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How to Make a Young Child Smarter: Evidence From the Database of Raising Intelligence

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Abstract

Can interventions meaningfully increase intelligence? If so, how? The Database of Raising Intelligence is a continuously updated compendium of randomized controlled trials that were designed to increase intelligence. In this article, the authors examine nearly every available intervention involving children from birth to kindergarten, using meta-analytic procedures when more than 3 studies tested similar methods and reviewing interventions when too few were available for meta-analysis. This yielded 4 meta-analyses on the effects of dietary supplementation to pregnant mothers and neonates, early educational interventions, interactive reading, and sending a child to preschool. All 4 meta-analyses yielded significant results: Supplementing infants with long-chain polyunsaturated fatty acids, enrolling children in early educational interventions, reading to children in an interactive manner, and sending children to preschool all raise the intelligence of young children.

Keywords

intelligence, developmental psychology, early childhood, interventions, preschool

A good deal of research confirms what most people consider self-evident: Intelligence matters for academic and life success (Herrnstein & Murray, 1994; Nisbett et al., 2012). Accordingly, many researchers and educators have attempted to increase the intelligence of children—particularly children from disadvantaged backgrounds. The question of the modifiability of intelligence remains a contentious one, and findings from these famous interventions are nuanced enough to support the conclusions of either side in the debate about the nature and nurture of intelligence. Thus, effective interventions are cited as evidence by "IQ environmentalists" to make the case that intervention can raise IQ (e.g., Nisbett, 2009), whereas "IQ nativists" cite the same results when making the case that such gains are fleeting or even illusory (e.g., Herrnstein & Murray, 1994). We believe that a more complete consideration of the best available data is required if we are to determine whether interventions can meaningfully raise IQ and, if they can, to consider how we might go about constructing these interventions. To this end, we have compiled all available and relevant high-quality studies into a central location, the Database of Raising Intelligence (DORI).

Database of Raising Intelligence

The DORI is a continuously updated compendium of every randomized controlled trial (RCT) designed to increase intelligence across all age levels. To be included in the database, a study has to include each of the following components: a sample drawn from a general, nonclinical population; a pure randomized controlled experimental design; a sustained intervention; and a widely accepted, standardized measure of intelligence as an outcome variable.

Our first criterion is that the participants must have been 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 his or her disabilities or intellectual deficits (e.g., Klingberg et al., 2005, on training working memory of children with attention deficit/hyperactivity disorder) may not have the same effects for members of a nonclinical population. In addition, the amelioration of mental retardation may entail different processes than the general increase of intelligence (Spitz, 1986).

Our second criterion is that the authors must have used a pure RCT. At the beginning of the study, every participant 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

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control group, used classroom or cluster randomized trials, or compared an experimental group with a group selected *after the fact*.

Our third criterion is that the intervention must be sustained and not a "one-shot" treatment. Some authors alter the procedures, instructions, or context of intelligence test administration to examine effects on IQ scores. Studies on "score optimization," which involves experimentally modifying the behavior of intelligence testers, often yield significant effects on the IQ scores participants obtained (Zigler & Butterfield, 1968); however, these effects are obtained as a result of the one-time manipulation during the testing experience. Although such interventions are informative, we exclude them from DORI because their effects may reflect the role of extraneous factors in the testing environment rather than genuine increases in underlying intelligence.

Finally, only studies where the authors use widely accepted standardized measures of intelligence are included in DORI. Studies where authors incorporated tests of infant cognition were included only if they demonstrated a substantial relationship with measures of later cognitive ability. The two most common tests of infant cognition are the Bayley Scales of Infant and Toddler Development (Bayley, 1969) and the Fagan Test of Infant Intelligence (FTII; Fagan & Shepard, 1987). The Bayley test is used to measure early motor and developmental behavior (e.g., copying the experimenter in arranging blocks into a pyramid, pointing to the same item on a page that an administrator had pointed to), but its scores have little or no relationship to later cognitive ability (e.g., SAT) or to indices of adult IQ (Columbo, 1993). The FTII, by contrast, is a measure of information processing (specifically, how long a child takes to habituate to a stimulus); its scores significantly predict SAT scores 16 years later ($r = .59$; Fagan, Holland, & Wheeler, 2007). DORI thus includes interventions in which experimenters used the FTII but excludes those in which experimenters used the Bayley test.

