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Raising IQ among school-aged children: Five meta-analyses and a review of randomized controlled trials

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ABSTRACT

In this paper, we examine nearly every available randomized controlled trial that attempts to raise IQ in children from once they begin kindergarten until pre-adolescence. We use meta-analytic procedures when there are more than three studies employing similar methods, reviewing individual interventions when too few replications are available for a quantitative analysis. All studies included in this synthesis are on non-clinical populations. This yields five fixed-effects meta-analyses on the roles of dietary supplementation with multivitamins, iron, and iodine, as well as executive function training, and learning to play a musical instrument. We find that supplementing a deficient child with multivitamins raises their IQ, supplementing a deficient child with iodine raises their IQ, and learning to play a musical instrument raises a child's IQ. The role of iron, and executive function training are unreliable in their estimates. We also subject each meta-analytic result to a series of robustness checks. In each meta-analysis, we discuss probable causal mechanisms for how each of these procedures raises intelligence. Though each meta-analysis includes a moderate to small number of studies (< 19 effect sizes), our purpose is to highlight the best available evidence and encourage the continued experimentation in each of these fields.

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Introduction

A child's intelligence is among the most important predictors for the rest of their academic life (Herrnstein and Murray, 1994; Nisbett et al., 2012). Children with higher IQs are less likely to be held back a grade (Safer, 1986), less likely to commit delinquent acts (Moffitt and Silva, 1988), less likely to be rejected by their peers or have few friends (Wentzel and Erdley, 1993). It would only seem natural to want to increase this trait in children, to enrich their cognitive lives and potentially prevent negative outcomes.

Long-standing debates question whether IQ at any age can be raised (e.g. Haier, 2014; Herrnstein and Murray, 1994; Jensen, 1969). In addition, there is argument that it would only be possible or profitable to raise IQ during a child's earliest years (e.g. Heckman, 2006). The fact that, as children age, more IQ variance is explained by genetic differences (e.g. Haworth et al., 2010; Briley and Tucker-Drob, 2013) could be interpreted as intelligence becoming less malleable with age. This assumption, we hope to show, is unsupported by the causal evidence. While genes are unquestionably the most important factor in determining someone's intelligence (e.g. Davies et al., 2011; Panizzon et al., 2014; Rimfeld, Kovas, Dale, & Plomin, 2015), the purpose of this paper is to aggregate the causal evidence on environmental factors on intelligence, not to argue for where intelligence comes from.

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The theoretical reason for this paper is to simplify the debate on the mutability of intelligence in childhood. A main issue holding up this debate is the lack of a systematic collection of the work on raising IQ. It is often the same few high profile studies that are highlighted. The actual literatures span many different fields. Such diversity of fields and widespread nature of the evidence contributes to the continued belief that intelligence is still something that cannot be raised (Haier, 2014). What is needed is a comprehensive set and analyses of all causal studies.

We present such a systematic collection on raising IQ among the school-aged. All interventions met the following requirements: (i) the sample was from a general, nonclinical population; (ii) the randomization occurred at the individual-level; (iii) the intervention was a sustained intervention; (iv) the outcome measure was a measure of intelligence.

Our first criterion was that the participants must be drawn from the general population. Although data from clinical populations can be informative, generalizing the effects of interventions designed for clinical populations to nonclinical populations is problematic. An intervention that helps someone overcome their disabilities (e.g., Klingberg et al., 2005, on training working memory of children with attention deficit/hyperactivity disorder) may not generalize to the nonclinical population (Spitz, 1986). Previous meta-analyses can often include clinical and nonclinical samples for meta-analytic purposes (e.g. Melby-Lervåg and Hulme, 2013).

Our second criterion was that the authors must have used an individual-level randomized controlled trials (RCTs). Participants must have had an equal chance of being assigned to an intervention or a control group. We thus exclude studies that first enrolled an experimental group and then later enrolled a control group, used classroom or cluster randomized trials, or compared an experimental group with a group selected later. This strategy allows us to investigate robustness of the causal role or specific environmental variables through the logic of counterfactual causation (e.g. Rubin, 2004). In addition, the use of randomized groups allows us to avoid problems that arise in non-experimental samples (such as Flynn effects (e.g. Pietschnig and Voracek, 2015), as both groups are equally affected by them; e.g. Kanaya, Ceci, & Scullin, 2005). Briefly, the Flynn effect is the finding that IQ scores are higher the further away from the standardization sample on an IQ test is. So a sample of 10 year-olds will score higher on an IQ test normed in 1970 than they would if it were normed in 1990. Since both groups would be equally affected, they are not a concern in randomized trials.

Furthermore, we omit evidence such as correlations between participation and the activities they engage in (e.g. Sabaratnam and Klein, 2006). This is in contrast to many meta-analyses that either only use non-experimental data (e.g. Qian et al., 2005) or mix experimental, quasi-experimental, and non-experimental data (e.g. Jaschke, Eggermont, Honing, & Scherder, 2013; Warthon-Medina et al., 2015). Such alternate approaches dilute the strength of the causal interpretation that can be gleaned not only from each study—but from all of the experiments as a whole.

Our third criterion was that the intervention must have been a sustained treatment and not alteration to test administration. Some authors alter the procedures, instructions, or context of intelligence test administration to examine effects on IQ scores. Such effects are obtained as a result of the one-time manipulation during the testing experience. Although such interventions are informative, we exclude them because their effects may reflect the role of extraneous factors in the testing environment rather than genuine increases in underlying intelligence. Our definition for a sustained intervention was one that treats participants for at least two weeks. The purpose of this criteria is to avoid studies which, for example, give IQ tests in different colors and report changes in intelligence (e.g. Elliot, Maier, Moller, Friedman, & Meinhardt, 2007). We believe that only sustained interventions represent the potential to increase intelligence, opposed to removing barriers to performance.

Finally, we only included studies where the authors used accepted measures of IQ. Although imperfect, IQ scores represent a good approximation of the underlying latent variable shared by all cognitive tests (Jensen, 1998; Tommasi et al., 2015). For the purposes here, we only included studies that used accepted measures of IQ, not studies using a single subtest. While other constructs are important (e.g. school grades), they do not represent the same underlying construct. Full-scale IQ was always used here as the main dependent variable. If multiple measures existed, results were broken down by those different tests. Studies that used a single subtest from an IQ battery (block design, vocabulary) were not included. Many previous meta-analyses include studies that use any cognitive subtest (e.g. Hetland, 2000), or mix meta-analytic results of studies using IQ measures with those using other measures (single subtests or school grades; e.g. Falkingham et al., 2010). Therefore, our meta-analyses were specifically focused towards the increasing of IQ, not just any variable that can be considered cognitive. We go into further detail on these points in the limitations section.

At the end of the manuscript, we discuss some of the limitations of this work, including small number of randomized controlled trials (as a result of our strict inclusion criteria) and the difference between raising IQ and raising *g* (latent general intelligence). In short, raising IQ scores is a necessary but not sufficient condition for showing increases in underlying latent intelligence.

Overview of analyses

In this paper, we examined all RCTs involving school-aged children (roughly age five through pre-adolescence). The purpose of this was to isolate the effects of environmental manipulations on children's IQ after they have entered school. It is believed by some that it is too late to alter IQ at such ages (e.g. Heckman, 2006). Each study came from a search of the literature using PSYCHInfo and Google Scholar, using the keywords ~random, IQ, and cognitive. Every study that met all of the requirements was then subject to exhaustive forward and backward searches. Backward searches include every relevant

study cited in a manuscript are checked for inclusion. Forward searches use articles that cite a chosen study that are checked for inclusion. We also contacted the authors of each study for missed and unpublished work from either themselves or colleagues.

We used the effect size of each study calculated from the standardized difference between experimental and control groups' IQ scores at the end of the intervention. This was done to allow interpretations to be made about changes in constructs and not change scores (also eliminating the statistical problems that occur with change scores, see [Huck and McLean, 1975](#) for an introduction to both points).

In this investigation of the literature (published and unpublished studies were included in all analyses), we meta-analyzed 36 interventions that met our inclusion criteria. These 36 interventions yielded 41 effect sizes across 5730 children. Topics for how to raise intelligence were not chosen a priori, every intervention that met the inclusion criteria, regardless of type of intervention, were included. Five types of interventions had enough replications to warrant meta-analysis. The first half of the article describes research on nutritional supplements to children. We meta-analyzed the effects of providing: multivitamin supplements to children, iodine supplements to children, and iron supplements to children. The second half of this article describes research where investigators alter the educational environments of school-aged children. We examined the effect of Executive Function training on a child's IQ. We also examined the effects of learning to play a musical instrument.

Throughout the article, we reviewed interventions with too few replications within an experimental paradigm to merit meta-analysis. The purpose of this is for comprehensiveness of inclusion. We included all models run and those results. In addition, we included all studies originally found in our search of the published and unpublished literature and our rationale for removing certain studies from analysis.

All effect sizes were the standardized mean difference between experimental and control groups at immediate posttest. All studies were weighted by the standard errors calculated according to the following formula ([Hedges, 1981](#)):

$$SE = \sqrt{\frac{N}{n_E n_C} + \frac{g^2}{2N}}$$

We included no additional weighting of studies based on reliability, quality, or any other subjective weighting as doing so would introduce substantial subjectivity in the analysis ([Card, 2012](#)). In addition, as the number of studies is small in each of the analyses, we used fixed-effects models, as random-effects models have poor power with smaller number of studies ([Field, 2001](#)).

