Personality and Individual Differences 137 (2019) 184–191

Contents lists available at ScienceDirect

Personality and Individual Differences

journal homepage: www.elsevier.com/locate/paid

Are the effects of lead exposure linked to the *g* factor? A meta-analysis

Michael A. Woodley of Menie[a,e,*], Jan te Nijenhuis[b,c], Vladimir Shibaev[d], Miao Li[c], Jan Smit[b]

[a] Vrije Universiteit Brussel, Center Leo Apostel for Interdisciplinary Research, Belgium
[b] Work and Organizational Psychology, University of Amsterdam, the Netherlands
[c] Social and Organizational Psychology, Vrije Universiteit, Amsterdam, the Netherlands
[d] Vladivostok State University Economics and Service, Vladivostok, Russia
[e] Unz Foundation Junior Fellow, Palo Alto, CA, USA

ARTICLE INFO

Keywords:
Intelligence
g factor
Lead exposure

ABSTRACT

Does lead reduce IQ at the level of *g*, test specificities, or both? A bare-bones psychometric meta-analysis utilizing the Method of Correlated Vectors was performed on a sample of 16 studies for which subtest-level data could be obtained satisfying stringent inclusion rules. The aggregate correlation across samples between subtest-level estimates of both *g* loading (*g*) and the deleterious impact of lead exposure (*d*) was 0.10 ($K = 16$, total $N = 1935$, 80% CI after correction for sampling error = 0.10 to 0.10). So, lead exposure is associated with a slightly positive vector correlation, which is consistent with the results of other studies examining the effects of other neurotoxins on IQ using MCV; this outcome is consistent with two scenarios. The first is that lead exposure may have effects on both *g* and test specificities owing to systemic effects on many different brain regions. The second is that two antagonistic factors are at work. It might be that the 'control' and exposure groups used in these kinds of studies are confounded with pre-existing differences in *g* – lower *g* being a risk factor for poorer life outcomes (including lower socioeconomic status and concomitantly heightened risk of lead exposure), whereas lead has it's primary effect on the test specificities, with both effects opposing one another, as reflected in the small magnitude vector correlation value. Strategies for distinguishing between these scenarios are discussed.

1. Introduction

1.1. Lead exposure

Intelligence is known to be causally linked to school and work performance (Jensen, 1998; Hunter & Schmidt, 2004; Schmidt & Hunter, 1998), which are crucial factors for success in life. It is important therefore to be mindful of factors that lower intelligence. Lead exposure has been proposed as one of these factors (Nevin, 2000). Lead is a heavy metal, and exposure to lead has a toxic effect on the human body. The main sources of lead are lead-based water taps, lead-based paint in older housing, soil and dust contaminated with leaded paint and gasoline, and past and present mining and industrial activity (Koller, Brown, Spurgeon, & Levy, 2004). Fortunately, much has been done to minimize the use of lead. For example, water pipes no longer contain lead and are now made using nonlead alternatives. Also, whilst lead-based paint is still present in older houses, new paint does not contain lead. However, there continues to be major lead exposure through contact with contaminated soil and dust, and old water taps.

Although blood-lead concentrations have fallen substantially in a number of countries in the last few decades (Meyer, McGeehin, & Falk, 2003; Nevin, 2000), childhood lead poisoning continues to be a major public health problem in many countries. Children are most vulnerable to lead exposure for three reasons: a) they are more at risk of ingesting environmental lead through normal mouthing behaviors, b) absorption from the gastrointestinal tract is higher in children than adults, and c) the developing nervous system is more vulnerable to the toxic effects of lead than the mature brain (Koller et al., 2004; Landrigan et al., 1975). The fact that the child's developing nervous system is vulnerable could lead to a negative impact on children's intellectual development (Canfield et al., 2003; Lanphear et al., 2005).

There is much debate about the threshold blood-lead level for children, especially at what blood-lead level (BLL) there is a damaging effect on the children's intellectual functioning.

