GLOBAL JOURNAL OF MEDICINE AND PUBLIC HEALTH

Smoking – its imitative hand-oral behaviour and ingestion thereby of environmental toxins like lead (Pb)

David J Vance[*]

ABSTRACT

Objective

To examine the hypothesis that children imitate the non-food pseudo-ingestive hand-oral action of smoking, increasing their ingestion of lead (Pb).

Method

Using archival data from the Australian Institute of Health and Welfare's (AIHW) National Survey of Lead in Children (NSLC), two families (linear and logistic) of multiple regression equations were examined as to what extent interaction variables formulated from measures or proxies of exposure to environmental lead (age of child, time spent in environment (each of); with (each of) house-dust lead, yard-soil lead, dwelling age and flaking-paint lead; and, (each of, as the ultimate comparison) parentally-reported child soil-eating, or, smoking by others in the child's environment) would, with all subordinate constituent simple variables or interaction variables preliminarily entered into the regression model(s), be accepted into or not the multiple regression equation(s) predicting child blood lead.

Results

Comparison of the statistical significances of each interaction Independent Variable in its respective regression equation found that reported smoking by persons in the child's household was better than reported child soil-eating as a predictor of child blood lead.

Conclusions and Implications

The results support the hypothesis that children imitate the non-food pseudo-ingestive hand-oral action of smoking, increasing their ingestion of contaminant lead in their environment. Furthermore, that it is very likely that the same increase of ingestion, and even inhalation, of other environmental contaminants, particularly as are still more common in the Developing World, would apply.

GJMEDPH 2016; Vol. 5, issue 3

***Corresponding Author:**
David J Vance
Independent Consultant
davidjmvance@hotmail.com
Telephone Number: +610407635579

Conflict of Interest—none

Funding—Research Grants from Australian Institute of Health and Welfare

INTRODUCTION

Lead intoxication of children at the higher levels classically (in Developed nations) resulting from ingestion of high-lead paint is known to frequently cause sequelae including brain damage and decrement in mental abilities.[1-5]

Now in 2016 in the Developing World environmental contaminants of similar concern to what Pb was in Developed countries decades ago in the era of peak lead-petrol consumption and lead-paint flaking and even now still is near some mines and mineral processing plants as well as some remaining older

housing, include Pb and other heavy metals, and the health consequences of these are far from trivial.[6-13]

Tobacco smoking is now highly prevalent and increasing in the Developing world, with prevalences of up to 40%, and tobacco companies focusing their attention on habituating anyone there having disposable income to their health-destroying products.[14-18]

Over the last few decades the question of what the threshold of lead intoxication, below which decrement in mental abilities was unlikely to ensue, might be has been the subject of much study, including epidemiological,[19-24] animal-model experimental,[25-28] and in-vitro biochemical-physiological.[29-35]

The collective meaning of such works includes that an association between quite low levels of lead intoxication, and reduced mental ability, has been well shown, even though it may not be claimed that this association has been shown to be free of confounding by the action of other quite plausible factors such as psychosocial or genetic.[36]

Notwithstanding the lack of definite proof of causality of child blood lead levels of < 0.48 uM (< 10 ug/dL) and lowered IQ, on balance including conclusive causation of higher blood Pb levels and more severe outcomes including death, there has been a general consensus that cost-benefit estimations indicated that the reduction of lead intoxication in young children was a goal worth pursuing. [8, 9, 13]

The nature of the various sources of the lead involved in human urban lead intoxication has also been the subject of much study, with the major sources and many minor sources having been long known,[37] and including all of the aspects detailed below in the Data Collection section. The relative magnitude of the contributions amongst these to child lead acquisition has not been adequately quantified using techniques such as multiple regression data analysis methodology (most of the minor sources are sufficiently rare as to not be feasible of analysis by this methodology).

