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

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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

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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).

1.4. MCV applied to other neurotoxins

Thus far, no researchers have looked at whether or not *g* moderates the effect of lead on IQ using MCV. However, some researchers have used this technique to examine the pattern of moderation with respect to other neurotoxins on IQ. Based on the results of these analyses there is mixed evidence for Jensen and anti-Jensen effects. Debes, Ludvig, Budtz-Jørgensen, Weihe, and Grandjean (2015) examined the impact of prenatal methylmercury exposure on IQ subtests, finding that it is associated with a modest Jensen effect among young adults and young children ($r = 0.42$, $p < 0.001$, $N = 1022$). As these researchers had raw scores, they were able to conduct a CFA, which yielded results consistent with their MCV analysis – i.e., modest indications of moderation on the effect of methylmercury exposure stemming from *g* modeled as a latent variable (Debes, Weihe, & Grandjean, 2016). Flynn, te Nijenhuis, and Metzen (2014) examined the effects of prenatal alcohol and cocaine exposure on IQ, finding evidence of very small magnitude Jensen and anti-Jensen effects (respectively) ($\rho = 0.12$, $p = 0.182$, $N = 125$ for fetal alcohol exposure and $\rho = -0.23$, $p < 0.001$, $N = 215$ for fetal cocaine exposure). Finally, Metzen (2012) reanalyzed data originally published in Calderón-Garcidueñas et al. (2008), who examined the negative effects of atmospheric pollution (which causes neuroinflammation) on cognitive ability, using the WISC-R for an exposure sample of 55 individuals and a 'control' group of 18 sourced from Mexico City and the countryside, respectively, finding a low-magnitude anti-Jensen effect ($r = -0.17$, $p = 0.150$, $N = 73$) in this quasi-experiment. The sample-size weighted aggregate vector correlation across the four neurotoxins is 0.27 ($p < 0.001$; $N = 1435$). These vector correlations are generally weak to modest in magnitude and are quite heterogeneous in sign across different neurotoxin types (two are positive and two are negative in sign), suggesting that neurotoxic substances might not have consistent effects on either *g* or specialized components of cognitive ability.

If the results of applying MCV to lead exposure follows the pattern found for these other neurotoxins then we might expect an essentially low-magnitude positive correlation across studies, which would be consistent with either systemic effects on different brain regions associated with both general and specific manifestations of intelligence, or alternatively differences in the levels of *g* between comparison groups resulting from improper matching, counteracting the potentially direct effects of lead exposure on more specialized and less heritable abilities. This latter scenario would be consistent with the known role of socioeconomic status (SES) as a confounding factor in studies of the effects of lead exposure (Bellinger, 2008). SES is genetically correlated with cognitive ability (Trzaskowski et al., 2014). As it is not possible to estimate the extent to which SES and covarying factors (such as *g*) may confound group comparisons between control and exposure groups, it is not possible to estimate its magnitude. The results of our aggregating across vector correlation values sourced from various already published studies must therefore suffice as a guide as to what sort of an effect we might expect to see in the case of MCV as applied to lead exposure.

2. Method

To determine the magnitude, and direction of the correlation between the magnitude of *g* loadings and IQ scores of children with high lead levels, a meta-analysis was performed on vector correlations obtained from all studies that reported IQ scores for at least four subtests from children with BLLs > 10 μg/dl.

Meta-analysis is a statistical method where the results of all studies on a particular topic are aggregated allowing the use of powerful statistical techniques. The goal of the present study is to provide an estimate of the vector correlation between lead exposure and the magnitude of *g* loadings. We here carry out a meta-analysis based on the approach pioneered by Schmidt and Hunter (2015).

In general, *g* loadings were computed by submitting a correlation matrix to a principal-axis factor analysis and using the loadings of the subtests on the first unrotated factor. In some cases, *g* loadings were taken from studies where other procedures were followed; these procedures have been shown empirically to lead to highly comparable results (Jensen & Weng, 1994). Finally, vector correlations were computed (via Pearson correlation) using score differences between a high lead-level group and a comparison group, and the subtest *g* loadings.