In each meta-analysis, we conducted a series of robustness checks to test the reliability of the meta-analytic results. In every meta-analysis we tested for meta-analytic outliers (different from normality tests due to the weighting of studies). Heterogeneity was tested by both the Q and I^2 statistics, which have adequate power to detect moderate heterogeneity in meta-analyses of more than about 17 studies ([Huedo-Medina, Sánchez-Meca, Marín-Martínez, & Botella, 2006](#)).

We also tested for missing studies (publication bias) in each meta-analysis. The metric of testing publication bias was chosen to maximize power for each analysis. The power of different tests of publication bias change based on the number of studies, as well as the degree of heterogeneity. For large homogenous tests, the Egger tests has the best power ([Idris, 2012](#)), for example. When the number of studies is small, the trim and fill method has shown the greatest power for detecting publication bias ([Terrin, Schmid, Lau, & Olkin, 2005](#)). When results of publication bias tests were ambiguous or heterogeneous, we ran multiple tests of publication bias and report each one. In the meta-analyses where publication bias may exist, we test different corrections. Unfortunately, monte-carlo tests of different corrections for publication bias show that they can produce worse estimates than uncorrected ones ([Reed, Florax, & Poot, 2015](#)). Because of this, we present multiple analyses using weighted least squares (WLS) and precision-effect estimate with standard error (PEESE) corrections. These two corrections have been shown to introduce the least amount of bias in correcting for publication bias in small meta-analyses ([Reed et al., 2015](#)).

We were unable to perform p -curve or p -curve adjusted effect sizes (e.g. p -TES) in any of the meta-analyses as such procedures require large numbers of statistically significant estimates (p -curves are tests of excessive significant effects; [Simonsohn, Nelson, & Simmons, 2014](#)). This limitation has the added benefit of making the results unusually robust to p -hacking. It is unlikely that a researcher would p -hack to a non-significant value. We still attempted to address this in each meta-analysis by imputing all statistically significant results with the largest non-significant effect size given the sample size. All meta-analyses and tests of publication bias were run in STATA v.13.1; outlier analysis and possible publication bias corrections were run in R using the *metafor* package ([Stanley and Doucouliagos, 2014](#); [Viechtbauer and Cheung, 2010](#)).

Nutritional supplements

We first analyzed the effects of several nutritional supplements given to children in the hopes of raising their intelligence. We discuss each of these interventions as well as possible causal mechanisms. Readers should note that the causal mechanisms discussed are not exhaustive. We direct interested readers to the relevant literature for a more thorough treatment of the biochemical interactions of supplements and their effects on human cognitive function.

Multivitamins and intelligence: a meta-analysis

Our search of the literature yielded 15 studies contributing 18 effect sizes across 1901 participants. In these studies, children were randomly assigned to receive either a daily multivitamin or a placebo for an average of 19 weeks.

Methods

The first issue is with one study from a previous analysis (Schoenthaler and Bier, 1998) that could not be located. The study was listed as “(Wender)” in the paper, with 11 students receiving supplements and seven receiving a placebo. There was no corresponding citation. The age of the participants was listed as 12. The text of the article refers to a study by Snowden (1997) and indicates it was included in the analysis, which also includes 11 experimental, and seven control participants but lists their age as 9–10. In addition, the Wender and Snowden articles have different effect sizes. As there are different ages and effect sizes, both are included in this meta-analysis. In addition, one study (Deinard, List, Lindgren, Hunt, & Chang, 1986) was removed due to previous criticisms of its methodological and randomization quality (Grantham-McGregor and Ani, 2001). Finally, we only included studies of children with mild deficiency. There is a linear relationship between degree of deficiency and intellectual impairment (Venables and Raine (2015), so it is still possible that even mild deficiencies can cause deficits in intelligence.

Two studies (Nelson, Naismith, Burley, Gatenby, & Geddes, 1990; Southon et al., 1994 for both boys and girls) included data on both verbal and non-verbal composite scores (with no overall IQ score); we only analyzed the nonverbal tests in these instances. Removing these two studies had no effect on the results.

One study (Osendarp, Baghurst, Bryan, et al., 2007) included a third intervention group (that received multivitamins and fatty acids). We removed this third group so only the multivitamin vs. placebo was included in the meta-analysis. Finally, two further studies (Nga et al., 2011; Solon, Sarol, Bernardo, et al., 2003) included a third intervention that includes multivitamins and a de-worming agent. We only included the non-de-worming multivitamin vs. placebo in the meta-analysis, though de-worming on its own has no effect on IQ (Nga et al., 2011). An additional study used children who receive a low level of multivitamins and long-chain polyunsaturated fatty acids as the control group (Muthayya et al., 2009). As this does not provide a proper counterfactual in line with the rest of the studies, we removed this study from the analysis.

Results

We analyzed 15 studies on multivitamin supplementation and children’s intelligence, yielding 18 effect sizes. Results suggested that there was an incredibly small but significant effect of taking multivitamins on IQ ($g = 0.097$, 95%CI = 0.006 to 0.187; see Fig. 1).

There was no evidence of heterogeneity in the sample, ($Q(17) = 18.24$, $p > 0.374$; $I^2 = 6.8\%$, $p > 0.373$), suggesting the results are not moderated by already-investigated study characteristics.

Robustness checks. Meta-analytic outliers: There were no outliers in the multivitamin supplementation data (all externally studentized residuals < 1.33)

Missing Studies (Publication Bias): There was no evidence of missing studies (publication bias) in our data ($p_{Egger} > 0.446$). *p*-hacking adjustment: Two studies showed statistically significant effects of multivitamin supplementation on mildly deficient children (Benton and Cook, 1991; Snowden, 1997). We shrunk the effect sizes from those studies to 0.607 and 0.992 (respectively) to represent $p = 0.0505$ and again shrunk them to 0.507 and 0.82 (respectively) to represent $p = 0.099$. Re-running the meta-analysis using these values shrunk the meta-analytic estimate to 0.091 and 0.087. The results remained significant in the $p = 0.0505$ imputation but not in the $p = 0.099$ imputation. Therefore, if we believe that the two statistically significant studies (Benton and Cook, 1991; Snowden, 1997) had really observed *p*-values between 0.051 and 0.099 and then *p*-hacked their data, we may say that multivitamin supplementation does not alter the IQ of mildly deficient children. We cannot know whether this was the case and do not claim the authors behaved that way without proof. What it does tell us, however, is that the meta-analytic result on supplementing deficient children with multivitamins is sensitive to small alterations in effect sizes. This could have also been surmised from the confidence interval of the original meta-analytic estimate (95%CI = 0.006 to 0.187).

Unsubstantiated study: Removing the suspicious “Wender” study did not substantially alter the results ($g = 0.092$, 95%CI = 0.001 to 0.183; $Q(16) = 17.137$, $p > 0.377$; $I^2 = 6.6\%$, $p > 0.376$).

Discussion

Multivitamins contain an array of vitamins and minerals. This creates a problem when trying to identify a single causal agent within a multivitamin, as it is not clear which vitamins or combinations of vitamins may be raising IQ. Two studies included no information on what was in the multivitamins (Todman, Crombie, & Elder, 1991; “Wender” study). Every other multivitamin included at least: 1.3mg Iron, 3.75mg Zinc, 40mg Ascorbic Acid, and .5mg B6, except for one study that used an Indian blend of supplements (Nidich, Morehead, Nidich, Sands, & Sharma, 1993).

Taking a multivitamin can serve one of two purposes, either to remediate an incomplete diet with vitamins and minerals or to complement a complete diet for further intake of those healthy elements. We explored the conditions under which taking multivitamins raises IQ to distinguish which of these two situations (remediate or complement) was more congruent with the literature. Note that the data was not available in this analysis to solve this question meta-analytically.

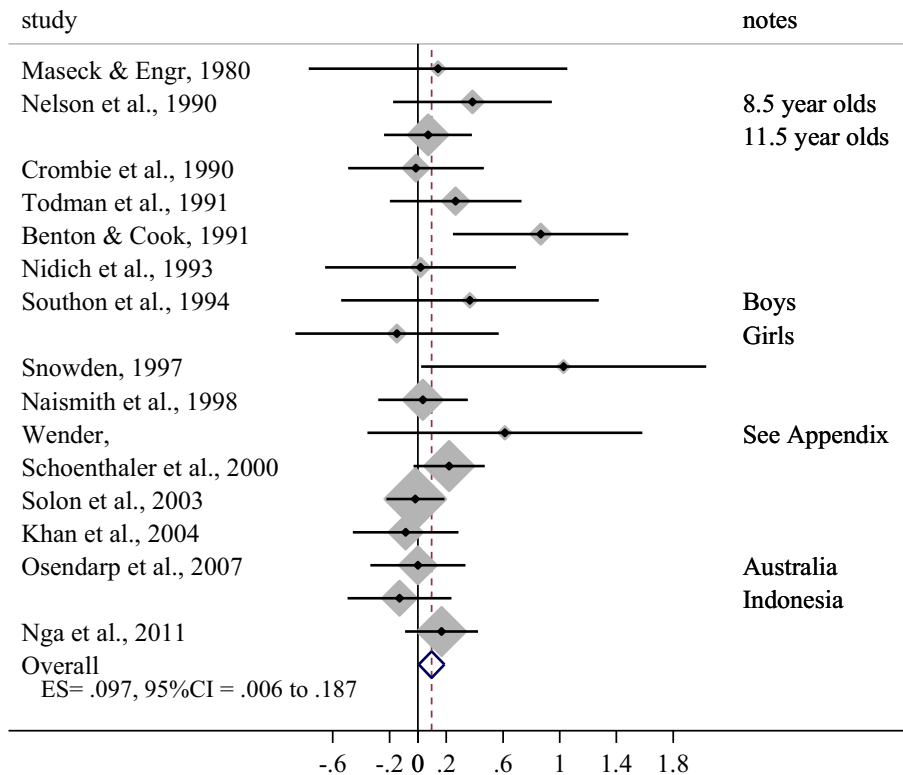


Fig. 1. Meta-analysis of multivitamin supplementation on children's IQ.