At the time of the execution of the Australian Institute of Health and Welfare (AIHW)'s National Survey of Lead in Children (NSLC),[38] however, it was generally thought that across the population there would be an extremely close correlation between (lower) lead levels in human blood and the level of lead pollution originating from the burning of leaded petrol by motor vehicles, by appropriate extrapolation of the information provided by UK and USA studies.[39-42] Higher (particularly at the highest) levels in Australian human child blood predominantly result from ingestion of leaded paint in housing constructed in the era of the use of high-lead paint, and (less particularly the highest) from proximity to particular lead-involving industrial activities, notably mining and smelting.[19, 18, 43-47]

It has been noted in other works that smoking is associated with blood lead in children, but due to a lack of behavioural science acumen and a failure to properly estimate the likely contribution of cigarette smoking to lead pollution of the environment (which in nearly all cases is trivial), this has been erroneously attributed to the latter.[48]

Young children are highly imitative of older humans, and the younger the child the more generalized is this imitation.[49, 50] The hypothesis that smoking by members of the household functioned rather as a source of non-food hand-oral pseudo-ingestive behaviour that the child generally imitated, thereby increasing the amount of the environmental lead ingested from these activities, was investigated.

METHODS

The National Survey of Lead in Children (NSLC)[38] was carried out by the Australian Institute of Health and Welfare (AIHW) in early 1995. This was a stratified, randomized, cross-sectional study of Australian children aged 1 to 4 years (inclusive), which incorporated venous blood lead assay, environmental lead assays, and the gathering of questionnaire data on various environmental, occupational, social, behavioural, and other factors.

Participants

To meet the requirements for some compromise between the need on the one hand to minimise

resource requirements, and on the other to minimise intra-class correlation, the 27,673 Census Collector Districts (CDs) that the Australian Bureau of Statistics (ABS) has formulated (by pooling some of the more sparsely-populated of the original 31,404) for the ABS Labour Force Surveys (a national general data acquisition program) were used as the sampling frame basically as follows: Randomly, (other than exclusion on the basis of involvement in the ABS Labour Force Survey) 1.2% (316) of the non-remote CDs were selected, and each of these contributed one geo-demographical third, while (likewise randomly) 0.4% (13) of the remote CDs were selected, each of these contributing it's entire population. Minor deviations from this included a 2% over-representation of remote CDs. This sampling frame theoretically contained approximately 4,000 children aged 1-4 years, and the number who eventually supplied blood samples was 1,575 (35%), from whom only 757 (17%) had datasets that were complete with respect to the assaying of the environmental lead samples (yard soil, house dust, drinking water), and did not contain conflicting information. 30 of the 757 were however, allocated a middle income category in lieu of missing information, on the grounds that we were not particularly interested in this variable per se, it's association having been well established long ago.

This final sample was constituted, purportedly due to limitations of time permitting only a subset of the intended laboratory analysis of environmental sample lead, by all of the 304 (out of the group of 1,575) children from South Australia, Tasmania, the Australian Capital Territory, and the Northern Territory; by 85 out of 115 cases (from any state) with blood lead >0.48 umol/L; and the balance being a systematic random sample from the remainder of the other 4 states: Queensland, New South Wales, Victoria and Western Australia.

All survey activities were endorsed by the Ethics Committee of the AIHW in accordance with its standard practices, participation being voluntary, with ability to withdraw at any stage. This is in accordance with the ethical standards of the APA.

Data Collection

The information acquired from the sample included blood lead levels, yard soil lead levels, house dust lead levels, drinking water lead levels, peeling/flaking paint lead levels, age, position in family, family structure, proximity to main roads, ethnicity, wall paint condition, dwelling structure, education, income, motor vehicle ownership and leaded petrol applicability of these, smoking, dog ownership, cat ownership, vegetable garden ownership; occupation in: building, scrap metal, paint removal, road construction/maintenance, battery work, lead smelting, lead mining, brass-work, solder-work, glass manufacture, automotive repair, panel beating; hobby involvement in: home renovation involving removal of old paint, pottery or ceramic making, painting china, staining glass, playing games with lead models, making fishing sinkers, shooting, panel beating or spray painting of cars, automotive repairs; paint removal at the dwelling, presence during such, drinking water source, roofing material, dwelling ownership, finger/thumb sucking, toy sucking/chewing, soil eating, minding away from home, and date of dwelling construction.

Statistical Analysis

The association between parentally-reported smoking by others in the (1-4 year old) child's household and parentally-reported frequency of soil-eating by the child was firstly examined. Parentally-reported smoking by others in the child's household was also compared with parentally-reported frequency of soil-eating by the child, as a predictor of levels of blood lead, when used in interaction with environmental lead measures.