2.1. Searching and screening studies

Two separate searches were carried out for English and Chinese-language studies – these being the two most frequently used languages in scientific publications. For the English-language studies, three methods were used to identify studies that contained IQ scores from the high BLL groups. First, an electronic search for published research using PsycINFO, ERIC, MEDLINE, PiCarta, Academic search premier, Web of science, Google Scholar, and PubMed was conducted. General keywords used were: *intellectual development*, *cognitive development*, *mental ability*, *intelligence*, *IQ*, *WISC*, *Wechsler*, and combinations of these terms, combined with: *lead*, *lead level*, *blood lead*, *tooth lead*, *bone lead*. Second, the reference lists of all important articles were scrutinized in search of additional studies. Third, cited reference searches were conducted using Web of Science to search for articles citing significant articles. The third strategy did not lead to additional English-language studies.

For the Chinese-language studies the same three methods were used, but only Chinese-language scientific databases were used. The China National Knowledge Infrastructure (CNKI) is the world's largest full-text information database and digital library and it is one of the most commonly used databases in the Chinese-language-speaking part of the world (Kong, 2010).

Wanfang Data is anther large database in China with broadly similar coverage. There is no evidence to show whether CNKI or Wanfang Data is the more comprehensive database. Most of the resources of the two databases overlap, so that most Chinese researchers use only one of them for searching the Chinese language literature (Kong, 2010).

Taiwan Scholar Journal Database (TWS) was also searched. This database covers > 85% of Taiwan's academic publications and is the most complete Taiwanese database (Taiwan Scholar Journal Database, n.d.). Finally, Airiti Library was searched. This database encompasses the Taiwan Electronic Periodical Services (TEPS). There are about 520,000 academic papers in Airiti Library (Airitilibrary, n.d.).

2.2. Inclusion rules

For studies to be included in the meta-analyses four criteria had to be met: 1) in order to obtain a reliable estimate of the true correlation between lead level and the *g* loadings, the cognitive batteries had to have a minimum of four subtests of the intelligence test; 2) the IQ test had to be well-validated; 3) the mean composite scores for the high lead-level groups had to be lower than the mean scores of the comparison group (the control group or the standardization sample of the IQ test); 4) only studies published in English or Chinese were used. Figs. 1 and 2 show the PRISMA flowcharts of, respectively, the English-language studies and the Chinese-language studies.

2.3. Computation of score differences between the groups of interest and a comparison group

Most of the studies in our meta-analyses used a 'control' group and a comparison group, but some groups did not have a 'control' group. In one case (Wu, Cen, Zheng, & Li, 2000) we were able to create a synthetic 'control' group by using a 'control' group from a highly comparable sample employing the same IQ battery (He, Wu, Long, Lu, & Tian, 2009). In a second case (Hu, Dong, Ren, & Cai, 1999) there were six samples, of which two were used as 'control' groups and four as high BLL groups. To see whether these five comparisons gave different