Some of the earliest RCTs into the role of taking a multivitamin and IQ occurred in the late 1980s and showed that giving multivitamins to adolescents increased their IQ (Benton and Roberts, 1988). These reports were subsequently critiqued in a series of arguments (Bates, Thurniam, & Powers, 1988; Benton, 1988; MacFarlane, 1988; Yudkin, 1988). The resolution to this was partially finalized with the publication of a study (Benton and Buts, 1990) that answered much of what was under debate: are multivitamins a miracle pill?

The answer was no. In fact, they do not work for a large portion of the population. Instead, having a poor diet, meaning a diet deficient in many vitamins and minerals, leads to a decrease in IQ. This follows from the finding that supplementing depleted children raises their IQ, while supplementing replete children has no effect. Multivitamins work to raise IQ by counteracting this loss; they indeed remediate a deficient diet. Children who have a poor diet gain the most benefit from taking multivitamins, while children whose diets are sufficient gain no advantage (Benton and Buts, 1990). Therefore, a multivitamin is only a partial remedy for a poor diet, not a get-smart pill on its own (Maseck and Engr, 1980).

These results mostly support past review articles (e.g. Benton, 2001; Eysenck and Schoenthaler, 1997). Many review articles also look at non-experimental literature to bolster claims or compare gain scores opposed to test scores—potential sources of bias that are avoided here. The effect size from this meta-analysis is small and the 95% confidence interval comes very close to zero. Six additional studies are also included here that have been conducted since the 1997 review, all of which are larger than any previous RCT in school-aged children. In addition, we focused on school-aged children and do not include work in adolescence; previous work suggests the effects may be stronger for pre-adolescent children opposed to adolescents (Eysenck and Schoenthaler, 1997).

Iodine and IQ: a meta-analysis

The role of iodine in intelligence was first investigated by reversing the endemic effects of iodine deficiency and goiter. Remedying these diseases led to massive increases in IQ across populations, often through public policy interventions like iodized salt (Feyrer, Politi, & Weil, 2013). While to date there has been no work on iodine supplementation in replete children, the RCTs presented here represent the effect of reversing only mild iodine deficiency.

Methods

We originally found five studies providing six effect sizes on the effects of iodine supplementation and IQ. One study was removed for using children with severe iodine deficiency (Bautista, Barker, Dunn, Sanchez, & Kaiser, 1982). A further study

included two experimental groups, one with iodine supplementation and another with iodine and iron supplementation (Shrestha, 1994). We only included the iodine only supplementation in this meta-analysis.

Results

We analyzed four studies contributing one effect size each across 907 children who were randomly assigned to receive either iodine supplements or a placebo for between four and ten months. Across these studies, we found remedying mild iodine deficiency increased the IQ of school-aged children by over half a standard deviation ($g = 0.531$, 95%CI = 0.394 to 0.668; see Fig. 2). There was, however evidence for heterogeneity ($Q(3) = 123.236$, $p < 0.001$; $I^2 = 97.6\%$, $p < 0.001$).

Robustness checks. Meta-analytic outliers: There was one meta-analytic outliers present in this analysis, with an externally studentized residuals of 2.719 (Shrestha, 1994). Removal of the outlier attenuated the effect size but did not negate it ($g = 0.308$, 95%CI 0.164 to 0.451); nor did it resolve the problem of heterogeneity ($Q(2) = 9.843$, $p < 0.007$; $I^2 = 79.7\%$, $p < 0.008$).

Missing Studies (Publication Bias): There was no evidence for missing studies (publication bias) in this data. To test this, as the number of studies was so small, we used the trim and fill method (Duval and Tweedie, 2000) which has been shown to have high power to detect moderate to severe publication bias even in small meta-analyses (Idris, 2012). This method has also been shown to have inflated type I errors as well, meaning a “non-significant test from trim and fill method clearly indicates the presence of publication bias has been ruled out” (Idris, 2012; p. 1416; see also Terrin et al., 2003). *p*-hacking adjustment: Two studies showed statistically significant effects of iodine supplementation on mildly deficient children (Shrestha, 1994; Zimmermann, 2011). We shrunk the effect sizes from those studies to 0.232 and 0.157 (respectively) to represent $p = 0.0505$ and again shrunk them to 0.195 and 0.133 (respectively) to represent $p = 0.099$. Re-running the meta-analysis using these values shrunk the meta-analytic estimate to 0.145 and 0.133. The results remained significant in the $p = 0.0505$ imputation but not in the $p = 0.099$ imputation. Therefore, if we believe that the studies from Shrestha and Zimmerman really had observed effect sizes of 0.195 and 0.133, and then *p*-hacked their data to the reported effect sizes of 3.017 and 0.586, we may say that iodine supplementation does not alter the IQ of mildly deficient children. We think this scenario is unlikely.

Discussion

It is extremely important to emphasize the limitations of this analysis. First, there is significant heterogeneity within the results. Second, all of the children were mildly iodine deficient. Mild iodine deficiency was defined as clinically low levels of iodine intake without additional diseases caused by deficiency (e.g. goiter). There was no experimental research to date on iodine supplementation in replete children. Therefore, the findings from this meta-analysis cannot be used to argue that iodine supplementation raises IQ for all children.

Severe iodine deficiency in childhood causes mental retardation, stunted growth, and cretinism (Zimmerman, 2011). Supplementing children with moderate to severe iodine deficiency can reverse some of the negative cognitive effects (Bleichrodt and Born, 1994). Here we show iodine supplementation can raise the IQ of children who are even mildly deficient. Iodine deficiency slows the production of thyroid hormones (Zimmerman, 2011). Since thyroid hormones (especially triiodothyronine; Calzà et al., 2010; Gordon et al., 2009) are partly responsible for the myelination of neurons (Dussault and Ruel, 1987; Kester et al., 2004), part of the reason iodine deficiency leads to intellectual delays is through

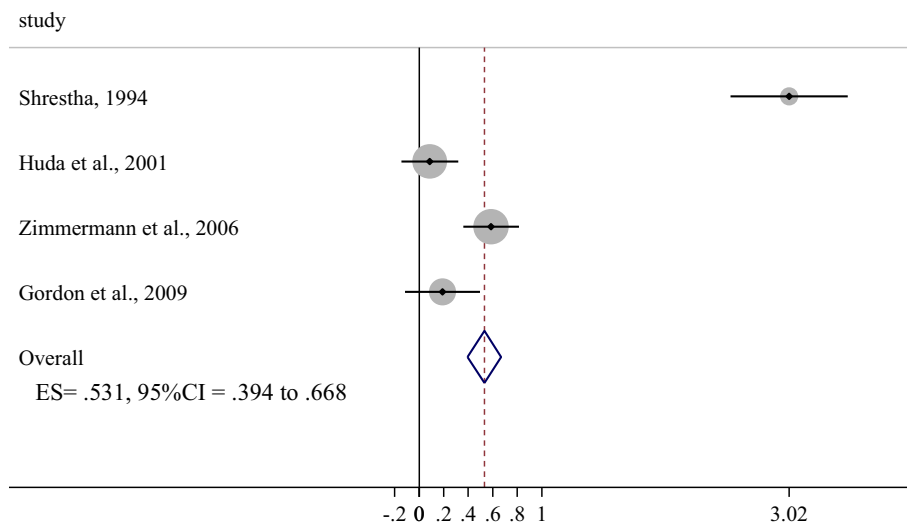


Fig. 2. Meta-analysis of iodine supplementation on children's IQ. Effects of daily iodine supplementation on IQ for mildly iodine-deficient children. Note the results remain significant with the removal of the Shrestha study.

slowing the myelination of neurons in the brain. This would suggest that iodine supplementation increases the IQ of mildly deficient children through increased white matter development.

The development of white matter in the brain is most pronounced through childhood and adolescence and supports the development of intelligence (Ferrer et al., 2013). Iodine deficiency can slow down this entire process, including restricting the development of intelligence through a stunting of myelination. It is not readily understandable whether supplementation of iodine replete children could raise their IQ, unless myelination over and above standard development would confer additional intellectual benefits. There is no direct evidence from this meta-analysis or any of the interventions included to suggest this possibility. Future research is necessary not only to confirm the findings of this heterogeneous, small meta-analysis—but also to explore whether supplementation in iodine-replete children has an effect on their white matter development and IQ.