Interventions that influence academic achievement (e.g., grades and other metrics of performance in school), although of obvious importance, do not qualify for inclusion because such measures involve more than intelligence. Academic tests ideally measure knowledge, intelligence, and motivation. The individual effects of each of these inputs cannot be separated fully.

Overview of Analyses

In this study, we examine a subset of DORI consisting of those experiments involving young children (from the prenatal period through 5 years of age). We coded all studies into effect sizes based on the postintervention differences in intelligence scores. In cases where no standard deviation data were available, we contacted the authors for the data. If the authors or the data were unavailable, we imputed the standard deviations by using the value from the standardization sample (most commonly 15 or 16). This represents a conservative approach, as many studies use restricted samples, reducing the standard deviation of the sample and increasing the effect sizes; using the larger standard deviation of the standardization sample deflates the postintervention effect sizes.

In our survey of both the published and unpublished literature, we found 74 interventions that were designed to raise the intelligence of young children and met our inclusion criteria. These 73 interventions yielded 181 effect sizes across 37,773 participants. However, only four types of interventions were numerous enough to allow meta-analysis.

The first half of the article describes research on nutritional supplement to mothers and children. Our meta-analysis examined the effects of providing long-chain polyunsaturated fatty acids (LC-PUFA) supplements to pregnant or breast-feeding mothers or directly to neonates and young children. We also examined studies where the authors supplemented young children with other nutrients.

The second half of the article describes research on environmental changes. In our second meta-analysis, we evaluated early educational interventions conducted before children began preschool. These include enrichment of the child's environment through home visits, parent training, special child development centers, or a combination of the three. In our third meta-analysis, we examined the effects of engaging young children in an interactive reading intervention at home. With our fourth meta-analysis, we examined the effects of sending a young child to preschool. Throughout the article, we also review the remaining interventions with too few replications to enable meta-analyses. All statistical tests and analyses are in the Appendix.

Nutritional Supplements

We first analyzed the effects of several nutritional supplements given to expecting mothers, new mothers, and their children in the hopes of raising the children's intelligence. We discuss each of these interventions, starting with those we are most confident can raise intelligence, as well as possible causal mechanisms. Readers should note that the causal mechanisms discussed in this article 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.

LC-PUFA supplements and intelligence: A meta-analysis

Using correlational studies, researchers have found that breast-fed children are more intelligent than their bottle-fed counterparts (Anderson, Johnstone, & Remley, 1999). Because many infant formulae do not contain breast milk's LC-PUFA, which is essential for nerve development, researchers interested in raising intelligence began studying the effects of supplementing formula with LC-PUFA.[1]

Our search of the literature (see Appendix) finally yielded 10 effect sizes across 844 participants (see Table 1). In these studies, mothers' diets were supplemented with over 1,000 mg

Table 1. Studies Used in the Meta-Analysis on the Effects of LC-PUFA Supplementation on IQ.

Study	Test	*g*	Duration (years)
Judge, Harel, & Lammi-Keefe, 2007	Fagan	−0.48	0.3
O'Connor et al., 2003; LC-PUFA from egg	Fagan	0.07	0.59
O'Connor et al., 2003; LC-PUFA from fish	Fagan	0.08	0.59
Auestad et al., 2001; LC-PUFA from fish	Fagan	0.05	0.64
Auestad et al., 2001; LC-PUFA from egg	Fagan	0.15	0.64
Werkman & Carlson, 1996	Fagan	0	0.75
Average (mixed)		−0.06	
Birch et al., 2007	WPPSI–R	0.37	0.33
Dunstan, Simmer, Dixon, & Prescott, 2008	PPVT	0.4	0.56
Helland, Smith, Saarem, Saugstad, & Drevon, 2003	K-ABC	0.44	1
Auestad et al., 2003	S-B L-M	−0.29	1
Average (Mixed)		0.24	