An assessment was made of the possible interaction between (respectively) these two variables (parentally-reported smoking in the residence, parentally-reported soil-eating frequency); and each of the four 'proxy' measures of environmental lead (house-dust lead, yard-soil lead, age of the dwelling, and flaking-paint lead); and none, one of, or both of, the other two variables that could theoretically contribute to interactions of interest (youth i.e. age of the child

reversed, and, the amount of time spent at the residential address).

The interaction terms were manually constructed from the simple terms by the multiplication of numerical information that had been adjusted as close to normality as feasible and to increase in theoretical risk in the same direction, and the interaction terms were then adjusted as close to normality as feasible, most often by the taking of some root.

Each interaction term was made available (in its respective regression analysis) for acceptance into the model, in a forward stepwise manner, alongside the other variables that had been selected for consideration (on the basis of statistically significant bivariate association with blood lead), with all the respective subordinate constituent terms (simple, and interaction also in the case of second or third order interaction terms being examined), of the respective interaction variable, preliminarily entered into the respective model.

Regression analyses were done using both linear and logistic methods, with the logistic method using a DV cut-off point of >0.48 uM (n = 85) / <0.48 uM (n = 673) blood Pb.

RESULTS

Summary Findings of the NSLC

The NSLC found that the blood lead levels in the group of children (approximately 35% of the target group) from which information was acquired was: arithmetic mean 0.28 uM (6 ug/dL), geometric mean 0.24 uM (5ug/dL), 7% > 0.49 uM (1O ug/dL), 2% > 0.73uM (15 ug/dL), maximum 1.58 uM (33 ug/dL). The authors stated that this was appreciably less than what had been expected on the basis of the very limited (note exclusively) Australian previous data. The target set by the NHMRC in 1993, 90% of children from 1 to 4 years of age to have a blood lead <0.48 umol/L by the end of 1998, had therefore been surpassed.

A number of factors were found to be associated with blood lead level, and with the exception of interviewer-assessed dwelling cleanliness, all had been reported previously, and most of these many times, e.g. by the Detroit Dept. Health in 1980 [37]. These factors included education, income, age of dwelling, peeling interior paint, house dust lead, yard soil lead, soil-eating, smoking, ceramic painting, lead-lighting, lead smelting, scrap metal work, solder work, ownership of leaded-petrol using vehicles.

Comparison of Smoking Presence and Soil-Eating

There was a non-significant positive association seen between the reported presence of a smoker in the household and the reported frequency of soil-eating by the child (p = 0.25). Due to the existence of possible psychological confounders (i.e. biased reporting) in this lack of statistical significance of association, this lack was thought not to preclude the carrying-out of the examination of the interaction terms of interest, but rather to add through default to the utility of examining the hypothesis.

The relevant results of the examinations of the interaction terms are presented as tabulation of only the statistical significance for entry into its respective regression model of the interaction term of focal interest, and also (in their respective regression models) of any subordinate constituent interaction IVs (some of these having only peripheral non-focal interest and therefore being presented only among the results for the first environmental lead proxy and not repeated among the results for the other three, as they are common to all four). These are presented (see Tables 1 and 2 in appendix) in such a way as to facilitate ready comparison as intended, by first separating on the basis of which of the four proxy measures of environmental Pb was incorporated into the interaction term, and then separating on the basis of which of the two focal IVs (reported smoker presence in the household, or, reported child lead-eating) were incorporated into the interaction, and also when neither was.

In the stepwise building of the most meaningful (guided with appropriate exclusions of IVs via reference to a cross-correlation matrix in conjunction with other knowledge) linear regression model, with the subordinate constituents (of the two interaction IVs that had been accepted into the model in earlier equitable contests from among those having $p < 0.10$) preliminarily entered into the model, the stepwise order of the IVs thereafter being accepted into the model was: house-dust Pb, reported child soil-eating frequency, domestic water Pb, financial income, reported smoker presence x yard-soil Pb x child minded at home, an ordinal Pb hobby IV, house cleanliness, aboriginality, and, active removal of domestic paint.

As noted, the relevant results of the examinations of the interaction terms are presented as tabulation of the statistical significance for entry into the respective regression model of the interaction terms of interest, and also of any subordinate constituent interaction terms (Tables 1 and 2). A comparison of the respective statistical significances thus measured for the interaction terms of focal interest (those of $p < 0.10$ are bolded) was made visually.