Iron and IQ: a meta-analysis

Iron is a crucial mineral for proper cell function (Watanabe, Kodama, & Hikosaka, 1997). Reversing iron-deficiency anemia through supplementation can reverse these decreases in cognitive ability (Idjradinata and Pollitt, 1993; Soemantri, Pollitt, & Kim, 1985). It therefore could be possible that iron supplementation in non-anemic individuals can also increase their IQ.

Methods

We originally found 11 studies contributing 21 effect sizes providing iron to children and testing if it raised their IQ. Two studies (Kashyap and Gopaldes, 1987; Seshadri and Gopaldes, 1989) were removed as they only included children with iron deficiency anemia. Two additional studies (Pollitt, Soemantri, Yunis, & Scrimshaw, 1985; Soemantri et al., 1985) included data on both anemic and non-anemic children, for the purposes of generalizability we only included non-anemic children where such a breakdown was available; fuller data from (Pollitt et al., 1985) was found in a different publication (Pollitt, 1997). Two studies included information on follow-up data from the children (Rico et al., 2006; Soemantri, 1989); we only included the immediate posttest after the intervention ends to see the immediate effect of iron supplementation on IQ. One study (Sungthong, Mo-suwan, Chongsuvivatwong, & Geater, 2004) included two intervention groups, one where children received iron daily and one where they received it once per week. To keep the data in line with the rest of the studies in this meta-analysis we only included the daily intervention. In the end, we were left with nine studies providing 10 effect sizes.

Results

We analyzed 10 effect sizes from nine studies across 2472 participants; all children were non-anemic though still mildly iron deficient. In each RCT children were supplemented with iron or a placebo daily for at least three months. Results indicated that supplementing a child who was iron deficient without iron-deficiency anemia does not reliably raise their IQ ($g = 0.032$, 95%CI = -0.048 to 0.111 ; see Fig. 3).¹

There was, however, significant heterogeneity in the sample ($Q(9) = 77.673$, $p < 0.001$; $I^2 = 88.4\%$, $p < 0.001$) so we must either remove outliers, look for moderators, or run a random effects analysis. Unfortunately, there were no outliers and not enough effect sizes for a reliable random effects analysis. In addition, there was very little power to detect moderators.

Robustness checks. Meta-analytic outliers: There were no meta-analytic outliers present in this analysis; all externally studentized residuals fell below 1.297.

Missing Studies (Publication Bias): There was mixed evidence of missing studies (publication bias) in our data. Estimation using the Egger test showed no bias ($p > 0.657$) but has low power in a meta-analysis of this size (Idris, 2012); estimation using the trim and fill method showed evidence of moderate publication bias, but this method also has a tendency towards inflated type I errors (Idris, 2012). In addition, trim and fill may spuriously indicate publication bias under heterogeneity (Terrin et al., 2005). We therefore report adjusted estimates using a couple of estimators (following Reed et al., 2015). The WLS estimator correcting for publication bias produced the same coefficient as the standard estimate ($g = 0.032$) but with wider standard errors and thus does not change the results here. The PEESE estimator also returned a similar coefficient and did not alter the results ($g = 0.015$, 95%CI = -0.235 to 0.265); nor did it demonstrate evidence for publication bias ($p > 0.157$). Therefore, whether there are missing studies adjusting for the possibility of publication bias would not alter the results here. Furthermore, as the meta-analytic results were not statistically significant it is unlikely the results are contaminated by influential p -hacking.

Other nutrients and combinations of nutrients—a review

Iodine and iron

In one study, mildly deficient children in Malawi were supplemented daily with either iron (60 mg) and iodine (490 mg) together or a placebo (Shrestha, 1994). Mirroring the effects from the Iodine meta-analyses, children who received the combined drugs had higher IQs than those who received the placebo.

¹ These results remain unchanged when three studies from infancy are included (Lozoff et al., 2003; Soewondo et al., 1989; Zhou et al., 2006)

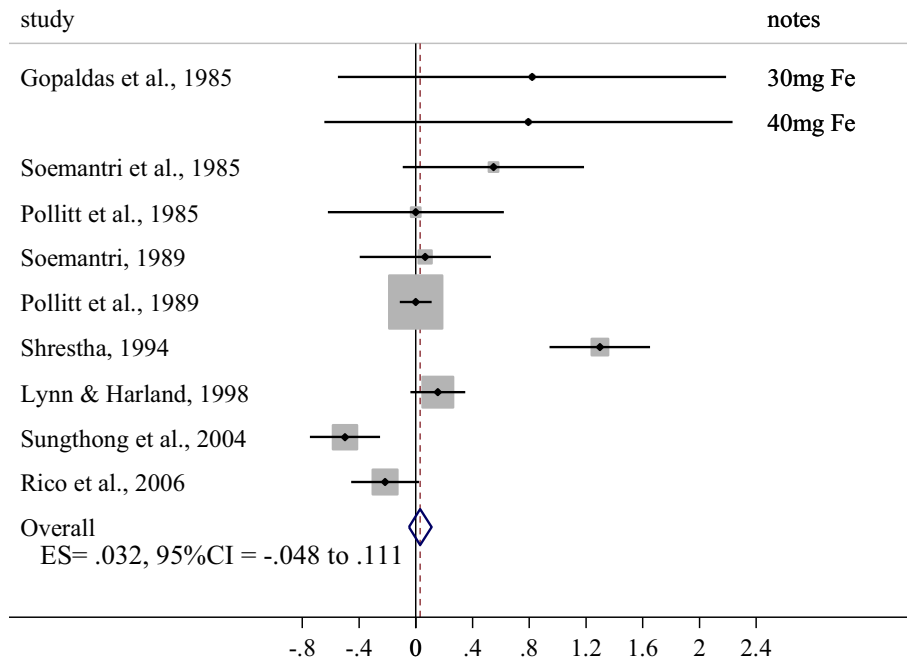


Fig. 3. Meta-analysis of multivitamin supplementation on children's IQ effects of daily iron supplementation on IQ for iron-deficient, non-anemic children.

Zinc

Three studies supplemented children with zinc to see if there were any differences in their IQ. In one study (Tupe and Chiplonkar, 2009) the authors administered either (i) daily zinc (16.6 mg) and Iron (0.74 gm) supplements; (ii) food fortified with 20mg zinc, or (iii) a placebo, to 170 Indian girls for 10 weeks. There was no effect on the girls' IQ. In another other study (Rico et al., 2006) the authors gave either Zinc (30 mg) or a placebo to 256 Mexican schoolchildren for six months. There was no effect on the children's IQ. Finally, supplementing 154 Guatemalan schoolchildren with Zinc offered no benefit to intelligence after 25 weeks of daily supplementation (Cavan et al., 1993). Therefore, it is safe to conclude Zinc does not affect intellectual ability within non-clinical ranges.

Zinc and iron

In one study the authors supplemented 259 Mexican schoolchildren with a combination of Zinc and Iron (30 mg each) or a placebo for six months (Rico et al., 2006). There was no effect.

Environmental changes on IQ

We now consider those interventions designed to increase intelligence by altering the environments of school-aged children. Most of these interventions aim to alter the educational practices of children either through different instruction practices or instruction in subjects not tested by standardized tests (i.e. the Fine Arts). In this section, we review interventions where there is insufficient data to enable meta-analysis and present two meta-analyses, one on the effects of executive function training and one of music instruction.

Balance and coordination exercise

One study looked at balance and coordination training on the IQ of school-aged children. In this study, 142 middle-schoolers were paired and randomly assigned to either their standard physical education class or a highly organized balance and coordination regimen. The intervention significantly raised the IQ of boys by about 3 points and significantly lowered the IQ of girls by about 3 points (Ismail, 1967). There was no rationale for analyzing the results by gender beyond the finding of a significant gender by treatment interaction. However, analyzing the overall results showed no effect of balance and coordination exercise on IQ. In light of these confusing results, we cannot draw any conclusion about such specific training on IQ.

Home academic support

In an effort to help children from high-risk homes, children in the Abecedarian project were randomized at birth to either experimental or control groups (Ramey and Campbell, 1991). Children in the experimental group had their entire early

environment altered for the first five years of their life with home visits, special center visits, and parental support. When the children were five years-old some children were re-randomized from both the experimental and control groups into a new condition where they were provided home tutors who gave individual help and supplemented school learning with activities for three years. These tutors met with the child a couple times per month at school and home for three years. Mothers of these children were given support and taught how to help their children with what they were learning in school with supplemental activities. Comparing children who only received the school-aged intervention to controls showed no effect on IQ. The results of the earlier parts of the intervention are treated elsewhere as that intervention was on young children and not school-aged children (Protzko, Aronson, & Blair, 2013).