Note. LC-PUFA = long-chain polyunsaturated fatty acids; Fagan = Fagan Test of Infant Intelligence; WPPSI–R = Wechsler Preschool and Primary Scale of Intelligence, Revised; PPVT = Peabody Picture Vocabulary Test; K-ABC = Kaufman Assessment Battery for Children; S-B L-M = Stanford-Binet Intelligence Scale, Form L-M.

of LC-PUFA, specifically docosahexaenoic acid (DHA), per day; infants who were supplemented received formula that ranged from 0.2% to 0.5% LC-PUFA.

We found that supplementing either a pregnant mother or supplementing infant formula with LC-PUFA raises a young child's IQ by more than 3.5 points[2] (g = 0.236, 95% $CI_{\text{Weighted Variance}}$ = .043 to .429); the benefits to LC-PUFA supplementation does not appear when the children are tested in infancy (g = −0.064).

LC-PUFA plus arachidonic acid supplements and intelligence

Two studies from the LC-PUFA supplementation literature merit closer attention, as they included a condition in which infants' diets were supplemented with not only DHA but also arachidonic acid (ARA),[3] a second, nonessential fatty acid. Although the two interventions introduced supplements to neonates' diets during the same time period (within 5 days after birth), they differed in duration. Whereas neonates in one study received supplements for only 3 months, those in the other continued on DHA- and ARA-supplemented diets for 12 months. It is perhaps surprising, then, that the shorter intervention proved more effective; toddlers who had received the supplements scored 6.5 IQ points higher than their peers in the control group who had not received dietary supplements (g = 0.54, $p < .001$; Birch et al., 2007). Participants in the longer intervention showed no such benefit (g = −0.142; Auestad et al., 2003). A simple average of the two effect sizes yields a small effect (g = 0.199); a weighted average yields an insignificant effect (g = 0.092). This pattern of results does not provide sufficient evidence for us to conclude that supplementing neonates' diets with both DHA and ARA will contribute to higher IQs in young childhood.

Our meta-analysis indicates that supplementing pregnant mothers' or neonates' diets with LC-PUFA raises a young child's IQ. There is a large literature explaining how such supplementation works. LC-PUFA are considered essential fatty acids because they provide the building blocks for nerve cell development that the body cannot produce on its own (Kurlak & Stephenson, 1999). Lipids (of which LC-PUFA are one class) make up the majority of dry matter in the brain, and during periods of deprivation, they are preferentially depleted from all of the body's organs but the brain (Salem, Kim, & Yergey, 1986).

Other researchers have found that supplementing the diets of children diagnosed with attention deficit/hyperactivity disorder with LC-PUFA can reduce attention problems, impulse problems, and inhibitory control problems (Stevens et al., 2003), all of which are mediated by the prefrontal cortex (Barkley, 1997). Therefore, it is probable that LC-PUFA supplementation raises IQ by providing critical resources for synaptogenesis, which neonates' brains then allocate to the development of the prefrontal cortex. Partial confirmation of this hypothesis comes from a study of brain activation after 8 weeks of LC-PUFA supplementation; children who had received these supplements demonstrated more baseline activation in the left dorsolateral prefrontal cortex than did those who had not (McNamara et al., 2009).

Overall, the research suggests that supplementing pregnant women's, breast-feeding women's, and neonates' diets with LC-PUFA increases young children's IQ; our analysis here confirms this finding.

Other nutrients

In additional interventions, researchers supplemented mothers' or young children's diets with iron; thiamine, ascorbic acid, and B-complex vitamins; multivitamins, or zinc.

