewhere. A significantly inverse correlation was earlier observed between maternal Cd and zinc concentration.[7] This suggests that as Cd concentration increases, there was a decline in maternal zinc concentration. Zinc is required for normal fetal development and it affects pregnancy outcome.[21]

Reduced uteroplacental blood flow, reduced nutrient transport, and placental toxicity are the proposed mechanisms by which Cd exerts its adverse effects leading to low birth weight, fetal toxicity, malformations, and death.[22] Cadmium competes with zinc for divalent cationic sites in metalloenzymes and Cd antagonizes the action of zinc such that a high Cd/Zn ratio is needed for toxicity.[23] Some authors have suggested that the toxic effects of Cd may be mediated by altered metabolism of zinc and copper since adequate availability of zinc is necessary for normal growth and development. Inadequate zinc or copper levels in fetal or early postnatal life is teratogenic, retards growth, and changes cognitive function.[23] It, thus, suggests that even when the pregnant women are taking adequate diet the nutrients may not be made available to the fetus. Cadmium also utilizes the transport pathways normally used by other biologically essential trace elements and hence causes death of cells through the disruption of important pathways like the electron transport chain.[24] Cadmium activates macrophages leading to secretion of numerous intracellular mediators, cytokines, and vasoactive amines.[25] These processes result in oxidative stress and DNA damage with their attendant consequences.[26] Cadmium was reported to cause cellular dysfunction through abnormal signal transductions[27] and formation of metallothioneins which retain Cd in the placenta to protect the fetus and disrupt placental zinc dynamics.[28,29] The association of maternal and cord blood Cd levels with low birth weight of babies is highly marked in this present study.

The maternal mercury level in this study was significantly higher than the levels in cord blood ($P < 0.001$). The observation did not agree with other authors,[6] who observed that the mercury values in the cord blood were significantly ($P < 0.001$) higher than their respective maternal blood. These observations show that there is the transfer of mercury across the placental barrier. Methylmercury (MeHg) easily pass through the placenta through active transport by amino acid carriers.[30,31] This is most active in the presence of higher concentration gradient of this metal in maternal circulation. The lower level in our study possibly explains why we observed higher maternal mercury levels than in cord blood. However, some authors[18] reported that the active transplacental transport and high affinity of MeHg to hemoglobin results in the bioaccumulation of this metal in cord blood where there is higher hemoglobin concentration than in maternal blood which results in higher cord to maternal mercury ratio in red blood cells. This could also be the reason for the observation of higher mercury levels in cord than in maternal blood.[6]

We observed also that the Hg levels in maternal and cord blood of babies with low birth weight were significantly ($P < 0.05$) higher than babies with normal birth weight. This could be one of the probable causes of small for date's babies observed in this study. However, this was not the case with some studies.[32,33] They reported that mercury levels in cord blood of normal birth weight babies were higher than those in small for dates babies. However, recent studies have attributed to the differences in the concentrations of mercury in the various reports to maternal fish consumption habits, as many species contain beneficial nutrients that can compensate for mercury toxic effects.[32] Unfortunately, we were unable to assess if the results from our study were affected by fish species consumed due to lack of field information.

CONCLUSION

It was observed that Pd, Cd, and Hg levels were significantly higher in maternal and cord blood of babies with low birth weight when compared with those with normal birth weight. However, there was a positive correlation between these metals in cord blood and birth weight. The increased levels of toxic metals observed in this study may be due to environmental exposure from vehicular exhaust smoke and waste, cigarette smoking, petroleum products, as well as fish consumption. This is because these pregnant women were occupationally unexposed to these toxic metals. It is also evident that transplacental transfer from maternal to fetus may be responsible for the levels of these toxic metals in cord blood and their possible association with intrauterine growth retardation as observed. Therefore, the measurement of the levels of toxic metals may be considered important as part of the routine antenatal investigations, especially in patients with previous clinical history of recurrent abortion, preterm delivery, and small for date's babies.