It can be seen that amongst the linear regression results in Table 1, comparing those interaction IVs having as their constituent simple IVs at least reported child soil-eating and one of the four environmental Pb proxies, with those having as their constituent simple IVs at least reported smoker presence and one of the four environmental Pb proxies, whether considering only the p values < 0.10, or all p values, that the group with reported smoker presence is a substantially better predictor of the child blood Pb DV than the group with reported child soil-eating. This observation is sufficient in and of itself to support the hypothesis, without any recourse to further statistical tests being necessary.

On the other hand, presented here also for proper rigour, it can be seen that amongst the analogous logistic regression results in Table 2, the group with reported child soil-eating is a better predictor of the child blood Pb DV than the group with reported smoker presence, but this is much less substantial and consistent than the association in the linear regression series. Furthermore, that collinearity and zero-cell problems severely afflicted the flaking-paint Pb series was evidenced by the peculiar behavior of the p values across orders of interaction, and diagnostically confirmed by the standard errors of the regression coefficients being larger than the coefficients themselves, so these few results were meaningless and therefore not rightly reportable.

Also, whereas the series of the other three environmental Pb proxies were not so afflicted to that extent, because they were continuous variables (flaking-paint Pb being a dichotomous variable), doubtlessly low numbers played some role in the general failure of the third order interaction IVs to achieve statistical significance in the face of very strong theoretical reasons that they might do so, as was evidenced by the examination of the distribution histograms during the normalizing transformations. Therefore, the results of the logistic regression series carries less weight than those of the linear regression series. In any case this (mild) reversal of direction of association does not in itself negate the (substantially numerically stronger) support of the hypothesis that the linear regression series had, because the linear and logistic DVs are not equivalent.

Moreover, in any case, if the magnitudes of the results of the linear and logistic regressions were to be combined into an average it can be seen by eyeball that the net direction of association would still be in the direction strongly supporting the hypothesis, as mere equivalency alone in the associations of reported smoking and of reported child soil-eating is sufficient for support of the hypothesis, and that equivalency is here well exceeded.

Table 1 Linear Regression p-value for admission of a Particular Interaction Term into the Respective Model, with all Subordinate Simple and Interaction Terms Preliminarily Entered*

Simple Constituents	1st Order Interactions	2nd Order Interactions	3rd Order Interactions
Floor Dust Pb	Dust.Age .335	Dust.Age.Mind .922	
Child Age Reverse	Dust.Mind .147		
Minded in Home	Age.Mind .493		
	Eat.Age .435	Eat.Age.Mind .758	
Eating Soil	Eat.Mind .230	Eat.Dust.Age .961	
	Eat.Dust .531	Eat.Dust.Mind **.053**	Eat.Dust.Age.Mind .884
	Smoke.Age .373	Smoke.Age.Mind .704	
Smoker Present	Smoke.Mind .230	Smoke.Dust.Age .744	
	Smoke.Dust .969	Smoke.Dust.Mind .791	Smoke.Dust.Age.Mind .577
Home Age	Hmag.Age .524	Hmag.Age.Mind .016	
Child Age Reverse	Hmag.Mind		
Minded in Home	Age.Mind (As Above)		
Eating Soil	Eat.Hmag .094	Eat.Hmag.Age .442	
		Eat.Hmag.Mind .930	Eat.Hmag.Age.Mind .595
Smoker Present	Smoke.Hmag .531	Smoke.Hmag.Age .550	
		Smoke.Hmag.Mind **.001**	Smoke.Hmag.Age.Mind .495
Yard Soil Pb	Soil.Age .362	Soil.Age.Mind .721	
Child Age Reverse	Soil.Mind .510		
Minded at Home	Age.Mind (As Above)		
Eating Soil	Eat.Soil .307	Eat.Soil.Age .301	
		Eat.Soil.Mind .755	Eat.Soil.Age.Mind .297
Smoker Present	Smoke.Soil .499	Smoke.Soil.Age .104	
		Smoke.Soil.Mind **.005**	Smoke.Soil.Age.Mind .222
Flaking Paint Pb	Flake.Age .590	Flake.Age.Mind .723	
Child Age Reverse	Flake.Mind .756		
Minded at Home	Age.Mind (as Above)		
Eating Soil	Eat.Flake .886	Eat.Flake.Age .552	
		Eat.Flake.Mind .456	Eat.Flake.Age.Mind .589
Smoker Present	Smoke.Flake **.088**	Smoke.Flake.Age .970	
		Smoke.Flake.Mind .814	Smoke.Flake.Age.Mind .577

