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Research Article

Tobacco Use in Adolescents Exposed To Lead: Preliminary Results

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Abstract

Background: Cross-sectional studies report associations between early lead exposure and behavior problems in childhood and adolescence, but little is known about later tobacco use.

Objective: This study investigated the associations between lead level in childhood or adolescence and tobacco use.

Methods: Children (n=89) with elevated lead levels in childhood were tested in adolescence using atomic absorption spectrometry. To examine the influence of lead levels in childhood and adolescence on tobacco use comparisons between lead levels in childhood and adolescence were conducted between smokers and non-smokers.

Results: Over 90% of the sample had BLLs above 10 µg/dl in childhood (mean BLL's =14.2 µg/dl). Although adolescent smokers did not differ from non-smokers in childhood lead levels, adolescent lead levels were higher among those who were using tobacco.

Conclusion: Adolescent tobacco use may contribute to levels of lead among adolescents previously exposed to environmental lead.

INTRODUCTION

Uruguay is a middle-income country in Latin America with a population of over 3 million. Adolescent substance use has been of increasing concern to the public health authorities in Uruguay. Alcohol and tobacco consumption are highly relevant problems for this age group, both due to the magnitude of the problem, the negative consequences as a result of use and abuse, and the long-term health problems [1]. Although recent national surveys indicate that tobacco use prevalence may be declining [2, 3], data from the Organization of American States Inter-American Drug Abuse Control Commission indicates that Uruguay is among the countries with the highest prevalence of past-month cigarette use across a range of years [4]. Recent research indicates that about 18% of adolescents aged 13 to 18 years of age are current smokers [5].

Lead is an environmental contaminant and has long been established as a potent neurotoxin, damaging the developing nervous system in children and potentially interfering with their long-term behavioral development [6]. Cross-sectional studies have reported an association between early lead exposure and behavior problems as well as psychiatric co morbidity in later childhood and adolescence, but little is known about environmental lead exposure in childhood and later tobacco use [7-10].

Lead exposure in latin america

In low- to middle-income countries, exposure to lead during childhood is a public health concern [11]. Until December 2003, the primary gasoline used in Uruguay contained tetraethyl lead, a significant source of environmental lead exposure [12]. Other sources of lead exposure include metallurgies, dust and chips from old paint, battery recyclers and plumbing made of lead pipelines, some of which pose a continuing public health risk in Uruguay [13,14]. Across the Latin American countries surveyed in the late 1990's, childhood lead exposure levels ranged between 11.5 µg/dl (SD=3.7) to 39.0 µg/dl (SD=5.0). In Uruguay, a recent report indicates that among preschool children, mean blood lead levels are 9.0 µg/dl [15]. According to the US Centers for Disease Control and Prevention (CDC) action level for blood lead was 10µg/dL. This level has been recently questioned, because many recent studies demonstrate neuro developmental

effects well below the CDC threshold of 10 μg/dL, with no lower threshold for injury identified as of yet [16]. The CDC Advisory Committee for Childhood Lead Poisoning Prevention (ACCLPP) now recommends using a reference value based on the 97.5th percentile of the BLL distribution among children 1–5 years old in the United States (currently 5 μg/dL) [17].

OBJECTIVE

The present study investigated whether 2001 blood lead levels (BLL) or current BLL collected in 2011 had an effect on drug use in this group of adolescents.

METHODS

Sample

Between June and December 2001 a cohort of children were identified with elevated blood lead levels (BLL's) in Montevideo, Uruguay. Faculty from the Department of Toxicology at the University of the Republic collected data and blood samples from this group. Inclusion criteria include previous participation in the biomonitoring project in 2001 and a recorded blood lead level greater than 5 μg/dL in 2001. Subjects were identified through medical records from the Department of Toxicology. The parents of potential subjects were telephoned and asked if they were interested in enrolling their children in a lead exposure follow-up study. A total of 89 adolescents agreed to participate in the study completed an assessment focused on social and demographic variables.

Measures

Cigarette use: Use of various substances was assessed by the Spanish translation of the CRAFFT to participants in 2011 when they were adolescents. The CRAFFT is a drug use screening tool [18,19]. Because of the prevalence of tobacco used in Uruguay, a variable indicating whether participants were current smokers was created based on participants' answers to the substances queried by the CRAFFT.

Mean lead levels: Participants' mean blood level levels were assessed in 2001 and in 2011.

Employment status: Adolescent participants were asked if they were currently enrolled in school, working, or unemployed.

Grade in school: A single question asked adolescent participants to report on whether they were attending primary or secondary school.