Reasoning training

In an attempt to increase the IQ of school-aged children, there has been a push for many years to train some of the specific skills tapped by IQ tests—most specifically non-verbal tests. While certain IQ subtests such as vocabulary and information tap knowledge (questions like what does the word vapid mean, or who was Susan B. Anthony), non-verbal tests tap ways of finding relations between objects. This inductive reasoning, finding patterns and commonalities, is one specific component of the mental processes needed to solve non-verbal IQ tests. As non-verbal tests are more frequently being used for admission requirements into selective schools, (e.g. *NYC DoE FAQ, 2013*) it is not surprising that such skills have become more highly prized. Elementary math textbooks in the early 20th century, for example, focused on teaching counting, number concepts, and simple addition; by the end of the 20th century, math texts shifted to heavily teaching items and processes copied from non-verbal IQ tests (Baker et al., 2010).

Reasoning training sometimes suffers from the problem of teaching to the test. Often the teaching process involves training on measures nearly identical to the IQ tests. Some programs explicitly train students on matrix items (e.g. unit 2, Adams, 1986) nearly identical to those from the Ravens' Progressive Matrices and Cattell Culture Fair Intelligence Test. Some programs have children make pictures with different pieces of colored paper (e.g. unit 5, Adams, 1986) similar to block design subtests of some IQ tests. Some programs specifically train children with pictures making up a story and ask them to extrapolate the problems or structure of the story, similar to the picture completion subtest from the WISC (Illustrations instrument from Feuerstein, Rand, Hoffman, & Miller, 1980).

We believe, quite subjectively, that these studies are too similar to teaching to the IQ test. Teaching to the test and practice on adapted problems has been shown to raise IQ scores; those gains, however, have no effect on the central factor common to IQ tests, suggesting that such practice effects do not represent genuine increases in intelligence (te Nijenhuis, van Vianen, & van der Flier, 2007). Similarly, schools have used teaching to the test to raise achievement test scores under the No Child Left Behind law; this has resulted in an increase in academic achievement performance with no carryover to intelligence (Finn et al., 2014).

In addition, one of the reasons one reasoning training program was created was for the purpose of exposing disadvantaged children to the information and procedures common in intelligence testing (Feuerstein et al., 1980). This program has also been shown to have no effect on g-scores of IQ tests (te Nijenhuis et al., 2007).

Some reasoning training paradigms have shown subsequent increases in educational achievement (e.g. Klauer, 1998), a finding that would not be consistent with the totality of effects being from training to the IQ test. Although such practices are admittedly only part of much larger programs, we are too concerned with the similarities of the training material and the posttest material to be confident some of the IQ gains from such programs are not instances of teaching to the test. Therefore, instead of meta-analyzing the literature, we present the relevant citations in Table 1. Overall, we are simply too skeptical to be confident in any conclusions regarding IQ that can be drawn from reasoning training.

Executive function training: a meta-analysis

Executive functions can be considered control mechanisms that orchestrate different processes of human cognition (Miyake et al., 2000). Training this general control is a proposed way to increase intelligence. Standard procedures involve computer training with a program that stresses executive functions. The reason such training could raise IQ is the high correlation between certain executive functions and intelligence (Blair, 2006). In addition, IQ and many executive functions share a similar cognitive architecture (Barbey, Koenigs, & Grafman, 2012). It therefore seems natural that training similar skills in the same parts of the brain should lead to increases in IQ. Results from children may provide especially useful and different results from experiments with adults (e.g. Shipstead, Redick, & Engle, 2012) as executive functions, IQ, and the common neural architecture are still developing in children (Engle, Kane, & Tuholski, 1999).

Methods

We combined working memory training and executive control training under the heading of executive function training, unlike analyses in adults (e.g. Karbach and Verhaeghen, 2014). We did this because abilities like working memory and effortful control represent a strong unitary ability (executive functions) in the ages studied here; it is not until later ages that executive functions diverge into separate constructs (Brydges, Fox, Reid, & Anderson, 2014).

One study only used one subtest (Rueda, Checa, & Combita, 2012) and was thus not included in this meta-analysis. One study included follow-up data after executive function training (Jaeggi, Buchkuehl, Lonides, & Shah, 2011); we only used the

Table 1

List of studies where children are taught reasoning skills.

Study	Notes
Feuerstein et al. (1980)	See Romney and Samuels (2001) for a meta-analysis of effects on disparate outcomes; Picture Completion trained with alternate pictures
Levinson (1971)	Spatial Group is trained with materials from the Stanford-Binet test; Verbal Group is trained in word concepts not included in the posttest
Riding and Powell (1987)	Some lessons like Completing Series and Find the Pattern may be very similar to matrix reasoning
Klauer (1998)	Some lessons such as Cross-Classification & Systems Construction are very similar to matrix reasoning. See Klauer and Phye (1994)
Olton and Crutchfield (1969)	Lessons are mysteries and hypotheticals to be solved using productive thinking; program emphasizes thinking of multiple solutions as opposed to being worried of finding the correct solution the first time
Kafadar, Akıncı, and Çakır (2014)	Children underwent training tasks to improve memory, attention, and reasoning for Verbal, Numeric, and Visual tasks

data from the immediate posttest. Another study included three experimental groups with one control, all included executive functioning training but two additional groups included verbalization and switching stimuli. We only included the basic executive function training in this meta-analysis. This left us with eight studies.

Results

We found eight studies, contributing one effect size each, on 740 children who underwent executive function training for at least two weeks. Results indicated that executive function training may raise a child's IQ ($g = 0.151$, 95%CI = 0.006 to 0.296; see Fig. 4). There was no evidence for heterogeneity within the sample ($Q(7) = 10.47$, $p > 0.233$; $I^2 = 23.6\%$, $p > 0.233$).

Robustness checks. Meta-analytic outliers: There were no meta-analytic outliers present in this analysis; all externally studentized residuals fell below 1.

Missing Studies (Publication Bias): We found evidence of missing studies (publication bias) in this data. To test this, as the number of studies was so small, we used the trim and fill method (Duval and Tweedie, 2000) which has been shown to have high power to detect moderate to severe publication bias even in small meta-analyses (Idris, 2012). This method showed evidence of publication bias through imputing three studies in four iterations. To adjust for this bias, we ran both a WLS and PEESE estimator. The WLS estimator returned the same estimate ($ES = 0.151$) but with wider confidence intervals which included zero 95%CI = -0.044 to 0.346). Furthermore, the PEESE estimator also returned a smaller, non-significant estimate ($ES = 0.023$, 95%CI = -0.088 to 0.134). Therefore, there exists evidence for publication bias in the literature on training children's Executive Functioning. The degree of this bias is enough to conclude that there is not enough evidence to believe such training reliably increases IQ. *p*-hacking adjustment: There was one study to produce statistically significant results at posttest (Zhao, Wang, Liu, & Zhou, 2011). We therefore shrunk their effect sizes to 0.5, which would represent

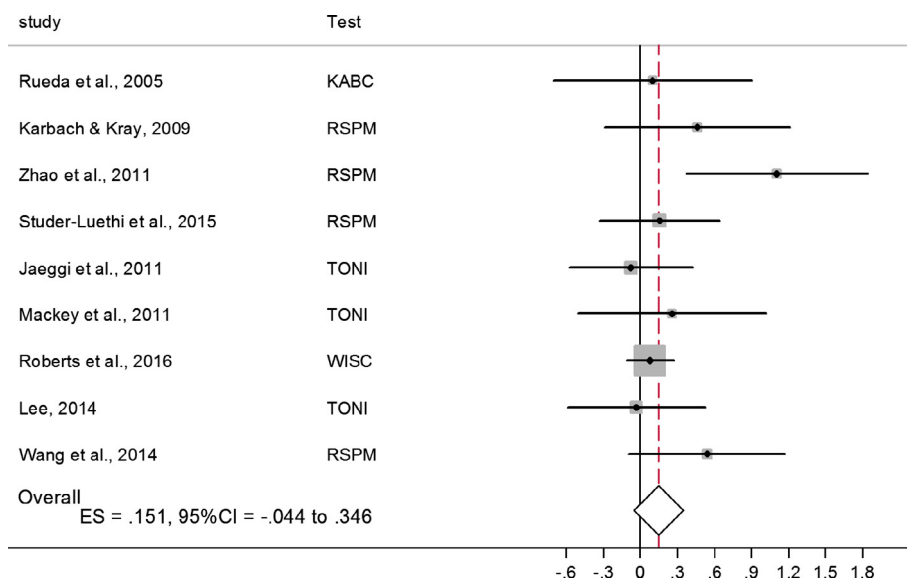


Fig. 4. Effects of executive function training on the IQ of children. Notes: Karbach and Kray (2009) is effortful control training; TONI analyzed for Jaeggi et al. (2011) due to nonstandard administration of the RSPM. WLS estimates corrected for publication bias shown for overall effect.

$p = 0.0505$; and again shrunk the effect sizes to 0.288, which would represent $p = 0.099$. Doing so shrunk the overall effect size, which does not alter the results from the p -hacking adjustment. Therefore, if the results from Zhao et al., 2011 were p -hacked from marginal to statistical significance, we would not see an effect of executive function training on IQ (although this would entail p -hacking from 0.5 to 1.107, which we find very unlikely). We do not believe this is the case, and would not suggest one to ever conclude a researcher engaged in questionable research practices without explicit evidence. What this does highlight, however, is how the change of one estimate can influence the interpretation of results.