***Abbreviation codes: 'Dust' = house floor dust Pb level, 'Age' = the age of the child, reversed to 'youth' of the child, 'Mind' = minding of the child at home ie not elsewhere, 'Eat' = soil-eating by child, 'Smoke' or `Smo' = smoker present in home, `Hmag' = age of the home, 'Soil' = house yard soil Pb level, 'Flake' or `Flk' = Pb level of (any) flaking paint, 'as above' = term is a common constituent and is already presented in the group of results concerning the house floor dust Pb, in the analogous position**

Table 2 Logistic Regression p-values for admission of a particular interaction term into the model, with all subordinate simple and interaction terms preliminary entered, Blood Pb cut at >0.48µM (n=85), <0.48 µM (n=673)*

Simple Constituents	1st Order Interactions	2nd Order Interactions	3rd Order Interactions
Floor Dust Pb	Dust.Age .671	Dust.Age.Mind .379	
Child Age Reverse	Dust.Mind .331		
Minded in Home	Age.Mind .215		
	Eat.Age .005	Eat.Age.Mind .761	
Eating Soil	Eat.Mind .647	Eat.Dust.Age .674	
	Eat.Dust .335	Eat.Dust.Mind **.022**	Eat.Dust.Age.Mind .091
	Smoke.Age .615	Smoke.Age.Mind .140	
Smoker Present	Smoke.Mind .622	Smoke.Dust.Age .744	
	Smoke.Dust .372	Smoke.Dust.Mind .609	Smoke.Dust.Age.Mind .675
Home Age	Hmag.Age .483	Hmag.Age.Mind .834	
Child Age Reverse	Hmag.Mind .701		
Minded in Home	Age.Mind (As Above)		
Eating Soil	Eat.Hmag .780	Eat.Hmag.Age .700	
		Eat.Hmag.Mind .496	Eat.Hmag.Age.Mind .658
Smoker Present	Smoke.Hmag .246	Smoke.Hmag.Age .175	
		Smoke.Hmag.Mind **.088**	Smoke.Hmag.Age.Mind .798
Yard Soil Pb	Soil.Age .606	Soil.Age.Mind .100	
Child Age Reverse	Soil.Mind .647		
Minded at Home	Age.Mind (As Above)		
Eating Soil	Eat.Soil .608	Eat.Soil.Age .408	
		Eat.Soil.Mind .198	Eat.Soil.Age.Mind .676
Smoker Present	Smoke.Soil .211	Smoke.Soil.Age .718	
		Smoke.Soil.Mind .263	Smoke.Soil.Age.Mind .241
Flaking Paint Pb	Flake.Age .595	Collinearity	Zero Cell Problems
Child Age Reverse	Flake.Mind .857		
Minded at Home	Age.Mind (as Above)		
Eating Soil	Eat.Flake .381		
Smoker Present	Smoke.Flake .379		

***Abbreviation codes: 'Dust' = house floor dust Pb level, 'Age' = the age of the child, reversed to 'youth' of the child, 'Mind' = minding of the child at home ie not elsewhere, 'Eat' = soil-eating by child, 'Smoke' or `Smo' = smoker present in home, `Hmag' = age of the home, 'Soil' = house yard soil Pb level, 'Flake' or `Flk' = Pb level of (any) flaking paint, 'as above' = term is a common constituent and is already presented in the group of results concerning the house floor dust Pb, in the analogous position**

DISCUSSION

Parentally-reported smoking by others in the (1-4 year-old) child's household appeared to be substantially better than parentally-reported frequency of soil-eating by the child, when used in interaction with lead in the child's home environment, as a predictor of child blood lead. This is not what would have been expected if smoking had contributed only through raising environmental Pb, which, as smoking contributes relatively trivial amounts of Pb to a domestic environment, would have been a very substantial reversal of comparative direction of the associations observed.