ACKNOWLEDGMENTS

We appreciate the contributions of all medical and nursing staff of Stella Obasanjo Women and Children Hospital for their assistants toward the completion of this study.

REFERENCES

1. Eltahir ME, Gerd S. The effect of maternal anthropometric characteristics and social factors on gestational age and birth weight in Sudanese newborn infants. BMC Pub Health 2008;8:244-51.
2. Salpietro CD, Gangemi S, Minciullo PL, Briuglia S, Merlino MV, Stelitano A, *et al.* Cadmium concentration in maternal and cord blood and infant birth weight: A study on healthy non-smoking women. J Perinat Med 2002;30:395-9.
3. Berghella V. Prevention of recurrent fetal growth restriction. Obstet Gynecol 2007;110:904-12.
4. Iijima K, Otake T, Yoshinaga J, Ikegami M, Suzuki E, Naruse H, *et al.* Cadmium, lead, and selenium in cord blood and thyroid hormone status of newborns. Biol Trace Elem Res 2007;119:10-8.
5. García-Esquinas E, Pérez-Gómez B, Fernández-Navarro P, Fernández MA, de Paz C, Pérez-Meixeira AM, *et al.* Lead, mercury and cadmium in umbilical cord blood and its

association with parental epidemiological variables and birth factors. BMC Public Health 2013;13:841.
6. Obi E, Okafor C, Igwebe A, Ebenebe J, Afonne OJ, Ifediata F, *et al.* Elevated prenatal methyl mercury exposure in Nigeria: Evidence from maternal and cord blood. Chemosphere 2015;119:485-9.
7. Tawari EP, Anetor JI, Charles-davis MA. Cadmium levels in pregnancy, influence on neonatal birth weight and possible Amelioration by some essential trace elements. Vitam Trace Elem 2012;1:103.
8. Nishijo M, Nakagawa H, Honda R, Tanebe K, Saito S, Teranishi H, *et al*. Effects of maternal exposure to cadmium on pregnancy outcome and breast milk. Occup Environ Med 2011;59:394-7.
9. Kippler M, Hoque AM, Raqib R, Ohrvik H, Ekström EC, Vahter M, *et al.* Accumulation of cadmium in human placenta interacts with the transport of micronutrients to the fetus. Toxicol Lett 2010;192:162-8.
10. Dwivedi D, Madhu J, Shuchi J. An association between maternal lead and cadmium levels and birth weight of babies in North Indian population. O J Obst Gyneacol 2013;3:331-6.
11. Zhang YL, Zhao YC, Wang JX, Zhu HD, Liu QF, Fan YG, *et al.* Effect of environmental exposure to cadmium on pregnancy outcome and fetal growth: A study on healthy pregnant women in china. J Environ Sci Health A Tox Hazard Subst Environ Eng 2004;39:2507-15.
12. Odland JO, Nieboer E, Romanova N, Thomassen Y, Lund E. Blood lead and cadmium and birth weight among sub-arctic and arctic populations of Norway and Russia. Acta Obstet Gynecol Scand 1999;78:852-60.
13. Olmedo P, Pla A, Hernández AF, López-Guarnido O, Rodrigo L, Gil F, *et al.* Validation of a method to quantify chromium, cadmium, manganese, nickel and lead in human whole blood, urine, saliva and hair samples by electro thermal atomic absorption spectrometry. Anal Chim Acta 2010;659:60-7.
14. Fong BM, Siu TS, Lee JS, Tam S. Determination of mercury in whole blood and urine by inductively coupled plasma mass spectrometry. J Anal Toxicol 2007;31:281-7.
15. Osman K, Akesson A, Berglund M, Bremme K, Schütz A, Ask K, *et al.* Toxic and essential elements in placentas of Swedish women. Clin Biochem 2000;33:131-8.
16. Bogden JD, Thind IS, Louria DB, Caterini H. Maternal and cord blood metal concentrations and low birth weight - a case-control study. Am J Clin Nutr 1978;31:1181-7.