Finally, there was one study we included in this meta-analysis where the control group received treatment in speed of processing training (Mackey, Hill, Stone, & Bunge, 2011); as the control group received a non-innocuous treatment the results from this study may underrepresent the true effect size. It could be argued that this would make the inclusion of this study more conservative if the speed-of processing training also had an effect on IQ.

Overall, more work is needed in training school-aged children on executive functions to see if it reliably raises their intelligence, especially at the latent level due to concerns over the specific posttests used. Results from adults suggest executive function training may increase visual mental rotation skills but not underlying intelligence at the latent level (e.g. Colom et al., 2013).

Finally, it is becoming clearer that executive function training may not be an unconditional causal effect; meaning, the effects may be moderated by such factors as personality (Schwaighofer et al., 2015; in adults) or temperament (Studer-Luethi, Bauer, & Perrig, 2015; in children). If this turns out to be true across experiments, we will not be able to make claims about whether executive function training is effective. The truth may be more complicated.

Music lessons and IQ: a meta-analysis

Musicians have many advantages over non-musicians. Other than the ability to make beautiful music, people who know how to play a musical instrument score higher on standardized achievement tests (Hetland and Winner, 2001), exhibit better executive functions (Bugos, Perlstein, McCrae, Brophy, & Bedenbaugh, 2007), have higher IQs (Forgeard, Winner, Norton, & Schlaug, 2008) and higher levels of latent g (Silvia, Thomas, Nusbaum, Beaty, & Hodges, 2016). Children who choose to learn a musical instrument, however, are very different from those who do not; coming from wealthier, two-parent homes (Kinney, 2010), for example. Some studies have shown that musical students do no better on cognitive tests once a host of confounds are taken into account (Elpus, 2013). This is why it is important to investigate the experimental evidence.

Methods

Two studies were excluded because one used only one subtest of an IQ measure (Rauscher, Shaw, & Ky, 1993) and one only taught children musical concepts instead of a musical instrument and did not have enough data to calculate an effect size (Moreno et al., 2011).

Results

We found five RCTs where children learned to play a music instrument. These five studies contributed six effect sizes over 303 participants (see Fig. 5). These interventions involved instruction and practice on musical instruments multiple times per week for many weeks. All participants had no prior training.

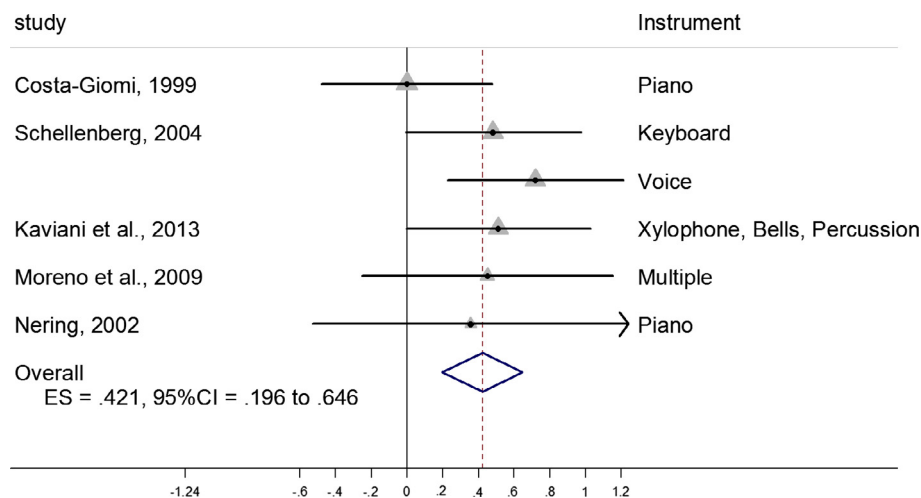


Fig. 5. Effects of learning to play a musical instrument on IQ.

We found that teaching a child a musical instrument raises their IQ by over a third of a standard deviation ($g = 0.421$, 95% CI = 0.196 to 0.646; see Fig. 5). In addition, there was no evidence for heterogeneity in this sample ($Q(5) 4.68$, $p > 0.466$; $I^2 = 0\%$).

Robustness checks. Meta-analytic outliers: There were no meta-analytic outliers present in this analysis; all externally studentized residuals fell below 1.

Missing Studies (Publication Bias): There was mixed evidence of missing studies (publication bias) in our data. Estimation using the Egger test showed no evidence for bias ($p > 0.572$); this test, however, has low power in a meta-analysis of this size to detect bias (Idris, 2012). We also tested publication bias using the more powerful trim and fill method which showed possible evidence of small publication bias. This method, however, also has a tendency towards inflated type I errors (Idris, 2012). We therefore carried out corrections to possible missing studies using multiple estimators (following Reed et al., 2015). The WLS estimator correcting for publication bias produced the same coefficient as the standard estimate ($g = 0.421$) but with a larger confidence interval (95%CI = 0.137 to 0.704) that remained statistically significant. The PESE estimator also returned a similar coefficient ($g = 0.42$, 95%CI = -0.356 to 1.197) but did not show evidence for publication bias ($p > 0.97$). Finally, for an added check, testing publication bias using the Vevea and Hedges (1995) Weight-Function Model also showed no evidence of publication bias at the level of $p = 0.1$ ($p > 0.228$). Therefore, there is little reason to suspect missing studies and most corrections for this possibility do not alter the results. *p*-hacking adjustment: There was one study to produce statistically significant results at posttest (Schellenberg, 2004). We therefore shrunk their effect sizes to 0.493, which would represent $p = 0.0505$; and again shrunk the effect sizes to 0.413, which would represent $p = 0.099$. Though the meta-analytic effect sizes were attenuated, ($g = 0.373$, 95%CI = 0.148 to 0.598; $g = 0.356$, 95%CI = 0.131 to 0.581), they remained statistically significant. Therefore, if the results from Schellenberg were *p*-hacked from marginal to statistical significance, we would still see the effect of learning to play a musical instrument on IQ.

Clustering of effect sizes: One study included two musical experimental groups using the same control group (Schellenberg, 2004). Removing the higher effect size also does not change the results.

Imputed missing study: A study in Los Angeles randomly assigned 29 students to learn a musical instrument for a year or be added to the waitlist to learn the instrument the following year (Slater, Tierney, & Kraus, 2013). One of the measures taken at posttest was the Wechsler Abbreviated Scale of Intelligence, a short form IQ test (D. Strait, personal communication). Unfortunately, the data from this intervention are not yet available and so it cannot be added to this meta-analysis. However, if we impute the data as having absolutely no effect ($ES = 0$; worst case plausible scenario) we find that the overall effect size shrinks to a still significant 0.351 (95%CI = 0.146 to 0.557). Not only would the results of our analysis remain significant, the updated meta-analytic effect size including the worst case scenario would still be within the original confidence interval.

Discussion

To understand how learning to play a musical instrument would raise IQ, we try to understand what can be seen across all of these studies. First, only two of the studies included learning to read sheet music (Costa-Giomi, 1999; Schellenberg, 2004), while the other two did not. Second, some studies focused on a “high-brow” instrument like the piano (Costa-Giomi, 1999; Schellenberg, 2004), while others focused on more “low-brow” instruments like percussion (Kaviani et al., 2013). Therefore, we find it unlikely that any increase is due to priming or expectation effects. Third, two studies included active control groups (drama: Schellenberg, 2004; painting: Moreno et al., 2009); while the other two did not. This suggests the results are unlikely to be due to Hawthorne effects, but does not prove so. In addition, this result seems to be specific to learning to create music (learning an instrument). Simply exposing preschool children to music environments, for example, allowing them to freely dance to music, and singing to and with them does not confer any benefit to IQ (Mehr, Brady, Katz, & Spelke, 2013).

Instead, we argue (very speculatively), musical instrument instruction may raise IQ through strengthening white matter tracts in the prefrontal cortex associated with rhythm perception and discrimination. Rhythm perception and discrimination are one of a family of sensory discrimination tasks (Troche and Rammsayer, 2009). Rhythm ability can be tested a number of ways, two of the most common being discriminating whether two beats have the same rhythms, or tapping in time to a beat and continuing after the beat ends. Musicians are unsurprisingly more accurate at these tasks than non-musicians (Chen, Penhune, & Zatorre, 2008; Rammsayer et al., 2012). Randomly assigning children to learn a musical instrument increases this rhythm ability as well (Slater et al., 2013).

Rhythm ability is related to general intelligence. People who have a better sense of rhythm have higher IQs (Madison, Forsman, Blom, Karabanov, & Ullén, 2009). This remains true when controlling for other forms of sensory discrimination and measuring intelligence as *g* as opposed to an IQ score (Rammsayer and Brandler, 2007). In addition, these relationships remain when controlling for speed of processing and working memory (Troche and Rammsayer, 2009; Troche and Rammsayer, 2011) and complex reaction time (Helmbold, Troche, & Rammsayer, 2007). Overall, sensory discrimination, of which rhythm ability is a part, is directly related to general intelligence. Crucially, aside from being related, intelligence and rhythm ability share white matter tracts in the prefrontal cortex (Ullén, Forsman, Blom, Karabanov, & Madison, 2008)—part of the brain network for intelligence (Jung and Haier, 2007).