That the interaction(s) of focal interest here (apart from any of its subordinate constituents, recall) competed so well with the other well-established factors for acceptance into the stepwise most meaningful linear multiple regression model, as

detailed at the end of the results section above, is fair evidence of a non-trivial role of smoking-imitative non-food hand-oral pseudo-ingestive behaviour in Pb toxicology in young children.

Initial inspection of the models built during the respective stepwise processes showed these to be quite stable, and that therefore the preliminary entering of the lower-order constituent terms was highly unlikely to have produced any spurious associations impacting on the aspect of interest.

Note also, that the preliminary entering of subordinate constituents into the model is a very strong foil to the confounding of associations with higher order IV, which may then only occur at the interaction level of the IV of interest and not at any lower level, notwithstanding the possibility that something may be preliminarily causal to all subordinate constituents. Any proposed confounding must be plausible as the interaction, and not only between any subordinate constituents, to constitute a substantial gainsay to the indication of associations here reported.

It was concluded that the hypothesis, that children would imitate the non-food hand-oral action that is smoking, in a generalized way that would lead to an increase in the ingestion of lead from the domestic environment, was solidly supported.

It is further suggested that this finding has ramifications that possibly extend to other oral phenomena, such that the observation of smoking by role models may result in an increase in intake of oral intoxicants and a replacement of higher quality organically-shaped wholefoods with lower-quality synthetically-shaped processed foods. This is notwithstanding the sometimes somewhat similar outcomes engendered by cognitive processes involving abstract thought, that come increasingly into play with the aging of children.

ACKNOWLEDGEMENT

This study was supported in part by the Australian Institute of Health and Welfare, Canberra, Australia. Neither the Australian Institute of Health and Welfare nor the Australian Bureau of Statistics is responsible for the analyses or interpretations of data in this study.

REFERENCES

1. Fejerman N, Gimenez ER, Vallejo NE, Medina CS. Lennox's syndrome and lead intoxication. Pediatrics, 1973; 52: 227-34.
2. Chisolm JJ Jr. Increased lead absorption: toxicological considerations. Pediatrics, 1971; 48: 349-52.
3. Perlstein MA, Attala R. Neurological sequelae of plumbism in children. Clin Ped, 1966; 5: 292-8.
4. Chisolm JJ Jr, Harrison HE. The exposure of children to lead. Pediatrics, 1956; 18: 943¬-57.
5. Mellins RB, Jenkins CD. Epidemiological and psychological study of lead poisoning in children. JAMA, 1955; 158: 15-20.
6. Wang MD, Gomes J, Cashman NR, Little J, Krewski D. A meta-analysis of observational studies of the association between chronic occupational exposure to lead and amyotrophic lateral sclerosis. J Occup Environ Med, 2014; Dec 56(12): 1235–1242. Published online 2014 Dec 8 doi: 10.1097/JOM.0000000000000323
7. Ahmad SA, Khan MH, Khandker S, Sarwar AFM, Yasmin N, Faruquee MH, Yasmin R. Blood lead levels and health problems of lead acid battery workers in Bangladesh. Scientific World J, 2014; 2014; 974104. Published online 2014 Feb 25. doi: 10.1155/2014/974104
8. Van der Kuijp TJ, Huang L, Cherry CR. Health hazards of China's lead-acid battery industry: a review of its market drivers, production processes, and health impacts. Environ Health, 2013; 12: 61. Published online 2013; Aug 3. doi: 10.1186/1476-069X-12-61
9. Attina TM, Trasande L. Economic costs of childhood lead exposure in low- and middle-income countries. Environ Health Perspect, 2013; Sep, 121(9): 1097–1102. Published online 2013; Jun 8, .doi: 10.1289/ehp.1206424
10. Lo YC, Dooyema CA, Neri A, Durant J, Jefferies T, Medina-Marino A, de Ravello L, Thoroughman D, Davis L, Dankoli RS, Samson MY, Ibrahim LM, Okechukwu O, Umar-Tsafe NT, Dama AH, Brown MJ. Childhood lead poisoning associated with gold ore processing: a village-level investigation—Zamfara State, Nigeria, October–November 2010. Environ Health Perspect, 2012; October; 120(10): 1450–1455. Published online 2012 Jul 5 doi: 10.1289/ehp.1104793