According to the World Health Organization and Centers for Disease Control's guidelines BLLs < 10 μg/dl can be regarded as safe, whereas medical evaluation and, in some cases, treatment is recommended for BLLs above 20 μg/dl (e.g. Roper, Houk, Falk, & Binder, 1991).

According to the CDC and the WHO, BLLs falling within the general boundaries of 10 to 20 μg/dl can be regarded as low. Despite being

* Corresponding author.
E-mail address: Michael.Woodley@vub.ac.be (M.A. Woodley of Menie).

https://doi.org/10.1016/j.paid.2018.09.005
Received 14 June 2018; Received in revised form 30 August 2018; Accepted 5 September 2018

termed as 'low' BLLs, the majority of the literature suggests that there are deleterious effects of BLLs of 10 to 20 μg/dl on intellectual functioning, including lowered intelligence (Baghurst, McMichael, Wigg, & Vimpani, 1992; Bellinger, Stiles, & Needleman, 1992; Pocock, Smith, & Baghurst, 1994; Rice, 1993; Yule, Lansdown, Millar, & Urbanowicz, 1981). So, in the present study we also expect a negative impact for BLLs of 10 to 20 μg/dl.

1.2. Competing theories

Other than the fact that it is detrimental to performance on IQ tests, precisely how lead influences intelligence is not known. Human intelligence is a complex phenotype that is organized hierarchically, with a highly general, overarching mental ability called *general intelligence* or *g* being situated at the apex of this hierarchy, and narrower and more specialized abilities being located further down the hierarchy (Carroll, 1993). From this, the following question arises: is the negative effect of lead exposure restricted to specific cognitive abilities, located further down the hierarchy, *g* (at the apex of the hierarchy) or both? It is important to note that IQ tests are indexes of performance with respect to both the *g* factor and also specific abilities (Carroll, 1993), thus it is possible to depress IQ scores via suppressing either general or specific performance, or both. To give some examples, a lowered IQ could be caused by a lowered level of *g*; a lowered level of for instance fluid abilities, short-term memory, and long-term memory; or a lowered level of *g* combined with a lower level of, for instance, crystallized ability and broad visual perception. The question of precisely how lead impacts intelligence has never been investigated comprehensively before however.

Predicting how lead may interact with the phenotype of intelligence is difficult, as there are indications in the literature of general, specific and mixed effects. Finkelstein, Markowitz, and Rosen (1998) note that lead exposure has toxic effects on a variety of brain regions, including the cerebral cortex, the hippocampus, and the cerebellum, which suggests that it might deleteriously influence many aspects of cognitive functioning leading to a decrease in *g*. Conversely, it has been noted that lead has asymmetric impacts on different cognitive abilities, suppressing processing speed, whilst leaving verbal ability intact (Lezak, 1983). This would be consistent with narrow impacts on ability. It is also possible that lead may have highly systemic effects on cognition in development, damaging neuroanatomical systems and structures that subserve both general and specific manifestations of intelligence. A second possible explanation is that in many of these studies the 'control' and exposure groups are not precisely matched in terms of level of *g*, with the former possibly exhibiting higher *g* due to the negative association between *g* and poorer life outcomes (including environmental and occupational exposure to neurotoxins) (Gordon, 1997; Gottfredson, 1997). Thus, the unique effects of neurotoxins on IQ may primarily be at the level of test specificities, however, the underlying difference in *g* between the 'control' and exposure groups may be acting in the opposing direction.