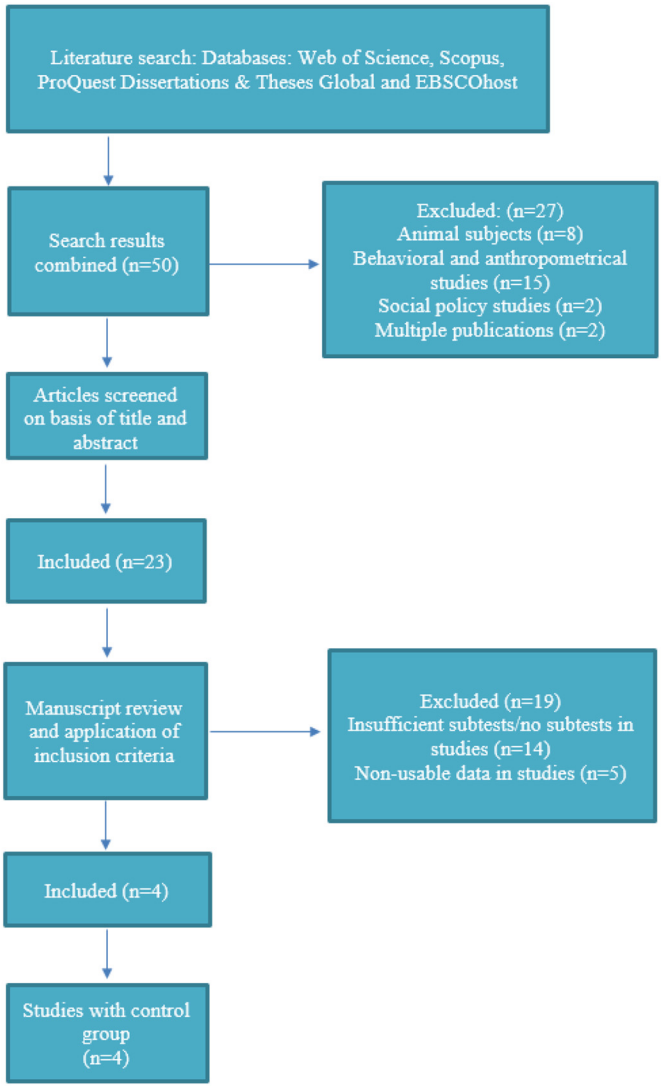

Fig. 1. PRISMA flow chart for selection of *r* (*g* × *d*), English-language articles.

outcomes, we ran a moderator analysis contrasting data points using original 'control' groups and data points using designated 'control' groups.

Score differences between a) the high BLL groups, and b) a 'control' group were computed by subtracting the mean score of the high BLL groups on the particular composite score in question from the mean score of the 'control' group, and then dividing the result by the *SD* of the standardization group. The *g* loadings were also computed matching the age range of the lead-exposed groups as close as possible.

2.4. Corrections for sampling error

Bare-bones psychometric meta-analytical techniques (Hunter & Schmidt, 1990, 2004) were applied using the software package developed by Schmidt and Le (2004). In the present meta-analysis, we corrected for one artifact identified by Hunter and Schmidt (1990) that alters the value of outcome measures: sampling error. The Method of Correlated Vectors is in essence a correlation between a number of *g* loadings and the same number of associated effects, so we used the number of subtests in the IQ battery as our measure of sampling error. Thus, test batteries with a smaller number of subtests have more sampling error than test batteries with larger numbers of subtests.

2.5. Multiple comparisons

In four cases, two high BLL samples were compared to the same 'control' samples, that is, a single sample was contributing to two effect sizes and therefore the contributing effect sizes were not fully independent. In our approach to dealing with multiple comparisons we lean heavily on the arguments put forwards by te Nijenhuis, Willigers, Dragt, and van der Flier (2016), who also carried out a meta-analysis using the Method of Correlated Vectors. Eight out of 16 comparisons use a high BLL group and a 'control' group, and eight out of 16 comparisons involve two high BLL groups being compared to the same 'control' group, so there is a modest dependence. Schmidt and Hunter (2015, p. 437) state that if a very large number of comparisons makes use of the same small sample, this may distort estimates of sampling error which then leads to undercorrections for this statistical artifact. However, in the present meta-analysis the four 'control' groups were paired with lead-exposure data from just two data points, so there is little error in the resulting aggregates. Schmidt and Hunter (2015, p. 452) review the literature and conclude that the distortion caused by dependent samples is probably negligible in real data. Following te Nijenhuis et al. (2016), we decided to not correct for the modest dependence in the samples.

3. Results

The results of the studies on the vector correlation between *g* loadings and the score differences between lead-exposed groups and 'control' groups (*d*) are shown in Table 1. The table gives data derived from 12 studies, yielding 16 data points, with participants numbering a total of 1935. It also lists the study citation, the cognitive ability test used, the vector correlation between *g* loadings and *d*, the sample size, and the mean age (and range of ages).