17. Irgens A, Krüger K, Skorve AH, Irgens LM. Reproductive outcome in offspring of parents occupationally exposed to lead in Norway. Am J Ind Med 1998;34:431-7.
18. Chen Z, Myers R, Wei T, Bind E, Kassim P, Wang G, *et al.* Placental transfer and concentrations of cadmium, mercury, lead, and selenium in mothers, newborns, and young children. J Expo Sci Environ Epidemiol 2014;24:537-44.
19. Garza A, Vega R, Soto E. Cellular mechanisms of lead neurotoxicity. Med Sci Monit 2006;12:RA57-65.
20. Hernandez-Avila M, Peterson KE, Gonzalez-Cossio T, Sanin LH, Aro A, Schnaas L, *et al.* Effect of maternal bone lead on length and head circumference of newborns and 1-month-old infants. Arch Environ Health 2002;57:482-8.
21. Singh PP, Khushlani K, Veerwal PC, Gupta RC. Maternal hypozincemia and low-birth-weight infants. Clin Chem 1987;33:1950.
22. Lewis RM, Cleal JK, Godfrey KM. Placental role in fetal programming. In: Gupta RC, editor. In: Reproduction and Developmental Toxicology. London: Elsevier Inc.; 2011. p. 1039-49.
23. Emokpae MA, Agbonlahor OJ, Evbuomwan AE. The relationship between maternal blood cadmium, zinc levels and birth weight of babies in non-occupationally exposed pregnant women in Benin City, Nigeria. Journal of Medicine and Biomedical Research 2016;15(1):55-61.
24. Flora SJ, Pachauri V, Saxena G. Arsenic, cadmium and lead. In: Gupta RC, editor. Reproduction and Developmental Toxicology. New York: Elsevier Inc.; 2011. p. 41538.
25. Rikans LE, Yamano T. Mechanisms of cadmium-mediated acute hepatotoxicity. J Biochem Mol Toxicol 2000;14:110-7.
26. Zenzes MT. Smoking and reproduction: Gene damage to human gametes and embryos. Hum Reprod Update 2000;6:122-31.
27. Satoh M, Kaji T, Tohyama C. Low dose exposure to cadmium and its health effects. (3) toxicity in laboratory animals and cultured cells. Nihon Eiseigaku Zasshi 2003;57:615-23.
28. Llanos MN, Ronco AM. Fetal growth restriction is related to placental levels of cadmium, lead and arsenic but not with antioxidant activities. Reprod Toxicol 2009;27:88-92.
29. Tian LL, Zhao YC, Wang XC, Gu JL, Sun ZJ, Zhang YL, *et al.* Effects of gestational cadmium exposure on pregnancy outcome and development in the offspring at age 4.5 years. Biol Trace Elem Res 2009;132:51-9.
30. McAleer MF, Tuan RS. Metallothionein overexpression in human trophoblastic cells protects against cadmium-induced apoptosis. *In Vitro* Mol Toxicol 2001;14:25-42.
31. Kajiwara Y, Yasutake A, Adachi T, Hirayama K. Methylmercury transport across the placenta via neutral amino acid carrier. Arch Toxicol 1996;70:310-4.
32. Ramón R, Ballester F, Aguinagalde X, Amurrio A, Vioque J, Lacasaña M, *et al.* Fish consumption during pregnancy, prenatal mercury exposure, and anthropometric measures at birth in a prospective mother-infant cohort study in Spain. Am J Clin Nutr 2009;90:1047-55.
33. Oken E, Kleinman KP, Olsen SF, Rich-Edwards JW, Gillman MW. Associations of seafood and elongated n-3 fatty acid intake with fetal growth and length of gestation: Results from a US pregnancy cohort. Am J Epidemiol 2004;160:774-83.

How to cite this article: Agbonlahor OJ, Emokpae AM, Evbuomwan AE. Toxic metals levels in cord and maternal blood and possible association with low birth weight of babies. Int J Med Sci Public Health 2018;7(1):9-14.

Source of Support: Nil, **Conflict of Interest:** None declared.