Iron. Authors of several studies provided pregnant women and young children with iron supplements in the hopes of

increasing these children's intelligence. In one, researchers randomly assigned pregnant women to receive either 20 mg of iron supplements or a placebo daily for the final 20 weeks of their pregnancies. The supplementing of these pregnant women's diets with iron had no effect on the children's IQ by the time they had reached the age of 4 (Zhou, Gibson, Crowther, Baghurst, & Makrides, 2006). A large-scale RCT in Chile, in which infants were randomly assigned to receive either iron-supplemented formula or unsupplemented formula for the second 6 months of life, produced similarly disappointing results (Lozoff et al., 2003).

Although providing iron supplements to pregnant mothers and infants may not boost young children's IQ, introducing them later in a child's life might. In one study, researchers randomly assigned 35 Indian children (M = 5.5 years old) to receive either 20 mg of iron and 0.1 mg of folic acid or a placebo daily for 60 days. Although the experimental group had a preexisting 10-point IQ advantage over the control group prior to receiving the iron and folic acid supplements, supplementation boosted its members' IQ, widening that gap to 23 points (g = 2.205). In light of these results, it seems that iron supplements may need to be administered during a specific period of development if they are to have salutary effects on young children's IQ.

A possible mechanism for iron's contributions to a young child's intelligence can be found in the reversibility of iron-deficiency anemia's detrimental cognitive effects. Children who fail to consume enough iron during their early development often develop iron-deficiency anemia, in which the body has too few oxygen-carrying red blood cells (American Society of Hematology, 2011). Frequently the children also suffer from cognitive delays that result from the anemia. When the brain lacks iron, it loses its ability to metabolize dopamine in neuronal areas of the cortex responsible for attention (Beard, 1995; Pollitt, 1993) and working memory (Watanabe, Kodama, & Hikosaka, 1997). Administering iron supplements to anemic children can reverse these cognitive delays (Idjradinata & Pollitt, 1993).

This reversibility of anemia's detrimental effects is consistent with the evidence that early iron supplementation is effective when intelligence is tested immediately after administration of the supplements (Seshadri & Gopaldas, 1989) but ineffective when there is a delay between administration of the supplements and intelligence testing (Zhou et al., 2006). Iron supplementation allows the brain to temporarily reallocate resources to dopamine transmission, which is responsible for attention processes and working memory; once supplementation is terminated, its salutary effects fade.

This argument may be frustrated by the failure of the large-scale Chilean iron supplementation study to improve scores on the FTII (Lozoff et al., 2003). Although the iron-supplemented children had longer looking times than did the control children (which may represent increased ability in attention), novelty preference—the FTII's benchmark—was unaffected. The null effect on the FTII, a test of information processing, paired with the observed increases in overall looking times, is consistent with reactive dopamine behavior. Therefore, although we cannot state with full confidence that iron supplementation raises a young child's IQ, the hypothesis that iron supplementation can produce salutary effects on IQ by influencing dopamine transmission related to attention processes and working memory capacity in the frontal cortex merits further investigation.

Thiamine, ascorbic acid, and B-complex vitamins. The work on thiamine, ascorbic acid, and B-complex vitamins comes primarily from one intervention conducted at two sites (Harrell, Woodyard, & Gates, 1955). Hundreds of expecting mothers at both sites (total N = 2,003) were randomly assigned to receive one of four supplements: (a) 200 mg of ascorbic acid; (b) 2 mg of thiamine (Vitamin B_1); (c) a B-complex vitamin consisting of 2 mg of thiamine, 4 mg of riboflavin (Vitamin B_2), 20 mg of niacin (Vitamin B_3), and 15 mg of iron; or (d) a placebo. Participants were instructed to take their assigned supplements daily during the last trimester of pregnancy and the first 6 months of their children's lives.