Table 2 presents the results of the bare-bones meta-analysis of the 16 data points. It indicates (from left to right): the number of effect sizes (*K*), total sample size (*N*), the mean observed vector correlation (*r*) and their standard deviation (SD_r), the mean standard deviation one can expect when corrections for sample size have been carried out (SD_{rho}). The next two columns present the percentage of variance explained by sampling error (%VE), and the 80% credibility interval after correction for sampling error (80% CI). This interval denotes the values one can expect for *rho* in sixteen out of twenty cases (see Hunter & Schmidt, 2004, pp. 205–207, for a detailed description). Note that we are working in the meta-analytical tradition of Schmidt and Hunter, where I^2 values are not used, instead percentages of variance explained by sampling error are estimated.

Table 2 indicates that the analysis of all 16 data points yields an estimated correlation (*rho*) of 0.10 with 135% of the variance in the observed vector correlations explained by artifactual errors. These values of variance explained of over 100% are not uncommon in Schmidt-and-Hunter-style meta-analyses and are caused by second-order sampling error (Hunter & Schmidt, 2004, pp. 399–401). According to Hunter and Schmidt (2004) in the absence of methods to correct for this, the most plausible way to interpret these values is that 100% of the variance between the data points in the meta-analysis is explained by sampling error. These credibility intervals were computed using *SD* values that had been corrected for sampling error (SD_{rho}), leading to a massive reduction in the value of the observed *SD*s. As all of the variation among studies is due to sampling error, the (corrected) 80% CI value range was necessarily 0.

We tested whether study quality acted as a moderator by running two separate meta-analyses for 1) all 11 studies with an original 'control' group and 2) all five studies with a designated 'control' group, where we assume the studies with an original 'control' group are arguably of better quality, as in these instances the 'control' group has been sourced from the same population as the lead exposure group. Table 2 indicates that the meta-analysis on all studies with an original

