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Association of lead and cadmium exposure with frailty in US older adults

Esther García-Esquinas [a,b,c,*], Ana Navas-Acien [c,d,e], Beatriz Pérez-Gómez [b,f], Fernando Rodríguez Artalejo [a,b]

[a] Department of Preventive Medicine and Public Health, School of Medicine, Universidad Autónoma de Madrid/ IdiPAZ, Madrid, Spain
[b] CIBER of Epidemiology and Public Health (CIBERESP), Madrid, Spain
[c] Department of Environmental Health Sciences, Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD, USA
[d] Department of Epidemiology, Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD, USA
[e] Welch Center for Prevention, Epidemiology, and Clinical Research, Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD, USA
[f] Environmental Epidemiology and Cancer Unit, National Center for Epidemiology, Carlos III Institute of Health, Madrid, Spain

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ABSTRACT

Background: Environmental lead and cadmium exposure is associated with higher risk of several age-related chronic diseases, including cardiovascular disease, chronic kidney disease and osteoporosis. These diseases may lead to frailty, a geriatric syndrome characterized by diminished physiologic reserve in multiple systems with decreased ability to cope with acute stressors. However, no previous study has evaluated the association between lead or cadmium exposure and frailty.

Methods: Cross-sectional study among individuals aged ≥60 years who participated in the third U.S. National Health and Nutrition Examination Survey and had either blood lead (N=5272) or urine cadmium (N=4887) determinations. Frailty was ascertained with a slight modification of the Fried criteria, so that individuals meeting ≥3 of 5 pre-defined criteria (exhaustion, low body weight, low physical activity, weakness and slow walking speed), were considered as frail. The association between lead and cadmium with frailty was evaluated using logistic regression with adjustment for relevant confounders.

Results: Median (intertertile range) concentrations of blood lead and urine cadmium were 3.9 μg/dl (2.9–4.9) and 0.62 μg/l (0.41–0.91), respectively. The prevalence of frailty was 7.1%. The adjusted odds ratios (95% confidence interval) of frailty comparing the second and third to the lowest tertile of blood lead were, respectively, 1.40 (0.96–2.04) and 1.75 (1.33–2.31). Lead concentrations were also associated with the frequency of exhaustion, weakness and slowness. The corresponding odds ratios (95% confidence interval) for cadmium were, respectively, 0.97 (0.68–1.39) and 1.55 (1.03–2.32), but this association did not hold after excluding participants with reduced glomerular filtration rate: 0.70 (0.43–1.14) and 1.09 (0.56–2.11), respectively.

Conclusions: In the US older adult population, blood lead but not urine cadmium concentrations showed a direct dose–response relationship with frailty. These findings support that lead exposure increases frailty in older adults.

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1. Introduction

Frailty is a geriatric syndrome characterized by a decreased ability to cope with acute stressors resulting from aging-related decline in reserve and function of multiple physiologic systems, including the cardiovascular, metabolic, immune, endocrine, or nervous systems (Clegg et al., 2013). The progressive accumulation of deficits in these systems increases the risk of disease onset and leads to a cycle of events (i.e. undernutrition, sarcopenia, diminished strength or exercise intolerance) that perpetuate frailty. With the addition of new stressors, the cycle of frailty can easily progress to disability (Vermeulen et al., 2011), institutionalization (Fried et al., 2001) and death (Graham et al., 2009; Song et al., 2010). Given the serious consequences of frailty and its high frequency, which reaches 10% of individuals over 60 years and 25% of those over 80 (Clegg et al., 2013), extensive research is being conducted to determine why some older adults become frail, and to identify preventable risk factors and mechanistic pathways. Despite this effort, few studies have evaluated the role of

* Corresponding author at: Department of Preventive Medicine and Public Health, School of Medicine, Universidad Autónoma de Madrid, Calle del Arzobispo Morcillo 4, 28029 Madrid, Spain.
E-mail address: esthergge@gmail.com (E. García-Esquinas).

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environmental contaminants on the risk of frailty, and most of them have focused on the effects of air pollution (Eckel et al., 2012; Myers et al., 2013).

The Environmental Protection Agency (EPA, 2014) has identified common environmental exposures that may especially harm the health of older adults, including metals, pesticides, water contaminants or air particles. For some contaminants (i.e. organic pollutants, certain metals) this increased risk may be related to bioaccumulation over the lifetime. Higher vulnerability in older adults may also partly result from age-related alterations in cellular function which impair the capability to maintain physiologic homeostasis (Geller and Zenick, 2005). Additionally, aging is associated with major changes in body composition that could influence the absorption and distribution of environmental chemicals in human tissues (Nordberg et al., 2007).

