

Heavy Metals among Female Adolescents Attending Secondary Schools in Kano, Nigeria

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Abstract— This study was conducted to examine the level of heavy metals among 192 apparently healthy female adolescents randomly selected from three different boarding secondary schools in the urban area of the most populated city in north-western part of Nigeria. Atomic absorption spectrometry (AAS) was used to determine the plasma levels of the heavy metals which include cadmium (Cd), cobalt (Co), chromium (Cr), copper (Cu), iron (Fe), manganese (Mn), molybdenum (Mo), nickel (Ni), lead (Pb) and zinc (Zn). Our findings revealed the following mean±SD values for each of the heavy metal; 0.11±0.01µg Cd/L, 0.09±0.02µg Co/L, 0.19±0.02µg Cr/L, 0.91±0.02µg Cu/L, 1.53±0.31µg Fe/L, 0.01±0.04µg Mn/L, 0.38±0.04µg Mo/L, 0.04±0.01µg Ni/L, 0.04±0.01µg Pb/L and 2.80±0.24µg Zn/L respectively. It was concluded that toxicity from heavy metals did not exist among female adolescents.

Keywords— Heavy metals, female, adolescents, Nigeria.

I. INTRODUCTION

HEAVY metals are ubiquitous in the environment [1] which overexposes humans to excessive concentrations that could invariably affect human health [2,3,4]. In fact, even individuals not occupationally exposed could get overexposure to certain metals from other sources such as food, beverages, or air [5,6]. In small quantities, certain heavy metals are nutritionally essential for a healthy life. Some of these are referred to as the trace elements (e.g., iron, copper, manganese and zinc). These elements, in free or complex state, are naturally found in foodstuffs, in fruits and vegetables, and in commercially available multivitamin products [7].

Although adolescents face a series of serious nutritional challenges, such as energy-protein malnutrition and micronutrient deficiencies [8]; they are however, particularly subject to the negative effects of a surplus of trace elements

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since they breathe more air per unit body weight, spend more time outdoors and has higher metabolic rates and organ development [9] than their adult counterparts. Studies have indicated that 60-80% of adolescents globally suffer from micronutrient deficiencies [10,11,12].

However, toxicity might results from nutritionally-essential trace minerals with potential toxicities at elevated intake or exposure [13,14]. Many toxic heavy metals act as molecular “mimics” of nutritionally essential trace elements which may compete with essential metallic cofactors for entry into cells and incorporation into enzymes [15].

For instance, cadmium and lead can effectively inhibit cellular glutathione peroxidase, reducing the effectiveness of this antioxidant defense system for detoxification [16]. Cadmium can compete with and displace zinc from proteins and enzymes while lead is chemically similar to calcium [15,17,18]. Lead can also displace magnesium and iron from certain enzymes involve in DNA synthesis as well as disrupt the function of zinc during heme synthesis [19]. Copper and iron can be displaced by mercury from the active site of enzymes involved in energy production which could induce mitochondrial dysfunction and oxidative damage [20].

Several toxic metals are lacking robust pathways for elimination and are known to remain in the body for a long time, thereby leading to accumulation which may increase with age [21]. We therefore aimed at assessing for the first time, the level cobalt, cadmium, copper, lead, zinc, manganese, iron, molybdenum, nickel and chromium in female adolescents residing in the urban area of the most populated city in West African sub region with several industrial enterprises which are in close proximity to residential buildings. There are also indiscriminate disposals and burnings of waste products these results in infringing on sanitary and protective zones. Such a situation is typical in industrialized countries, and leads to various undesirable effects on human health.

II. MATERIALS AND METHODS

A. Study site

Kano is the most densely populated city [22] with large transport and commercial centre situated in the North-western part of Nigeria. The global location of the city is between latitude 11° 30' north of the equator and longitude 08° 30' east of the Greenwich Meridian [23].

B. Subject and design

A total of one hundred and ninety two students were randomly recruited from three randomly selected female

boarding schools across the city for participation into the study following their written informed consent. Ethical permissions were obtained from the State Health and Secondary School Management Boards. Furthermore, the principal of each school authorized the study before the commencement.