The results differed by location. None of the supplements effectively increased IQ for children of participants at the first site; all three supplements were effective at the second site, with significant gains in IQ for children who received thiamine (g = 0.358), ascorbic acid (g = 0.255), and B-complex vitamins (g = 0.507). The authors suggested that these site-specific differences in effectiveness resulted from differences in mothers' diets and level of fidelity to the intervention. Mothers participating at the site where the supplementation failed to yield higher IQs had significantly better prenatal diets than did mothers participating at the site where supplementation produced IQ gains. Therefore, it is only with extreme caution that we conclude that B-complex vitamins can effectively raise young children's IQ.

There is not enough research on the effects of riboflavin, thiamine, and niacin on cognitive ability to draw confident conclusions about the mechanisms by which they might beneficially affect a young child's intelligence. However, we can speculate that these B-complex vitamins interact with other nutrients in the body and that their salutary effects result from these interactions. Specifically, each of the three B-complex vitamins supplemented shares interaction effects with Vitamin B_6 (Sauberlich, 1980), an important vitamin for the use-dependent growth of dendrites, specifically in the neocortex (Guilarte, 1993). Depriving rat neonates of Vitamin B_6 alters the behavior of the NMDA receptor-ion channel through a reduction in the number of glutamate- and glycerin-dependent 3[H]-MK-801 binding sites in the cortex (Guilarte & Miceli, 1992). This system is important for synaptic plasticity and dendritic arborization (Cotman et al., 1989); blocking NMDA glutamate receptors in rats prevents the growth of cells and dendrites in the cortex in response to novel experiences (Rema, Armstrong-James, & Ebner, 1998). In simpler terms, as the brain grows in response to new information and activity, it needs B_6 in order to make new nerves.

Generalizing from the results of studies conducted with rats to humans is not straightforward, especially when the human trials are inconsistent. However, if researchers do replicate the finding that B-complex supplementation can produce IQ gains in future studies, they would be wise to consider the role the interactions of these vitamins with B_6 play in use-dependent brain plasticity in their attempts to identify causal mechanisms.

Multivitamins. The majority of interventions involving multivitamin supplementation have been conducted with samples of school-aged children, with only one study where authors tested children before they entered school (Boggs, Scheaf, Santoro, & Ritzman, 1985). Multivitamin supplementation did yield some beneficial effects in an intervention conducted with Head Start children. Researchers randomly assigned six preschoolers to receive either daily multivitamins or placebos for 1 year. Before receiving the multivitamins, the children in the experimental group had tested 20 IQ points lower than those in the control group, a product of random sampling; after 1 year of supplementation, they had gained 3.6 more IQ points than the control group (Boggs et al., 1985). With such a small sample and such large differences in preintervention IQ, this study does not provide enough evidence to make a firm statement on the effects of multivitamin supplementation on young children's IQ.

Zinc. In a single study, women in their third trimester were randomly assigned to receive 25 mg of zinc supplements or a placebo daily through the remainder of their pregnancies. When researchers tested these women's children 5 years later, zinc supplementation had no effect on their children's IQ (Tamura, Goldenberg, Ramey, Nelson, & Chapman, 2003).

Overall, we see that most attempts to raise the IQ of young children through peri- and prenatal supplementation have not been effective. We can be confident that supplementing with LC-PUFA raises the IQ, and we can tentatively assert that supplementing with B-complex vitamins also does so. In addition, the work on iron supplementation is scattered enough that more work needs to be done before we can understand iron's role in intelligence.

Environmental Changes

We now consider those interventions designed to increase intelligence by enriching young children's environments. In this section, we include three meta-analyses, one on the effects of early educational interventions and one on preschool. We also review environmental interventions for which data are insufficient for meta-analysis.

After searching the literature (see Appendix), we finally found 16 RCTs where the authors provided intensive educational interventions to young children, yielding 43 effect sizes over 19,238 participants (Table 2). These interventions involved more than preschooling alone; rather, the interventions entailed extensive alterations to the child's environment.

We found that enrolling an economically disadvantaged child into an early education intervention raised his or her IQ by more than 4 points ($g = 0.271$, 95% $CI_{WV} = .114$ to $.429$) and that including a center-based education component raised his or her IQ by more than 7 IQ points ($g = 0.454$, $b = .183$, $p < .001$).