Lead and cadmium are two toxic metals widely distributed in the environment that accumulate in the human body, resulting in chronic endogenous exposure tissues (Nordberg et al., 2007). Increasing evidence supports the contribution of environmental lead and cadmium to the development of several age-related chronic diseases, including cardiovascular disease (Navas-Acien et al., 2007; Tellez-Plaza et al., 2013b), chronic kidney disease (Navas-Acien et al., 2009), and osteoporosis (Engstrom et al., 2011; Gallagher et al., 2008; Khalil et al., 2008). Despite this evidence, and the established connection between these diseases and the frailty syndrome (Heuberger, 2011), no previous studies have examined the association between lead or cadmium exposure and frailty. To evaluate this question, we used data from the third US National Health and Nutrition Examination Survey (NHANES III). We hypothesized that increasing concentrations of blood lead or urine cadmium are associated with increasing frequency of frailty in older adults.

2. Methods

2.1. Study participants

NHANES III was a multistage, stratified, clustered probability survey of the US civilian non-institutionalized population, conducted between 1988 and 1994 by the National Center for Health Statistics. The survey consisted of a household interview and a standardized physical examination performed in a mobile center. Our analysis was initially limited to adults ≥ 60 years of age who had completed the physical examination ($N=5724$). For our study, we further restricted the sample to participants who had either blood lead or urine cadmium determinations available ($N=5579$). The study was approved by the NHANES Institutional Review Board (IRB), and written informed consent was obtained from all subjects.

3. Study variables

3.1. Lead and Cadmium

Blood and spot urine samples were collected during the physical examination. All collection and storage materials used for metal analysis were prescreened for background contamination levels. Blood lead concentrations were measured by graphite furnace atomic absorption spectrophotometry. The limit of detection (LOD) for blood lead was 1 μg/dl and the interassay coefficients of variation ranged from 2.78% to 8.11%. For 2.8% of the participants with blood lead concentrations under the LOD, these were replaced by the LOD divided by the squared root of 2.

Urine cadmium was measured by Zeeman effect graphite furnace atomic absorption. The LOD for cadmium was 0.03 μg/dl and the interassay coefficients of variation ranged from 2.83% to 13.57%. Cadmium was undetectable for 2.5% of the participants and the levels replaced by the LOD divided by the square root of 2. To account for urine dilution, cadmium levels were either adjusted or divided by urine creatinine (μg/g). Additional details regarding laboratory procedures have been described in detail (Gunter et al., 1996).

3.2. Frailty

Assessment of frailty was performed using a modification of the definition developed by Fried et al. (2001) in the Cardiovascular Health Study (CHS). Individuals meeting ≥ 3 of the following criteria were considered as frail: (1) *Exhaustion*, defined as any of these responses "some difficulty", "much difficulty" or "unable to do it" to the question "How much difficulty do you have walking from one room to the other on the same level?". (2) *Low body weight*, characterized by a body mass index (BMI) ≤ 18 kg/m^2. (3) *Low physical activity*, considered present if the individual answered "less active" to the following question: "When compared to most men/women of your age, would you say that you are more active, less active or about the same?". (4) *Weakness*, defined as any of these responses "some difficulty", "much difficulty" or "unable to do it" to the question "How much difficulty you have lifting or carrying something as heavy as 10 pounds?". (5) *Slow walking speed*, defined as the worse quintile in the eight-foot walking speed test, adjusted for sex and height (Guralnik et al., 1994). The main modifications with respect to the CHS definition of frailty were that the nutrition criterion was based on low BMI rather than on weight loss in the preceding months, and that weakness was self-reported rather than based on the direct assessment of grip strength; similar modifications are common in the frailty literature, and usually result from the information available in each dataset.

3.3. Other variables

We collected data from a number of variables (gender, age, education, race/ethnicity, tobacco smoking, number of smokers at home and number of drug treatments used) that may act as potential confounders because they have shown to be independently associated with the outcome (Fried et al., 2001; Mello Ade et al., 2014) and with the exposure (Nordberg et al., 2007). Additionally, participants were asked about medical conditions that could be acting as mediators of the studied association (i.e. previous history of cardiovascular disease (congestive heart failure, coronary heart disease, angina or stroke), hypertension, diabetes, osteoarticular disease (osteoporosis, rheumatoid arthritis and osteoarthritis), respiratory disease (asthma, chronic bronchitis or emphysema) and cancer).

Weight and height were measured in standardized conditions, and BMI was calculated as weight in kg divided by squared height in m. During the medical examination, blood pressure was measured three times with the participant seated for 5 min and using an appropriate sized cuff. Hypertension was defined as self-reported physician diagnosis of high blood pressure or a mean systolic/diastolic blood pressure $\geq 140/90$ mmHg. Serum creatinine was measured using the modified kinetic Jaffe reaction, with a coefficient of variation that ranged between 0.2% and 1.4%. Chronic kidney disease was defined as a glomerular filtration rate (GFR) < 60 mL/min/1.73 m^2, estimated using the CKD-EPI equation (Levey et al., 2009). Serum cotinine was measured using high performance liquid chromotography/atmospheric pressure chemical ionization tandem mass spectrometry. The LOD for serum cotinine using this method was 0.05 ng/ml and values under the LOD were replaced by the square root of 2.

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