11. Dooyema CA, Neri A, Lo YC, Durant J, Dargan PI, Swarthout T, Biya O, Gidado SO, Haladu S, Sani-Gwarzo N, Nguku PM, Akpan H, Idris S, Bashir AM, Brown MJ.Outbreak of fatal childhood lead poisoning related to artisanal gold mining in northwestern Nigeria, 2010. Environ Health Perspect, 2012; Apr, 120(4): 601–607. Published online 2011 Dec 20. doi: 10.1289/ehp.1103965
12. Haefliger P, Mathieu-Nolf M, Lociciro S, Ndiaye C, Coly M, Diouf A, Faye AL, Sow A, Tempowski J, Pronczuk J, Filipe AF Jnr, Bertollini R, Neira M Mass lead intoxication from Informal used lead-acid battery recycling in Dakar, Senegal. Environ Health Perspect, 2009 October; 117(10): 1535–1540. Published online 2009 May 14. doi: 10.1289/ehp.0900696
13. Briggs D. Environmental pollution and the global burden of disease. Brit Med Bull, 2003; 68: 1-24.
14. Brathwaite R, Addo J, Smeeth L, Lock K. A systematic review of tobacco smoking prevalence and description of tobacco control strategies in sub-Saharan African countries; 2007 to 2014. PLoS One, 2015; 10(7): e0132401. Published online 2015 July 10. doi: 10.1371/journal.pone.0132401
15. Nikovska DG, Spasovski M, Gjorgjev D, Bislimovska JK, Isjanovska R, Tozija F. Social determinants of smoking in the population of the Republic of Macedonia – results from a nested case-control study. GJMEDPH, 2014; 3: 4. Available from: http://www.gjmedph.org/uploads/O3%20-V03N04.pdf
16. Fu SH, Jha P, Gupta PC, Kumar R, Dikshit R, Sinha D. Geospatial analysis on the distributions of tobacco smoking and alcohol drinking in India. PLoS One, 2014; 9(7): e102416. Published online 2014 July doi: 10.1371/journal.pone.0102416
17. Savadi P, Wantamutte AS, Narasannavar A. Pattern of tobacco use among primary school teachers in Belgaum city, India – A Cross Sectional Study. GJMEDPH, 2013; 2: 4. Available from: http://www.gjmedph.org/uploads/O8-V02N04.pdf
18. Das NK, Ghosh K, Sarkar GN, Shyamapada Paul S. Determinants of smoking and chewing habits among rural school children in Bankura district of West Bengal, India. GJMEDPH, 2012; 1: 6. Available from: http://www.gjmedph.org/uploads/O2_V01N06.pdf
19. McMichael AJ, Baghurst PA, Vimpani GV, Wigg NR, Robertson EF, Tong SL. Tooth lead levels and IQ in school-age children: the Port Pirie Cohort Study. Am J Epidemiol, 1994; 140: 489-99.
20. Needleman HL, Schell A, Bellinger D, Leviton A, Allred E. The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. N Engl J Med, 1990; 322: 83-8.
21. Needleman, H. L., Gatsonis, C. A. (1990). Low-level lead exposure and the IQ of children A meta-analysis of modern studies. JAMA, 263, 673-678.
22. McMichael AJ, Baghurst PA, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ. Port Pirie Cohort Study: environmental exposure to lead and children's abilities at the age of four years. N Engl J Med, 1988; 319: 468-75.
23. Wigg NR, Vimpani GV, McMichael AJ, Baghurst PA, Robertson EF, Roberts RJ. Port Pirie Cohort Study: childhood blood lead and neuropsychological development at age two years. J Epidemiol Public Health, 1988; 42: 213-19.
24. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Engl J Med, 1979; 300: 689-95.
25. Kitchen I. Lead toxicity and alterations in opioid systems. Neurotoxicology, 1993;14: 115- 24.
26. Kitchen I, Kelly M. Effect of perinatal lead treatment on morphine dependence in the adult rat. Neurotoxicology, 1993; 14: 125-30.
27. Winneke G. Animal studies. In: Part three: The effects of lead. In R. Lansdown, W. Yule (Eds.), The lead debate: the environment, toxicology and child health, 1986: 217-34. London: Croom Helm.
28. Winneke G, Brockhaus A, Baltissen R.. Neurobehavioral and systemic effects of longterm blood lead-elevation in rats. Arch Toxicol, 1977; 37: 247-63.
29. Goldstein GW. Evidence that lead acts as a calcium substitute in second messenger metabolism. Neurotoxicology, 1993; 14: 97-102.
30. Murakami K, Feng G, Chen SG.. Inhibition of brain protein kinase C subtypes by lead. J Pharmacol Exp Ther, 1993; 264: 757-61.
31. Regan CM. Neural cell adhesion molecules, neuronal developMent and lead toxicity. Neurotoxicology, 1993; 14: 69-74
32. Simons JB. Lead-calcium interactions in cellular lead toxicity. Neurotoxicology, 1993; 14: 77-86.