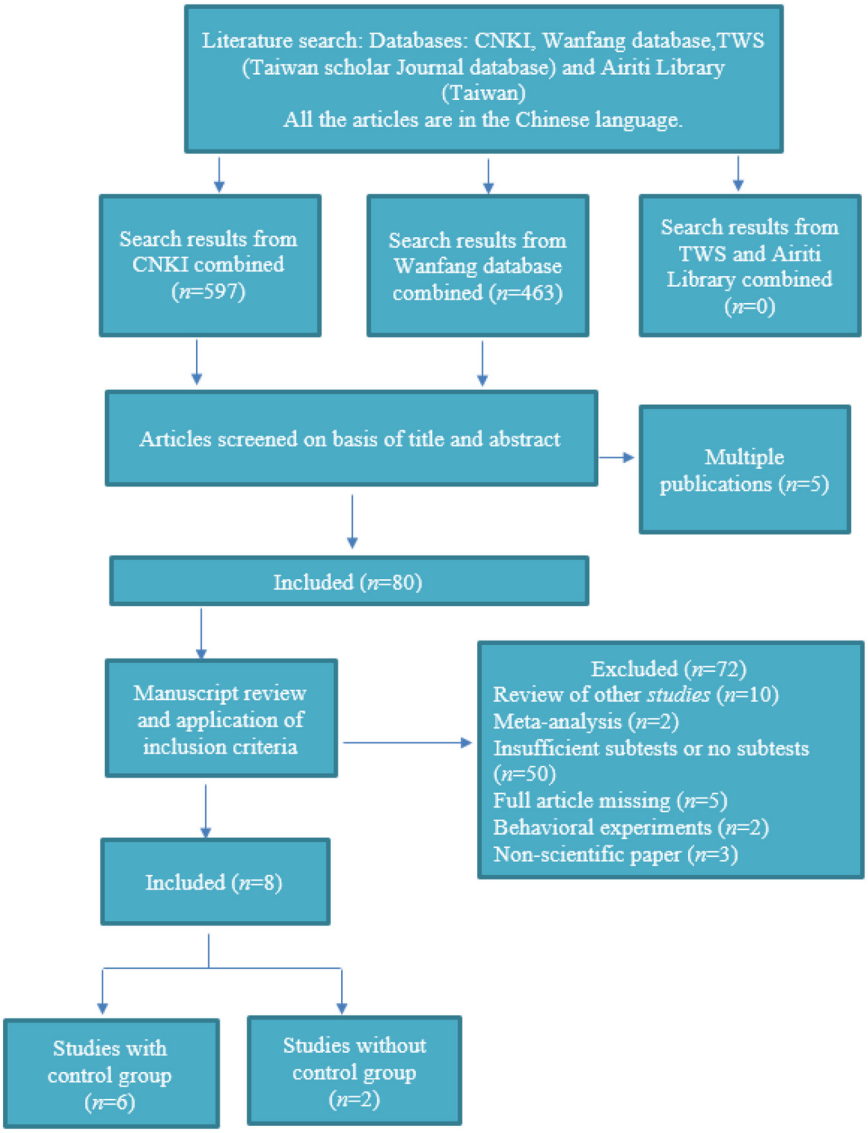

Fig. 2. PRISMA flow chart for r ($g \times d$), Chinese-language articles.

'control' group yields an effect size of 0.09 with virtually all variance between the 11 studies explained by sampling error. The meta-analysis on all studies with a designated 'control' group yields an effect size of 0.12 with all the variance between the five studies explained by sampling. So, the effect sizes are virtually identical and all or virtually all of the variance is explained by sampling error, just as is the case in the main meta-analyses. This means there is no clear moderator effect.

4. Discussion

We carried out a meta-analysis involving MCV on the role of *g* as a moderator of the impact of lead on IQ performance based on sixteen studies. There is no hard-and-fast rule as to the minimum number of studies to include in a meta-analysis, but sometimes it is stated that having ten data points makes for an adequately powered meta-analysis. We have no less than sixteen studies, and the total N is 1935. We conclude therefore that this is likely sufficient for the purposes of allowing the drawing of clear conclusions. Having sixteen data points means that high confidence can be placed in the resultant estimates of the influence of sampling error on the differences between the studies (Schmidt & Hunter, 2015). We were thusly highly successful in explaining the variance between the studies, which is an important goal of meta-analysis.

So, how does lead exposure negatively impact human intelligence? Does it reduce IQ at the level of *g*, specialized abilities and test specificities, or does it have mixed effects? The meta-analytic vector correlation between the magnitude of the depressing effect of lead exposure on subtest scores and their *g* saturations is positive in sign, but is weak in magnitude, in line with several other studies involving other neurotoxic substances (reviewed in the Introduction). This is consistent with the hypothesis that lead may have effects on both *g* and specialized abilities/test specificities owing to systemic effects on many different brain regions. However, this finding is also consistent with the expectation that two antagonistic factors are at work. Firstly, lead may have exclusively domain-specific impacts on the functioning of the central nervous system, inhibiting the brain regions that subserve short-term memory, such as the hippocampus and motoric speed, but not long-term memory, which would explain why lead exposure has been observe to suppress processing speed, but not verbal ability (Lezak, 1983). This suggests that neurotoxins (such as lead) should have their principal effects at the level of specialized abilities and narrow test specificities. General intelligence by contrast is highly distributed with respect to the brain's neural architecture (Haier, 2017) and is robustly canalized in development against the impact of factors that would

Table 1
Vector correlations between *g* loadings and lead exposure.