C. BMI and Laboratory Analysis

Height was measured (with the subjects standing on bare feet) using a non-elastic measuring tape fastened to a vertical rod, to the nearest 0.5 cm. Weight was measured (with the subjects on bare feet and with light clothing) using an electronic weighing balance, to the nearest 0.1 kg. From the heights and weights got, Body Mass Index (BMI) was calculated using the formula BMI = Weight (kg)/[Height (m²)] [24,25].

Ten ml (10ml) blood samples were collected by venipuncture using either the antecubital vein or the dorsal vein and dispensed into lithium heparin anticoagulant bottles [26] and plasma separated by centrifugation at 3000 rpm for 5 minutes. Each sample (3ml plasma) was transferred into 100 ml conical flasks. Perchloric and nitric acids were added in the ratio 1:3. The conical flask was covered with an evaporating dish and the mixture digested at low temperature using a thermostated Bitinett hot plate until a clear solution was obtained. The digest was made up to 20ml with deionized water in a 20ml standard flask [27]. The sample solutions were then analyzed for cobalt, cadmium, copper, lead, zinc, manganese, iron, molybdenum, nickel and chromium using a Buck Scientific Atomic Absorption Spectrophotometer, model VPG 210.

D. Statistical Analysis

All data were presented as mean±SD of the heavy metals. Relationship between age and BMI with heavy metals is determined by Pearson's correlations using the Statistical Package for Social Sciences (SPSS version 15.0 for windows 2003, IBM Corporation, NY, USA).

III. RESULTS

TABLE I
CONCENTRATIONS OF HEAVY METALS BY AGE AMONG FEMALE ADOLESCENTS IN KANO

Age (years)	Cd	Co	Cr (µg/L)	Cu	Fe
12	0.09±0.03 ^a	0.14±0.04 ^a	0.20±0.10 ^a	0.84±0.36 ^a	1.32±0.21 ^a
13	0.12±0.05 ^a	0.07±0.04 ^a	0.16±0.11 ^a	0.73±0.11 ^a	1.22±0.18 ^a
14	0.11±0.05 ^a	0.08±0.04 ^a	0.16±0.10 ^a	0.86±0.19 ^a	1.31±0.19 ^a
15	0.11±0.05 ^a	0.07±0.03 ^a	0.21±0.15 ^a	0.77±0.12 ^a	1.28±0.30 ^a
16	0.11±0.06 ^a	0.08±0.05 ^a	0.18±0.16 ^a	0.81±0.14 ^a	1.44±0.48 ^a
17	0.10±0.05 ^a	0.08±0.04 ^a	0.18±0.11 ^a	1.04±0.02 ^a	1.76±0.68 ^a
18	0.12±0.06 ^a	0.10±0.04 ^a	0.22±0.11 ^a	1.45±0.21 ^a	2.04±0.92 ^a
19	0.10±0.03 ^a	0.09±0.04 ^a	0.21±0.07 ^a	0.74±0.21 ^a	1.90±0.82 ^a
Mean	0.11±0.01	0.09±0.02	0.19±0.02	0.91±0.02	1.53±0.31
Age (years)	Mn	Mo	Ni (µg/L)	Pb	Zn
12	0.08±0.03 ^a	0.46±0.01 ^a	0.17±0.01 ^a	0.04±0.03 ^a	3.11±0.90 ^a
13	0.11±0.05 ^a	0.42±0.09 ^a	0.02±0.10 ^b	0.04±0.02 ^a	3.20±0.97 ^b
14	0.12±0.06 ^a	0.37±0.09 ^a	0.02±0.01 ^c	0.04±0.02 ^a	2.74±0.77 ^c
15	0.11±0.04 ^a	0.34±0.11 ^a	0.02±0.01 ^d	0.05±0.02 ^a	2.68±0.93 ^d
16	0.10±0.05 ^a	0.33±0.12 ^a	0.02±0.01 ^e	0.04±0.01 ^a	2.64±0.84 ^e
17	0.13±0.05 ^a	0.37±0.16 ^a	0.02±0.00 ^f	0.03±0.02 ^a	2.46±0.70 ^f
18	0.15±0.06 ^a	0.41±0.16 ^a	0.02±0.00 ^g	0.03±0.01 ^a	2.82±0.72 ^g
19	0.13±0.07 ^a	0.36±0.05 ^a	0.02±0.01 ^h	0.04±0.01 ^a	2.80±0.73 ^h
Mean	0.01±0.04	0.38±0.04	0.04±0.01	0.04±0.01	2.80±0.24

^{a-h}Values are mean±SD, values with same superscripts within the same column are considered not significant (p<0.05).