Thus, learning to play a musical instrument raises two separate correlates of *g*, IQ scores and rhythm ability. These two separate correlates of *g* share some of the same neural architecture. Therefore, we propose that the reason learning to play a musical instrument raises intelligence could be through strengthening white matter tracts used in rhythm ability and

sensory discrimination, white matter tracts that are shared by general intelligence. To prove this mechanism, research should focus on the causal effect of learning to play a musical instrument on such shared white matter tracts. Though we believe this is a plausible account, we are scientists, not logicians. As such, very little can be guaranteed. Music training may only increase working memory, or some other component of intelligence.

Supplemental evidence can help strengthen the case for this shared white-matter tracts mechanism. Changes in other white matter tracts have already been shown in response to memory training (Engvig et al., 2012), mental calculation training (Hu et al., 2011), juggling training (Scholz, Klein, Behrens, & Johansen-Berg, 2009). Separately, training children in rhythm and tapping alone (without music instruments) causes an increase in academic achievement (Taub, McGrew, & Keith, 2007; Taub, McGrew, & Keith, 2015). Though the evidence here does not prove this causal mechanism, the weight of evidence suggests it would be a profitable place to look to better understand the causal effect of learning to play a musical instrument on IQ.

General discussion

Overview of nutrition interventions

The work presented here shows that multivitamins and iodine supplementation reliably raises the IQs of mildly deficient children. The results from other nutrients are either not reliable or absent. It is important to stress that the above work only uses children who are mildly deficient for overall nutrient intake.

This work is relatively silent on the effects of supplementing children who are not nutritionally deficient. The work on multivitamins suggests that complementing an already replete diet with either multivitamins or iodine would not have an effect on IQ. In addition, there may be dangers of over-supplementation to children who are nutritionally replete. Work should be conducted in a safe manner on non-deficient children to examine the effects of such supplementation on IQ.

It should be noted that some of these studies are conducted in non-Western countries. Some could consider this a source of bias in the use of IQ tests that are created and normed on Western samples. Most studies, however, use some version of non-verbal tests such as the Ravens Coloured Progressive Matrices (Raven, Raven, & Court, 1962) or the Test of Nonverbal Intelligence (Brown, Sherbenou, & Johnsen, 1988) or the Cattell Culture Free Intelligence Test (Cattell and Cattell, 1960; exceptions to this are: Nidich et al., 1993; Shrestha, 1994, and Osendarp et al., 2007). Debate on whether these non-verbal tests measure intelligence similarly in non-Western samples is beyond the scope of this paper. However, if a nutrient deficiency can lower scores on a test purporting to measure intelligence, and the remediation of that deficiency can raise scores on that test, that stands as evidence towards the validity that the test is indeed measuring some underlying ability (Wilson, 2005). This is hardly definitive and a better treatment of this debate can be found elsewhere (e.g. Brouwers, Mishra, & Van de Vijver, 2006; Jensen, 1998; Sternberg and Grigorenko, 2004).

Overview of environmental interventions

We see that executive function training may not have an effect on IQ; however, the results may also be limited to mental rotation skills. This would be in line with the results from adulthood. Either way, more work in school-aged children is needed before drawing any firm conclusions. The implications of whether EF training can transfer to intelligence has strong theoretical implications. Theories of the development of intelligence often rely on the assumption of causal connections between executive functioning and the rest of cognitive life (e.g. Kovacs and Conway, 2016). The ability for EF training to transfer is central to testing the causal implication of such arguments. While the results of such training have not upheld in adults (e.g. Melby-Lervåg et al., 2016), it has been suggested that this failure of transfer may be due to ceiling effects in individuals Executive functioning due to the ubiquity of use in daily life (Oberauer, 2016). If this line of reasoning is correct, the promise of increasing EF in childhood should hold the promise of not only transfer, but also tests of the validity of the causal connections between EF and *g*. The results of this synthesis do not unanimously uphold such causal connections, so it may be that EF, while trainable, is not causally related to intelligence (see Protzko, 2016b).

Learning to play a musical instrument, as shown here, raises the IQ of children. We posit that the reason this occurs is from strengthening the white matter tracts in the PFC that are shared by both general intelligence and rhythm ability. Neurological work is needed to confirm our prediction as this mechanism.

One concern with a project of this type is some may believe that the evidence provided here somehow excludes other causal possibilities. This paper is silent on such variables as schooling, family income, and literacy (e.g. Ritchie, Bates, & Plomin, 2015) as potential causes in intellectual development. It would be mistaken to assume that because they are excluded, this manuscript is claiming they could not be causes. To our knowledge there is no RCT that randomly assigned children to either attend or not attend school; no RCT that randomly assigned children to grow up in wealthy or poor homes; no RCT that teaches only some children to read. Quasi-experimental, “natural” experiments, regression discontinuity designs, and advanced statistical analyses have argued that schooling increases IQ (e.g. Cahan and Cohen, 1989; Ceci and Williams, 1997; Winship and Korenman, 1997; Brinch and Galloway, 2012); while others argue this increase is not to *g* but instead to extraneous variance on IQ tests (e.g. Ritchie et al., 2015; Tommasi et al., 2015) or school does not raise IQ at all (Brouwers et al., 2006). Similar arguments have been made regarding socioeconomic status (e.g. Hansen, Heckman, & Mullen, 2002; Reardon, 2011), as cash transfers to poor families have not shown positive effects on IQ

(Paxson and Schady, 2010), for example. While we are strong proponents of such approaches when individual-level RCTs are not possible, that is not the aim of this paper.

Potential limitations

The goal of this paper is not to be the final word on raising intelligence in school-aged children—but instead to be the aggregation and analysis of all of the fully causal work on the subject to date. This analysis is subject to the same limitation as any meta-analysis, that of inclusion criteria. We argue that the criteria we used are a benefit and not a limitation.

Focusing on individual-level RCTs removes the possibility of including cluster-randomized interventions (e.g. classroom, school, village), “natural” experiments, and advanced statistical analyses (instrumental variable, propensity-score). The reason for the restriction to individual-level randomization is to create a set of studies that only conform to the gold standard of causal analysis. Cluster-randomized studies, such as classroom- or school-randomized studies, can approximate individual-level counterfactual causal claims only under certain conditions, minimally such as having at least four to seven clusters (classrooms, schools, villages) per condition and the analysis taking into account the nesting of individuals within clusters on top of demonstrating a lack of or at least accounting for biases between clusters (e.g. Torgerson and Torgerson, 2008). Most of the cluster-randomized trials in intelligence research fall short of these minimums (see Herrnstein et al., 1986, for example).

It should also not be assumed that every RCT is perfectly conducted. Many of these problems, such as transfer of information from experimental to control groups, or efficacy of the manipulation, are likely to downward bias all results. Aggregating experimental studies via meta-analysis is one way to prevent imperfections of a single study from influencing an entire field. Again, we believe the wealth of experimental data on raising intelligence is not fully known or appreciated. Meta-analyses are only as good as the data they contain. As more work is done in these fields, we can get a clearer picture of the causal efficacy of environments in the development and raising of intelligence.

The first step in much of science is to establish causality—to establish *that x causes y*. In this case, to establish that certain environmental manipulations cause changes in intelligence. Once causality can be established, then searches for mechanisms and moderators can commence. It is the goal of this investigation to first assess the plausibility of causality. The question of whether the effects are moderated by study characteristics depends on first showing heterogeneity across studies. This heterogeneity was not found in the meta-analyses on multivitamins, executive function training, or learning to play a musical instrument. Unfortunately, power to detect heterogeneity with either the Q or I^2 is low in meta-analyses with low number of studies (Huedo-Medina et al., 2006) and cannot be completely discounted.

Range restriction is also a topic of concern. Specifically, participants in most of these studies represent those drawn from poverty. This causes a downward restriction in the total variance of intelligence within the samples. While this does not cause problems for within-study causal estimates (due to randomization), it does make understanding the generalizability to the rest of the population difficult. An intuitive fix would be to replace the sample standard deviations with population standard deviations (15 for IQ). This creates an unnecessarily conservative approach that necessarily depressed effect estimates. To understand the likely effect size in non-restricted populations, more advanced corrections for range restriction must be pursued.

As children in these studies generally come from impoverished environments, the cause of the range restriction is partly due to pre-existing cognitive ability, and partly due to other factors. As such, we can correct for this direct and indirect range restriction to understand the likely effects of the interventions to broader populations.² We therefore conducted a correction for range restriction on the meta-analytic estimates (see Hunter, Schmidt, & Le, 2006) for music training only to find the ‘practical’ IQ points return (see below). Correcting for range restriction in the nutritional interventions assumes homogeneity of results across populations. For multivitamins, we know this is untrue. For iodine, we do not know empirically but are doubtful iodine supplementation in non-deficient populations will have any effect.

The topic of raising intelligence is extremely important and extremely contentious. The belief that intelligence cannot be raised is strong (Haier, 2014; for example) and is unlikely to ever completely go away. The purpose of this project, and this paper, is to provide the scientific audience with a complete account of the experimental evidence on raising intelligence. If readers are concerned about the small number of studies, we suggest they are looking at the wrong number. These analyses are over hundreds if not thousands of children. The strongest causal evidence cannot be ignored, the inclusion of supplementary evidence would at best fall in line with the causal evidence, and at worst bias the results.