Health Co morbidities: The health concerns that participants were asked to report on include hypertension, neurologic problems, or psychiatric disorders. The presence of health concerns was coded as a binary where '1' indicated the presence of hypertension, neurologic, or psychiatric comorbidities and where '0' indicated no reported comorbidities.

Covariates: Age and gender were also assessed.

Data analysis

To examine the influence of lead levels in childhood and adolescence on whether participants were current smokers or not, as measured by the CRAFFT screening tool, mean comparisons of childhood and adolescent lead were carried out.

RESULTS

The resulting sample of participants who had complete data in childhood (in 2001) and adolescence (2011) included 89 participants (mean age 14.6, 57% male). Over half of the sample in 2011 (60.7%) included participants who were in secondary school. Among the total sample, 83.2% were exclusively students, 11.2% were employed, and 5.6% were not employed or enrolled in school. Participants reported having the following co-morbid health conditions: hypertension (3.4%), neurologic disorders (3.4%), psychiatric disorders (4.5%), none (19.1%), and other (69.7%).

Over 90% of the sample had BLL above 10μg/dl in 2001 with mean BLL at 14.2μg/dl. During adolescence mean BLL were 5.04 μg/dl. About 5% of the adolescents reported smoking, 41.6% reported using alcohol and 9% used marijuana and 9 % of the sample received a score of 2 or higher on the CRAFFT (Table 1).

Adolescents who were smokers were older (mean age 17.4) than those who were non-smokers (mean age = 14.5; p=0.006). Smokers had higher mean 2011 lead levels (mean lead = 8.68 μg/dl) than non-smokers (mean lead=4.83; p=0.011). However, adolescents who reported smoking in 2011 did not have higher

Table 1: Demographic characteristics among adolescents exposed to lead in childhood (n=89).

Demographic Variables	Mean (SD)/%	Range
Age	14.69 (2.28)	11-18
Sex (Female)	43.82%	
Percent in primaryschool	39.33%	
Percent in secondaryschool	60.67%	
Notemployed	5.62%	
Employed	11.24%	
Student	83.15%	
Healthcomorbidities:		
Hypertension	3.37%	
Neurologic	3.37%	
Psychiatric	4.49%	
None	19.10%	
Other	69.66%	
Substance Use Variables		
Percentwhocurrentlysmoke	5.62%	
CRAFFT total points	0.39 (0.95)	0-5
Percent with Score of 0 on CRAFFT	78.65%	
Percent with Score of 1 on CRAFFT	12.36%	
Percent with Score of 2 on CRAFFT	4.49%	
Percent with Score of 3 on CRAFFT	1.12%	
Percent with Score of 4 on CRAFFT	2.25%	
Percent with Score of 5 on CRAFFT	1.12%	
Percentreportingusing alcohol	41.57%	
Percentreportingusingmarijuana	8.99%	
Pb levels in 2001 (ug/dl)	14.27	
Pb levels in 2011 (ug/dl)	5.04	

Table 2: Comparisons between adolescent smokers and non-smokers exposed to lead in

	Smokers	Non-Smokers	Significance Level	
	Mean (SD)/%	Mean (SD)/%		Mean (SD)/%
Age	17.4 (0.89)			0.006
Percent female	25%			ns
Percent in Secondary school	80%	60%	ns	
Pb levels in 2011 (ug/dl)	8.68			0.011
Pb levels in 2001 (ug/dl)	14.48			ns
Health Comorbidities	40%			0.036

childhood lead levels (mean childhood lead=14.48) than non-smokers (mean childhood lead=14.25, ns). The proportion of adolescents who had health comorbidities did differ between smokers (40%) and non-smokers (10%; p=0.036). Adolescents did not differ on the percent female based on current smoking status or in the percent enrolled in secondary school, see (Table 2).

DISCUSSION

This study reports on findings on the tobacco use of adolescents who had elevated blood level levels during childhood in Uruguay. In addition to the previously known effects of lead on neural development and subsequent behavior problems, the preliminary results of this study suggest that smoking may lead to higher lead levels in adolescence. This finding is consistent with previous work suggesting that tobacco itself may contribute to current lead levels [20]. It has been established that tobacco contains lead, among other chemical compounds, and individuals who smoke tobacco have higher blood lead levels than non-smokers [21, 22]. Previous studies have found that non-smoking children exposed to second-hand smoke have higher lead levels [23]. Together with the findings in this study, there is enough evidence to suggest that future research to clarify tobacco's contribution to lead levels is therefore warranted to expose the etiological mechanism between lead, exposure to tobacco through smoking, and the simultaneous use of other drugs. Future lines of inquiry may focus on exploring whether the adverse health effects of tobacco smoking are exacerbated when individuals have been previously exposed to lead in the environment, whether smoking cessation attempts among these individuals are successful, and whether environmental lead exposure acts as a risk factor for the early initiation into tobacco use.