TABLE II
MEAN CONCENTRATIONS OF HEAVY METALS BY BMI AMONG FEMALE ADOLESCENTS IN KANO

BMI range	Cd	Co	Cr (µg/L)	Cu	Fe
<18.5 (underweight)	0.11±0.07 ^a	0.08±0.04 ^a	0.16±0.12 ^a	0.79±0.13 ^a	1.36±0.30 ^a
18.5-24.9 (normal)	0.11±0.05 ^a	0.09±0.05 ^a	0.22±0.15 ^a	1.02±0.19 ^a	1.64±0.72 ^a
25-30 (overweight)	0.09±0.04 ^a	0.07±0.03 ^a	0.13±0.08 ^a	0.80±0.22 ^a	1.66±0.61 ^a
30-40 (obesity)	0.11±0.02 ^a	0.06±0.04 ^a	0.14±0.05 ^a	0.89±0.16 ^a	1.42±0.05 ^a
BMI range	Mn	Mo	Ni (µg/L)	Pb	Zn
<18.5 (underweight)	0.11±0.04 ^a	0.38±0.11 ^a	0.20±0.01 ^a	0.04±0.01 ^a	2.75±0.85 ^a
18.5-24.9 (normal)	0.12±0.06 ^a	0.36±0.13 ^a	0.02±0.01 ^a	0.04±0.02 ^a	2.68±0.82 ^a
25-30 (overweight)	0.14±0.04 ^a	0.38±0.01 ^a	0.02±0.00 ^a	0.04±0.01 ^a	2.32±0.40 ^a
30-40 (obesity)	0.11±0.07 ^a	0.28±0.06 ^a	0.03±0.01 ^a	0.04±0.01 ^a	2.09±0.40 ^a

Values are mean±SD, values with different superscripts within the same column are considered not significant (p<0.05).

The mean concentrations of heavy metals in the study participants aged 12-19years showed 0.11±0.01µg Cd/L, 0.09±0.02µg Co/L, 0.19±0.02µg Cr/L, 0.91±0.02µg Cu/L, 1.53±0.31µg Fe/L, 0.01±0.04µg Mn/L, 0.38±0.04µg Mo/L, 0.04±0.01µg Ni/L, 0.04±0.01µg Pb/L and 2.80±0.24µg Zn/L respectively (Table 1).

Values for Co, Cr, Cu, Fe, Mn, Mo, and Pb did not differ significantly (p>0.05) across the individual age groups of the participants. Plasma concentrations of Ni is lower (p<0.05) in younger adolescent but Zn is significantly higher (p<0.05) in younger adolescents.

Tables 2 presents mean concentrations of heavy metals according to BMI. Plasma concentrations of Cd, Co, Mo, Pb and Zn do not differ significantly (p>0.05) between underweight (BMI<18.5) and normal (BMI 18.5-24.9) subjects respectively. Plasma concentration for Cr, Cu, Fe, Mn and Ni differ significantly (p<0.05) according to all the BMI range. Similarly, plasma level of Cd is not associated (p>0.05) with Co, Cr, Mn, Ni and Pb, but significantly associated (p<0.05) with Cu, Fe, Mo and Zn respectively. Also, Co level is not associated (p>0.05) with Cr, Mn, Ni and Pb, but significantly associated (p<0.05) with Cu, Fe, Mo and Zn respectively. Plasma level of Cr is not associated (p>0.05) with Mn, Mo, Ni and Pb, but significantly associated (p<0.05) with Cu, Fe and Zn.

IV. DISCUSSION

Heavy metals such as lead and cadmium have no biological function in human system and are potentially toxic even at trace concentrations [28]. Results from this study indicated a range of Cd levels from 0.02-0.27µg/L in 12-19 year female adolescents. Mean±SD plasma concentration (0.11±0.01µg Cd/L) was found among the 12-19 year study participants. Age does not influence cadmium plasma concentration, unlike the BMI of the study participants.