Study	Test	Nationality	*r* (*d* × *g*)	Number of subtests	*N* Lead	*N* comparison group	*N* all groups	Age mean lead	Age mean comparison group	Age range lead	Age range comparison group
Control group available											
Chen, Zhao, Zhang, Chen, and Wu (2000)	WISC-CR	Chinese	0.17	10	31	14	45			6–7	6–7
Chen, Li, Chen, Zhang, and Pu (2008)	WISC-CR	Chinese	0.62	10	77	31	108	9.92	8.95		
Ernhart, Landa, and Schell (1981)	McCarthy Scales	US	0.32	4	32	31	63			8–12	8–12
Hansen, Trillingsgaard, Beese, Lyngbye, and Grandjean (1989)	WISC	Danish	0.12	11	78	78	156	7	7	–	–
He et al. (2009)	C-WISC	Chinese	−0.50	11	40	154	194				
He et al. (2009)	C-WISC	Chinese	−0.53	11	6	154	160				
Kim, Yu, and Lee (2010)	KIT-P	Korean	0.18	5	151	151	302	10.5	10.5	9–12	9–12
Liu, Huang, and Pang (2004)	C-WYCSI	Chinese	−0.05	11	43	38	81			5–6	5–6
Needleman et al. (1979)	WISC-R	US	0.37	11	58	100	158	7.5	7.25		
Zhu et al. (2006)	C-WYCSI	Chinese	0.28	10	73	88	161			5–6	5–6
Zou et al. (2005)	WISC-CR	Chinese	0.25	10	85	33	118			6–13	6–13
Control group designated											
Wu et al. (2000)	C-WISC	Chinese	−0.08	11	62	154[a]	216	8.9	8.9	6–12	6–12
Hu et al. (1999)	C-WYCSI	Chinese	0.14	10	40	22	62				
Hu et al. (1999)	C-WYCSI	Chinese	0.12	10	20	21	41				
Hu et al. (1999)	C-WYCSI	Chinese	0.36	10	19	22	31				
Hu et al. (1999)	C-WYCSI	Chinese	0.07	10	18	21	39				

Note. The empty cells mean the values are not available.
r(d × *g*) = correlation between column vector of the impact of lead exposure on subtest score (d) and subtest *g*-loading; *N* lead = sample size for the lead exposure group; *N* comparison group = sample size of control group; age mean lead = age mean for lead exposure group; age mean comparison group = age mean for comparison group; age range lead = age range for the lead exposure group; age range comparison group = age range for the comparison group.
[a] Value estimated.

disturb its development, such as parentally-derived *de novo* mutations and presumably also neurotoxins (see discussion in: Woodley of Menie, Sarraf, Peñaherrera-Aguirre, Fernandes, & Becker, 2018). Secondly, as was mentioned in the introduction, the 'control' and exposure groups in these sorts of studies are typically not precisely matched in terms of levels of *g*. For individuals, low *g* is a risk factor for a variety of poorer life outcomes, including poorer quality diets, excessive consumption of alcohol and also living in poorer quality environments (i.e. living near to factories, power plants and other sources of pollution, or working in jobs in which neurotoxin exposure is an occupational hazard) (Gordon, 1997; Gottfredson, 1997). This does not mean that all low-*g* individuals will have poor life outcomes, but that on average they will have poorer life outcomes than high-*g* individuals. So, it could be argued that there might be an underlying difference in *g* favoring those in the 'control' group, i.e., those who are able to avoid exposure to neurotoxins by virtue of living in better environments or working non-hazardous 'white-collar' type jobs. As was mentioned already, it has been found that socioeconomic (SES) status confounds studies examining the impacts of lead exposure, as factors that covary with SES, but not with lead exposure can give rise to apparent differences between comparison groups (Bellinger, 2008). That *g* differences may be one factor that covaries with SES independently of neurotoxin exposure is supported by genetically-informed studies, that reveal substantial shared genetic variance between cognitive ability and SES (Trzaskowski et al., 2014). However, without precise information as to the extent of this confounding it is not possible to correct for this presently.