We make every effort to include every intervention in our study that meets the inclusion criteria. We also acknowledge that additional studies may exist; some may even be under way at the time of writing and publication. It is for this reason that we are updating continually as new studies are published and file drawers are opened. We invite scientists in all fields who have completed or are in the process of conducting studies that meet our inclusion criteria to send their results or relevant publications.

Are the gains to IQ gains to Intelligence?

There is a danger of equating manifest scores with latent traits. Although IQ tests represent exceptionally valid indicators of the underlying latent trait (e.g. Jensen, 1998), changes in the test do not *necessarily* correspond to changes in the trait. Work on non-experimental groups have often showed group differences to IQ do not correlate with latent intelligence differences

² We would like to thank Dr. Huy Le for his clarification on this point.

Table 2

List of all studies included in this meta-analysis. Effect size estimates have been trimmed to 3 decimal places. (H) indicates substantial heterogeneity.

Type of intervention	Citation	n _E	n _C	ES	Test used	Country
<i>Multivitamins</i>						
	Maseck and Engr (1980)	11	8	0.142	PPVT	USA
	Nelson et al. (1990)	25	25	0.385	Heim AH4	UK
	Nelson et al. (1990)	80	80	0.072	Heim AH4	UK
	Crombie et al. (1990)	26	47	-0.013	RCPM	UK
	Todman et al. (1991)	36	36	0.267	DH-4	UK
	Benton and Cook (1991)	22	22	0.866	BAS	UK
	Nidich et al. (1993)	18	16	0.018	C-FIT	USA
	Southon et al. (1994)	10	9	0.367	WISC-R	UK
	Southon et al. (1994)	12	20	-0.147	WISC-R	UK
	Snowden (1997)	11	7	1.027	Calvert	UK
	Naismith, Nelson, Burley, and Gatenby (1988)	77	77	0.035	WISC-R	UK
	Wender	11	7	0.613		
	Schoenthaler, Bier, Young, Nichols, and Jansenns (2000)	125	120	0.221	WISC-R nv	USA
	Solon et al. (2003)	185	183	-0.018	PMAT	Philippines
	Khan et al. (2004)	56	55	-0.086	RCPM	Bangladesh
	Osendarp et al. (2007)	66	70	0	g	Australia
	Osendarp et al. (2007)	61	54	-0.130	g	Indonesia
	Nga et al. (2011)	114	119	0.167	RCPM	Vietnam
Overall effect		0.097, 95%CI = 0.006 to 0.187				
<i>Iodine supplementation</i>						
	Shrestha (1994)	72	72	3.017	gF	Malawi
	Huda, Grantham-McGregor, and Tomkins (2001)	145	142	0.087	RCPM	Bangladesh
	Zimmerman et al. (2006)	159	151	0.586	RCPM	Albania
	Gordon et al. (2009)	84	82	0.19	Composite	New Zealand
Overall Effect		0.531, 95%CI = 0.394 to 0.668 (H)				
<i>Iron supplementation</i>						
	Gopaldes, Kale, and Bhardwaj (1985)	5	4	0.82	G-WISC	India
	Gopaldes et al. (1985)	4	4	0.794	G-WISC	India
	Pollitt et al. (1985)	19	21	0	PPVT	Egypt
	Soemantri (1989)	37	35	0.091	RCPM	Indonesia
	Pollitt, Hathirat, Kotchabhakdi, Missell, and Valyasevi (1989)	605	605	0	RCPM	Thailand
	Shrestha (1994)	76	72	1.298	gF	Malawi
	Lynn and Harland (1998)	208	205	0.156	RSPM	UK
	Sungthong et al. (2004)	139	122	-0.5	TONI-II	Thailand
	Rico et al. (2006)	138	128	-0.293	PPVT	Mexico
Overall effect		0.032, 95%CI = -0.048 to 0.111 (H)				
<i>EF training</i>						
	Rueda, Rothbart, McCandliss, Saccomanno, and Posner (2005)	12	12	0.1	KABC	USA
	Karbach and Kray (2009)	14	14	0.461	RSPM	Germany
	Zhao et al. (2011)	16	17	1.107	RSPM	China
	Studer-Luethi et al. (2015)	34	31	0.158	RAPM	Switzerland
	Jaeggi et al. (2011)	32	30	0.15	RSPM	USA
		32	30	-0.075	TONI	USA
	Mackey et al. (2011)	17	11	0.257	TONI	USA
	Roberts et al. (2016)	205	205	0.079	WISC	Australia
	Lee (2014)	25	25	-0.03	TONI	USA
	Wang, Zhou, and Shah (2014)	20	20	0.54	RSPM	China; 20 days group
Overall effect		0.151, 95%CI = -0.044 to 0.346				
<i>Musical instrument</i>						
	Costa-Giomi (1999)	32	35	0	DCAT	Canada
	Schellenberg (2004)	30	36	0.484	WISC	Canada
	Schellenberg (2004)	32	36	0.72	WISC	Canada
	Kaviani et al. (2013)	30	30	0.513	SB-IV	Iran
	Moreno et al. (2009)	16	16	0.45	WISC-III	Portugal
	Nering (2002)	10	10	0.357	WPSSI/WISC	USA
Overall effect		0.421, 95%CI = 0.196 to 0.646				

(e.g. Flynn, te Nijenhuis, & Metzzen, 2014), however these studies often fail to test to make sure the groups are retaining the same factor structure (e.g. Wicherts et al., 2004), a minimal requirement for latent-level group comparisons (see Jensen, 1998). Experimental tests have shown that retest effects increase IQ without altering *g* (Estrada, Ferrer, Abad, Román, & Colom, 2015). Intense early interventions, however, have shown to raise both IQ scores and latent *g* (Protzko, 2016a).

Fully testing for these effects require access to either the raw data of covariance matrices, neither of which we have access. Looking at patterns across subtests may be worthwhile, but this requires that we are correct about the causal structure of human abilities. If, for example, a hierarchical model is the true underlying causal model of human cognitive abilities, then gains to *g* should be proportional to sub-factors based on the loading from *g*. If, however, a bi-factor model is correct, gains to *g* can be seen without any increase to specialized abilities. This dichotomy of predictions represents a strong falsification test of the two models, and should be the focus of future experimental psychometric work (see Protzko, 2016b).

If all of the results here are to latent intelligence, then it represents another step forward in the understanding of the small but non-negligible effects of environmental differences on the development of intelligence. If the results are consistently *not* to latent intelligence, then it is important to understand what, if any, practical implications exist. Non-*g* variance of IQ tests still has predictive utility for both academic and economic outcomes (Coyle and Pillow, 2008; Woodley, 2012; respectively).

Conclusion

The present work has been a collection of nearly all of the data on raising the intelligence of school-aged children (see Table 2). From our analyses, we may draw the following tentative conclusions:

- Studies supplementing inadequate diets with multivitamins raised IQ
- Studies remediating mild iodine deficiency raised IQ
- Studies involving teaching a musical instrument raised IQ. After correcting for range restriction, this corresponded to an increase of 4.002 IQ points in the population.

We currently lack the data to determine whether any of the other interventions we examined are effective. Some of these meta-analyses are also met with heterogeneity, meaning more work needs to be done in each of these fields before we can firm in our conclusions. The methods here provide stricter controls on experimental methodology and intelligence measures than have been conducted in previous meta-analyses (explained in the inclusion criteria).

This analysis suggests some new avenues of research, aside from the need for additional replication. The analysis of iodine supplementation brings up the question of whether supplementing replete children would confer any benefit to IQ. It also brings up the possibility that supplementing deficient individuals would alter the white-matter architecture of their brain. The analysis of music instruction suggests rhythm may be correlated with IQ and specifically training rhythm in children may raise their IQ without the need to learn a musical instrument directly. What is most important is to understand that the results presented here also tell us about the sample sizes needed to further investigate effects. In the analysis with the largest effects (iodine supplementation), to achieve 80% power in future investigations requires a sample size of 57 participants per group. This grows larger as we move to interventions with smaller effects, such as learning to play a musical instrument (sample size for 80% power would be $n = 94$ per group). This is larger than any previous investigation, suggesting future investigations will also have to greatly increase the size of samples to confirm these effects as well as provide well-powered tests.

Future research should also focus on what factors may moderate the effectiveness of these interventions. Moderation of environmental effects on intelligence can include genetic moderation (e.g. Söderqvist, Matsson, Peyrard-Janvid, Kere, & Klingberg, 2013) or moderation by other psychological variables (e.g. Studer-Luethi et al., 2015). An environmental effect with no net effect may indeed have strong effects in a sub-population. An effect with strong net effects may have none in others. Finding, and more importantly, replicating these moderations will better help us understand the interplay of what small environmental effects may exist in intelligence.

In the end, it would be nicer if the bulk of our experimental knowledge pointed to a consistent story: yes intelligence can be raised by anything; no intelligence cannot be raised at all. The truth, however, is under no obligation to be a unified story to us. There have been hundreds of attempts to raise intelligence, some of them fail, some of them succeed. Focusing only on studies which push a narrative (i.e. mutability, immutability) prevents the field from seeing the whole picture. Not everything works—but some things do.

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* = studies that were included in a meta-analysis

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