Values of Cd recorded in our study is lower than plasma concentrations (0.08 to 0.5µg Cd/L) found in 8 to 12-year-old children living in polluted areas in France, Czech Republic and Poland [29]. Also, for children living in the vicinity of smelters in the Netherlands, the average was 0.76µg Cd/L [30]. Under the framework of the European Phime project, mean

concentration of Cd in female adolescents 15-20 years living in Bastogne, Lessines and Louvain-La-Neuve was found to be 0.18 $\mu\text{g/L}$ (normal 0.14-0.29 $\mu\text{g/L}$).

Cadmium bioavailability, retention and consequently toxicity are affected by several factors such as nutritional status (low body iron stores) and multiple pregnancies, pre-existing health conditions or diseases [31]. Environmental discharge of cadmium due to the use of petroleum products, combustion of fossil fuels (petroleum and coal) and municipal refuse contribute to airborne cadmium pollution [32] and possibly introduce high concentrations of this potential reproductive toxicant into the environment.

Thus, the Cd levels among participants of the present study seem to be lower than what was reported in the previous study originating from the different parts of the world. This is indeed interesting, because it suggests that, perhaps due to some presently unknown reasons, female adolescents from our part of the world do not uptake Cd and/or retains excessive levels of this potentially toxic heavy metal.

Concentration of cobalt identified among the study participants ranges from 0.00-0.25 $\mu\text{g/L}$. Mean plasma concentration (0.09 \pm 0.02 $\mu\text{g Co/L}$) recorded in this study indicated that our subject have values within acceptable plasma/serum limits of <1.2 $\mu\text{g/L}$ as reported by [32]. Our study did not indicate influence of age to Co exposure. Some studies however, show cobalt predominance in women [33,34]; some detergents and cosmetics may as well contain cobalt [35]. Toxic levels are anything over 85nmol/L (5 $\mu\text{g/L}$) [36].

[37] reported high level of cobalt (14.00 $\mu\text{g/g}$) in *Moringa oleifera*, 5.00 $\mu\text{g/g}$ in onion. [38] reported spider web content of 19.68 and 27.09 $\mu\text{g/g}$ in both indoors and outdoors areas within the study. Thus, our study participants are having values within acceptable reported limits despite possible sources of exposure to this heavy metal, which might indicate high tolerance rate by the body.

Chromium exposure of our study groups indicated levels ranging from 0.00-0.75 $\mu\text{g/L}$. Mean plasma concentration (0.19 \pm 0.02 $\mu\text{g Cr/L}$) recorded in this study indicated that our subject have values within acceptable plasma/serum limits of 0.04-0.35 μg

Cr/L for populations that are not occupationally exposed to chromium [39]. According to our study, BMI but not age has significant effect on plasma Cr concentration level.

In spite of the multiple exposure of the study participants to common household chromium sources such blue prints, primer paints, household chemicals and cleaners, cements, among others, findings from our study did not indicate an elevation of chromium levels, perhaps due to some activities that might limit the Cr entry and/or stimulate its elimination from the body system.

It was found in this study that the content of copper among the study participants ranges from 0.43-9.09 $\mu\text{g/L}$. Mean plasma Cu concentration of 0.91 \pm 0.02 $\mu\text{g/L}$ was recorded among our study participants. Free serum copper: 1.6-2.4 $\mu\text{mol/L}$ or 10-15 $\mu\text{g/dL}$ has been reported by [40] among USA female adolescents.

Our finding discovered a profound influence of BMI on plasma Cu concentration. Food is reportedly a major source of copper and its concentrations in foods may vary widely between countries due to growing conditions [41]. Copper ions

(originated from any copper salts or oxide) have an irritant effect on mucosal membranes and daily intakes ranging from 2 to 32mg in drinking water have been reported to cause symptoms of general gastric irritation [42].

A study in Wisconsin also suggested that high levels of copper in the water supply may increase the rate of gastrointestinal upsets [43]. A study on the acute gastrointestinal effects of drinking water containing graded levels of added copper [44] showed that an average daily consumption of 1.64 litres of drinking water containing 3mg/L ionised copper(II) was associated with nausea, abdominal pain or vomiting.