33. Tomsig JL, Suszkiw JB. Intracellular mechanism of Pb2+-induced norepinephrine release from bovine chromaffin cells. Am J Physiol, 1993; 265: c1630-6.
34. Malts RL, Schatz JR, Hurst R, Kagen R. Toxic heavy metal ions activate the heme-regulated Eukaryotic Initiation Factor-2alpha Kinase by inhibiting the capacity of hemin-supplemented reticulocyte lysates to reduce disulfide bonds. J Biological Chem, 1991;.266: 12695-702.
35. Raghavan SRV, Culver BD, Gonick HC. Erythrocyte lead-binding protein after occupational exposure 1. Relationship to lead toxicity. Environ Res, 1980; 22: 264-70.
36. Bellinger D, Leviton A, Waternaux C. Lead, IQ and social class. Int J Epidemiol, 1989; 18: 180-5.
37. Detroit Department of Health Lead Poisoning Control Program. Environmental Lead Investigation Questionnaire. In Needleman HL. Low level lead exposure: the clinical implications of current research, 1980:.248-51. Raven .Press.
38. Donovan J. Lead in Australian children: report on the National Survey of Lead in Children, 1996: Australian Institute of Health and Welfare, Canberra.
39. Rabinowitz M, Needleman HL, Burley M, Rees J. Lead in umbilical blood, indoor air, tap water, and gasoline in Boston. Arch Environ Health, 1984; 39: 299-301.
40. Rabinowitz M, Needleman HL. Petrol lead sales and umbilical cord blood lead levels in Boston, Massachusetts. Lancet, 1983; 1: 63.
41. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead level between 1976 and 1980. N Engl J Med, 1983; 308: 1373-7.
42. Kollerstrom N. Lead on the brain A plain guide to Britain's no 1 pollutant. 1982. London: Wildwood House.
43. Chiaradia M, Gulson BL, MacDonald K. Contamination of houses by workers occupationally exposed in a lead-zinc-copper mine and impact on blood lead concentrations in the families. Occup Environ Med, 1997; 54: 117-124.
44. Neville GR. Mt Isa community blood lead data. Report by the Environmental Health Branch, Queensland Health, Australia, 1994.
45. Galvin J, Stephenson J, Wlodarczyk J, Loughran R, Waller G. Living near a lead smelter: an environmental health risk assessment in Boolaroo and Argenton, New South Wales. Aust J Public Health, 1993; 17: 373-8.
46. Baghurst PA, Tong SL, McMichael AJ. Determinants of blood lead concentrations to age 5 years in a birth cohort study of children living in the lead smelting city of Port Pirie and surrounding areas. Arch Environ Health, 1992; 47: 203-10.
47. McMichael AJ, Baghurst PA, Robertson EF, Vimpani GV, Wigg NR. The Port Pirie Cohort Study Blood lead concentrations in early childhood. Med J Australia, 1985; 143: 499-503.
48. Baghurst PA, McMichael AJ, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ, Tong SL. Environmental exposure to lead and children's intelligence at the age of seven years. N Engl J Med, 1992; 327: 1279-84.
49. Shimada S.. Modelling effects and their maintenance on pretend actions in 1- and 2¬year-olds. RIEEC Report, 1987; 36: 119-26.
50. Abravanel E, Gingold H. Learning via observation during the second year of life. Devel Psycho!, 1985; 21: 614-23.