1.3. MCV/Jensen effects

To test whether the performance differences between the lead-exposure and 'control' group are moderated by the *g* saturation of the indicator, there are a variety of analytic techniques available. The most appropriate for use in *secondary analyses*, i.e. where the raw data are unavailable for reanalysis is the *Method of Correlated Vectors* (MCV). This technique simply involves taking the correlation between the *g* loadings of various subtests (termed the *g* vector) and the magnitude of an associated effect size (such as the impact of inbreeding depression on subtest scores; termed the *d* or *r* vector). If there is a positive correlation between the vectors, this indicates that *g* loading positively moderates an associated effect size, or in other words, the better a given subtest is at measuring the construct *g*, the larger the associated effect size. Such positive moderation effects have been termed *Jensen effects* (Rushton, 1998), after Arthur Jensen, the psychometrician who first developed MCV. It has been noted (e.g. Rushton, 1999) that Jensen effects are characteristic of *biological* phenomena, such as the heritability estimates for various IQ subtests (Voronin, te Nijenhuis, & Malykh, 2015, Table 3, p. 3), the negative association between IQ and fertility (Woodley of Menie et al., 2017) and factors such as processing speed and inbreeding depression effects (Jensen, 1998). The opposite phenomenon, i.e. when *g* loadings negatively moderate an effect size (*anti-Jensen effect*), are more characteristic of influences on IQ arising from the environment, such as practice effects (te Nijenhuis, van Vianen, & van der Flier, 2007), the IQ gains accrued amongst children via adoption into higher-IQ families (te Nijenhuis, Jongeneel-Grimen, & Armstrong, 2015), intensive educational interventions (such as the Head Start program) (te Nijenhuis, Jongeneel-Grimen, & Kirkegaard, 2014), and also the Flynn effect (te Nijenhuis & van der Flier, 2013). The existence of this broad pattern likely results from the fact that *g* is the principal (and in some cases the only) source of the heritability among IQ subtests (Panizzon et al., 2014), thus as biological variables will be further 'upstream' of genetics they will tend to associate most strongly with *g*. Environmental and cultural influences on IQ are further 'downstream' of genetics, thus will primarily impact the non-*g* residuals of IQ tests (i.e. the narrow and less heritable specialized abilities and test specificities). It should be noted that this pattern, whilst highly general, is not universal across studies utilizing MCV. Two notable exceptions to the pattern are the degree to which IQ subtests are *culture loaded*, which has been found to correlate positively with both subtest heritabilities (i.e. the degree to which the score on a specific subtest of an IQ test is influenced by genetic vs. environmental variation as typically measured using twin studies) and subtest *g* loadings (Kan, Wicherts, Dolan, & van der Maas, 2013), and also the degree to which performance on subtests across cohorts is sensitive to being boosted by the increased use of guessing in more recent cohorts, higher discriminability (more *g*-loaded) items being the ones that are more likely to elicit guessing as an answering strategy. This having been termed the *Brand Effect*, after the psychometrician Christopher Brand, who first proposed this as a potential contributor to the Flynn Effect (Woodley, te Nijenhuis, Must, & Must, 2014).

As was mentioned previously, one of the key advantages of MCV is that it can be used for meta-analyses involving secondary analyses of published data, relying only on correlation matrices, and/or published subtest *g* loadings and accompanying effect sizes. Other methods for examining moderation, such Confirmatory Factor Analysis (CFA), which measures the degree to which *g* is measurement invariant throughout the range of another variable, or in group comparisons, typically require the raw scores in order to yield quality data about the role of latent variables in moderating a given effect size, making the method suboptimal for meta-analysis, given that the vast majority of studies yield too little information for this method to work (unless the authors of those studies are forthcoming with their raw data, or the covariance matrix is employed in lieu of the raw data for the derivation of the relevant path coefficients). Furthermore, MCV has been refined into a relatively robust statistic via its marriage with the techniques of psychometric meta-analysis (Schmidt & Hunter, 2015). These techniques permit sources of sampling and measurement error (such as those associated with reliability and psychometric validity) to be explicitly quantified, and also corrected (via the use of imported meta-analytic values as the basis for synthetically disattenuating effect sizes). This strengthens MCV, as samples with small values of *N* and seemingly outlying vector-correlation values can be factored into meta-analyses, and corrected, yielding more accurate estimates of the aggregate vector correlation across studies (see: Woodley et al., 2014 for a more detailed treatment of the relative strengths and weakness of psychometric meta-analytic MCV vs. CFA).