The major source of water for the subjects in this study area was pipe-borne water [45], which might contain copper. According to an earlier study [46], Copper pipes may contribute substantially to the intake of copper and depending on the copper content of the local soil; 13% - 50% of copper in meals may be supplied by water [47]. Despite multiple sources of Cu exposure in our study area, female adolescents tend to have plasma Cu level within reported acceptable limits. This could probably be as a result of poor body uptake of Cu which is facilitated by cigarette smoking and alcohol consumption.

Our study revealed concentration of iron ranging from 0.41-3.82 $\mu\text{g/L}$ and a mean value of 1.53 \pm 0.31 $\mu\text{g/L}$. Plasma iron concentration is significantly determined by age and BMI. Normal serum level of 0.5-1.5mg/L was earlier reported by [48]. Iron overload is the accumulation of excess iron in body tissues, and it usually occurs as a result of a genetic predisposition to absorb iron in excess of normal [49].

However, it can also be caused by excessive ingestion of iron supplements or multiple blood transfusions [50]. In advanced stages of iron overload disease (hemochromatosis), the iron accumulates in the parenchymal cells of several organs, but particularly the liver, followed by the heart and pancreas; this condition can lead to organ dysfunction and even death [50]. There was no elevation of plasma iron concentration notable in this study, though possible sources of iron in drinking water, iron pipes, and cookware are common in the study area.

Findings from our study indicated concentration of manganese among the study participants ranges from 0.00-0.29 $\mu\text{g/L}$. Mean plasma Mn concentration (0.01 \pm 0.04 $\mu\text{g/L}$) was recorded in this study. Plasma concentration of Mn is significant associated with age and BMI among our study group. Normal ranges of manganese levels in body fluids are 4-15 $\mu\text{g/L}$ in blood, 1-8 $\mu\text{g/L}$ in urine, and 0.4-0.85 $\mu\text{g/L}$ in serum [51].

For children and adolescents, [51] scaled the adult UL values according to reference body weights for children and adolescents, noting that there were no reports of manganese toxicity in children and adolescents and that it was not possible to establish UL values for infants (0-12 months). Manganese is an essential ingredient in steel, dry-cell batteries, glass and fireworks, chemical manufacturing, certain paints, cosmetics, leather and textile industries and as a fertilizer. Organic forms of manganese are used as fungicides, fuel-oil additives, smoke inhibitors, an anti-knock additive in gasoline, and a medical imaging agent [51]. Normal plasma Mn in our study could probably be as a result of low outdoor activities by our study participants.

Our study revealed molybdenum values 0.05-0.75 $\mu\text{g/L}$. Mean plasma Mo concentration (0.38 \pm 0.04 $\mu\text{g/L}$) was recorded among our study participants. The range of Mo concentrations in sera collected from 110 healthy humans was 0.44-1.62 $\mu\text{g/L}$ [52]. There are no relevant studies of molybdenum or molybdate carcinogenicity in animals or humans [53]. In animals, acutely toxic oral doses of molybdenum result in severe gastrointestinal irritation with diarrhea, coma and death from cardiac failure. Subchronic and chronic oral exposures can result in growth retardation, anaemia, hypothyroidism, bone and joint deformities, sterility, liver and kidney abnormalities, and death. When the high dietary intake range of 96-500 $\mu\text{g/day}$ molybdenum for the European population is considered, the consumption of a food supplement providing 20 $\mu\text{g/day}$ would result in a total anticipated exposure of between 116 and 520 $\mu\text{g/day}$.

Very little is known about specific effects of molybdenum compounds on human health and there are no well designed chronic studies in man which can be used for risk assessment [54]. The available studies refer to putative effects of molybdenum in foods, drinking water or to data obtained by using stable isotopes of molybdenum as tracers. In an area in Armenia, where the population is exposed to a high dietary intake of molybdenum, due to geophysical soil levels of 77mg molybdenum/kg and 39 mg copper/kg, aching joints, elevated concentrations of uric acid in the blood and urine, increased blood molybdenum-containing xanthine oxidase (XO) activity and gout-like symptoms have been reported [55].