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REFERENCES

1. Uruguayan Drug Observatory. "De ruidos y nueces". Consumption of legal and illegal drugs in adolescence. National Drug Secretariat. Montevideo, Uruguay. 2012.
2. Uruguayan Drug Observatory. Fifth National High School Survey of Drug Consumption, 2011.National Drug Secretariat. Montevideo, Uruguay. 2012.
3. Uruguayan Drug Observatory. Fifth National Household Survey of Drug Consumption, 2011, National Drug Secretariat. Montevideo, Uruguay. 2012.
4. CICAD/OEA, Report Drug Use in the Americas, 2011. Inter-American Drug Abuse Control Commission.Organization of American States. Washington DC, 2012.
5. Abascal W, Esteves E, Goja B, González Mora F, Lorenzo A, Sica A, et al. Tobacco control campaign in Uruguay: a population-based trend analysis. Lancet. 2012; 380: 1575-1582.
6. WHO, W.H.O., ed. Environmental health criteria 165: Inorganic lead. W.H.O. International programme on chemical safety. 1995: Geneva, Switzerland.
7. Naicker N, Richter L, Mathee A, Becker P, Norris SA. Environmental lead exposure and socio-behavioural adjustment in the early teens: The birth to twenty cohort. Sci Total Environ. 2012; 414: 120-125.
8. Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL. Early exposure to lead and juvenile delinquency. Neurotoxicol Teratol. 2001; 23: 511-518.
9. Bellinger DC. Neurological and behavioral consequences of childhood lead exposure. PLoS Med. 2008; 5: e115.
10. Lane SD, Webster NJ, Levandowski BA, Rubinstein RA, Keefe RH, Wojtowycz MA, et al. Environmental injustice: childhood lead poisoning, teen pregnancy, and tobacco. J Adolesc Health. 2008; 42: 43-49.
11. Meyer PA, Brown MJ, Falk H. Global approach to reducing lead exposure and poisoning. Mutat Res. 2008; 659:166-175.
12. Mañay N, Cousillas AZ, Alvarez C, Heller T. Lead contamination in Uruguay: the "La Teja" neighborhood case. Rev Environ Contam Toxicol. 2008; 195: 93-115.
13. American Academy of Pediatrics Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. Pediatrics. 2005; 116: 1036-1046.
14. Romieu I, Lacasana M, McConnell R. Lead exposure in Latin America and the Caribbean. Lead Research Group of the Pan-American Health Organization. Environ Health Perspect. 1997; 105: 398-405.
15. Mañay N, Pereira L, Cousillas Z. Lead contamination in Uruguay. Rev Environ Contam Toxicol. 1999; 159: 25-39.
16. Abelsohn AR, Sanborn M. Lead and children: clinical management for family physicians. Can Fam Physician. 2010; 56: 531-535.
17. Centers for Disease Control and Prevention. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in "Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention"
18. Knight JR, Sherritt L, Shrier LA, Harris SK, Chang G. Validity of the CRAFFT substance abuse screening test among adolescent clinic patients. Arch Pediatr Adolesc Med. 2002; 156: 607-614.
19. PerezGomez A, Scoppetta O. El CRAFFT/CARLOS como instrumento para la identificación temprana de consumo de alcohol y otras SPA:

Una adaptación al español. Rev.colomb.psicol. 2011; 20:265-274.

20. Grasmick C, Huel G, Moreau T, Sarmini H. The combined effect of tobacco and alcohol consumption on the level of lead and cadmium in blood. Sci Total Environ. 1985; 41: 207-217.

21. Chiba M, Masironi R.Toxic and trace elements in tobacco and tobacco smoke. Bull World Health Organ. 1992; 70: 269-275.

22. Mannino DM, Homa DM, Matte T, Hernandez-Avila M. Active and passive smoking and blood lead levels in U.S. adults: data from the Third National Health and Nutrition Examination Survey. Nicotine Tob Res. 2005; 7: 557-564.

23. Mannino DM, Albalak R, Grosse S, Repace J. Second-hand smoke exposure and blood lead levels in U.S. children. Epidemiology. 2003; 14: 719-727.

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