In the scenario that there are two prospectively antagonistic factors at play, their net action will cause the value of the vector correlation to deviate away from either +1 or −1 (depending on whether the effect is completely moderated by *g* or by performance with respect to specialized abilities and test specificities). Thus, in line with the results of vector correlations sourced from other studies presenting results amenable to MCV, we observe a weak magnitude Jensen effect consistent with the scenario sketched above (i.e. that these two factors might be acting simultaneously on the samples). Thinking in terms of this scenario helps to account for other 'anomalous' imperfect results (i.e. $r < 1$ in magnitude) from applying the Method of Correlated Vectors to other forms of environmental insult such as iodine deficiency ($\rho = 0.01$, $p = 0.889$, $N = 196$) and traumatic head injury ($\rho = -0.07$, $p = 0.066$, $N = 629$) (Flynn et al., 2014), as in these cases, the direct deleterious impacts on IQ could also purely be at the level of specialized abilities and test specificities. However, as with lead exposure, in these

Table 2
Bare bones meta-analytical results for the vector correlations between lead exposure and *g* loadings after correction for sampling error.

	K	$N_{subtests}$	N_{total}	*r*	SD_r	SD_{rho}	% VE	80% CI after correction for sampling error (*rho*)
All studies	16	155	1935	0.10	0.30	0	135	0.10 to 0.10
Moderator: study quality								
Original control group	11	104	1545	0.09	0.35	0.07	96	−0.01 to 0.18
Designated control group	5	51	389	0.12	0.14	0	516	0.12 to 0.12

Note. Meta-analytical results for vector correlations between *g* loadings and lead exposure. *K* = number of effect sizes; *N* = number of subtests in all the IQ batteries = total sample size; *r* = weighted mean observed correlations; SD_r = standard deviation of observed correlation; SD_{rho} = standard deviation after correction for sampling error; %VE = percentage of variance accounted for by sampling error; CI = 80%(*rho*) = credibility interval for weighted mean observed correlations using standard deviation after correction for sampling error (*SD rho*).

cases the 'exposure' and 'control' groups may also differ in terms of *g*, given that lower *g* is a major risk factor for nutritional deficiencies and accidents (Gottfredson, 2007).

The scenario that group differences in studies such as these are confounded with an uncontrolled underlying difference in *g* stemming from socioeconomic factors has an unfortunate implication, namely that it makes it impossible to say precisely how lead exposure impacts IQ in terms of variance components. The methods employed here do yield some interesting insights however. It is notable for example that all of the variance among the studies included in the present meta-analysis can be accounted for with sampling error. This leaves no room for prospective moderators such as age, degree of lead exposure and country of origin among the studies sampled, indicating that absent sampling error, the effects of lead across subtests would be uniform across studies.

To get a better idea of how lead exposure actually impacts different variance components of IQ, it would be necessary to conduct a study in which great care was taken to impose equality constraints in terms of intelligence across the exposure and 'control' groups. Another line of research would involve longitudinal analysis of individuals who have become exposed to lead in the course of their lives, where cognitive-ability data collected before and after exposure can be compared. By restricting the analysis to single individuals, the problem of underlying differences in *g* associated with differential socioeconomic characteristics can be obviated. This approach would therefore allow for the scenario that neurotoxins like lead have effects only on specific variance components of IQ to be tested.

Finally, it should be noted that changing levels of lead exposure (along with mercury and dioxin exposure and alcohol consumption – operationalized as part of a common *industrialization* factor) does not predict the long-term (> 1 century) secular decline in indicators of *g* (Woodley of Menie et al., 2018), contrary to predictions made by Demeneix (2017). *g* in this study was operationalized as a common factor among measures of simple visual reaction times, working memory, use of high-difficulty vocabulary words, 'social intelligence' and per capita macro-innovation rates (for a cross-temporal meta-analysis of these trends see: Woodley of Menie et al., 2017). Secular declines in a common factor of polygenic scores from age-stratified samples sourced from the US and Iceland did however predict the decline in *g* – after controlling for the effects of neurotoxins and also time (so as to control for temporal autocorrelation; Woodley of Menie et al., 2018). This further militates against the idea that neurotoxins such as lead should have their primary effects on IQ scores at the level of *g*.

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