Findings from our study revealed Ni content between 0.00-0.05 $\mu\text{g/L}$ among female adolescents. Mean value for Ni (0.04 \pm 0.01 $\mu\text{g/L}$) recorded in this study is significant associated with age and BMI of the subjects. Nickel has not been shown to be essential for humans. Orally ingested nickel salts can cause adverse effects on kidneys, spleen, lungs and the myeloid system in experimental animals [54]. It is said to influence iron absorption and metabolism and the haemopoietic process. However, biochemical functions of nickel have not been demonstrated in humans and higher animals [54]. Reference values for nickel in healthy adults are 0.2 $\mu\text{g/L}$ in serum and 1-3 $\mu\text{g/L}$ in urine [56]. In non-occupationally exposed men, the mean concentration of nickel in whole blood and serum is in the range of 1-5 $\mu\text{g/L}$ and in urine less than 10 $\mu\text{g/L}$ [57]. Normal plasma Ni concentration in the present study could be attributed to lack of possible high risk contaminating industries around the study area.

Concentration of Lead ranges from 0.00-0.09 $\mu\text{g/L}$ among our study participants. Mean plasma Pb value (0.04 \pm 0.01 $\mu\text{g/L}$) discovered among the female adolescents is significant affected by age not by the BMI. The mean blood Lead level among children in the USA is 2 $\mu\text{g/dL}$ [58]. The geometric mean Blood Lead Level (BLL) for children aged 1-5 years is 1.5 $\mu\text{g/dL}$, aged 6-11 is 1.0 $\mu\text{g/dL}$, and aged 12-19 years is 0.8 $\mu\text{g/dL}$ [59]. Lead is associated with automobile related pollution [60]. The best-studied effect of lead toxicity is cognitive impairment, measured by IQ tests [61]. The strength of this association and its time course has been observed to be similar in multiple studies in several countries [62].

In most countries, including the United States, blood lead concentrations peak at approximately 2 years of age and then decrease without intervention [61]. High level of blood Lead is

not unexpected in our study area; where the municipal water supply pipes has been in existence for more than fifty years, there were also no existent legislative laws on prohibiting high content of lead in paints, petrol or other household utensils. There was also non existence of functional laws governing waste disposal. Automobile exhausts are also believed to account for more than 80% of the air pollution in some urban centres in Nigeria [63].

[37] reported high level of Lead (10.2-17.14 $\mu\text{g/g}$) in Okro and Onion and 28.00 $\mu\text{g/g}$ in soil around the study area. Similarly, spider webs were shown to contain high lead 503.34 and 662.50 $\mu\text{g/g}$ in both indoors and outdoors urban areas of study sites [38]. However, the most probable reason for low lead concentration among our study participants may be due to their poor uptake of these heavy metals from the environment and their nature of low outdoor activities which might limits their exposure to automobile pollutions.

Zinc concentration was found to be in the range 1.15-4.59 $\mu\text{g/L}$. Mean plasma Zn concentration (2.80 \pm 0.24 $\mu\text{g/L}$) found among our subjects is not associated with age and BMI. Serum normal zinc values among women are 83.3 $\mu\text{g/dL}$ [64]. [65] reported serum zinc normal range value of 84-159 $\mu\text{g/dL}$ among Japanese.

Zinc concentration found in our study could also be as a result of storage of food or drink in galvanized containers and the obsolete methods piping municipal water as well as the old-age habit of drinking water dropping from zinc coated roofs (due to acute shortages of drinking water) practiced in the study community which could eventually resulted to excessive quantities of zinc intake that may lead to both acute and chronic toxicity. Even though, multiple source of exposure is rampant in our study area, our study participants have plasma concentrations within reported acceptable limits. This may clearly shows their tolerance to this heavy metal.

Even though toxicity and the resulting threat to human health of any contaminant are functions of concentration, it is a known fact that chronic exposure to heavy metals at relatively low levels can cause adverse effects. In Nigeria, recent reports indicate that the major contaminants found in drinking water especially from wells are heavy metals [60]. These heavy metals find their way into the soil and groundwater through activities like intense agriculture, power generation, industrial discharges, seepage of municipal landfills, septic tank effluents, to mention a few. In fact, many authors have reported high levels of heavy metal ions in the soil, rivers and groundwater in different areas of Nigeria [37,38,66]. Indiscriminate disposal of toxic wastes therefore poses a great threat to human health [60].

CONCLUSION

Our study revealed no toxicity of heavy metals among our study groups. This however does not guaranteed future toxicity from these heavy metals due to high rate of environmental pollutions characterizing the study site.

CONFLICT OF INTEREST STATEMENT

We declare that we have no conflict of interest.

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