Because none of the studies involved children above lower-middle socioeconomic status (SES), we did not examine SES as a moderator. As a result, we must restrict the conclusions we draw from this analysis to children from low SES backgrounds and caution against broader generalization. In addition, contrary to theoretical assumptions (e.g., Ramey & Ramey, 1998), we found that interventions that started earlier in children's lives were no more effective than those that started later (see Appendix).

How Do Early Educational Interventions Raise Intelligence?

The goal of early educational interventions is to raise young children's intelligence while also fostering other desirable outcomes, such as improved social skills and self-regulation. The results of our meta-analysis support the idea that early educational interventions raise a young child's IQ by the time he or she completes the intervention. Because these interventions are multifaceted, we cannot identify any particular feature of it as a causal mechanism. However, our findings are consistent with the idea that environmental complexity promoting intelligence and providing a more cognitively stimulating and demanding environment raises the IQ of those who engage with it.

To our surprise, we found no evidence to support the notion that interventions conducted earlier in young childhood more effectively boost IQ than those that begin later. Although this finding contradicts the reasonable and widely held assumption that earlier is always better when educating children, it is consistent with the belief held by a prescient minority of researchers that the earliest few years of life are not the narrow windows of opportunity they were once thought to be (e.g., Bruer, 1999). We also found evidence to suggest that educational interventions involving activities at a specially designed center are more effective than simpler, home-based interventions. Our results underscore the likelihood that environmental complexity is the prime mechanism underlying gains in IQ; however, which specific aspects of that complexity are most effective or beneficial remain unknown.

Cognitive training

Researchers have made several attempts to raise young children's IQs by training early components of working memory (WM), nonverbal reasoning, and effortful control. Despite encouraging findings from studies conducted with adult participants (e.g., Jaeggi, Buschkuehl, Jonides, & Perrig, 2008), the work on WM training with samples of young children thus

Table 2. Studies Used in the Meta-Analysis on the Effects of Early Educational Interventions on IQ.

Study	*g*	Age (years)	Duration (years)	Notes
No center-based component				
Gutelius et al., 1972	0.82	−0.5	3.5	
Goodson, Layzer, St. Pierre, Bernstein, & Lopez, 2000	0.04	0	5	
Wasik, Ramey, Bryant, & Sparling, 1990	−0.7	0.08	3	No center
Love et al., 2005	0.09	0.42	1.83	Home-based
Bridgeman, Blumenthal, & Andrews, 1981	0.25	1	2	Houston Wave 1
	0.42	1	2	Houston Wave 2
	0.26	1	2	Houston Wave 4
	0.06	1	2	Houston Wave 5
	0.46	1	2	Houston Wave 7
	0.48	1	2	Detroit Wave 2
Jester & Guinagh, 1983	0.06	1	1	Experimental program in the 1st year, control condition in the 2nd and 3rd years
	−0.1	1	2	Experimental program in the 1st and 3rd years, control condition in the 2nd year
	0.40	1	2	Experimental program in the 1st and 2nd years, control condition in the 3rd year
	0.59	1	3	Experimental program in all 3 years
	−0.1	2	1	Control condition in the 1st and 3rd years, experimental program in the 2nd year
Scarr & McCartney, 1988	0.21	2	1.75	
Jester & Guinagh, 1983	0.7	2	2	Control condition in the 1st year, experimental program in the 2nd and 3rd years
Levenstein et al., 1983	0.11	2.17	2	1973 cohort
	−0.47	2.17	2	1975 cohort
	0.39	2.17	2	1976 cohort
	0.02	2.63	.62	1970 cohort
Jester & Guinagh, 1983	0.33	3	1	Control condition in the 1st and 2nd years, experimental program in the 3rd year
Average effect size (mixed)	0.27			
Center-based component				
Ramey et al., 1992	0.46	0	3	
Ramey, Yeates, & Short, 1984	0.79	0	4	
Wasik et al., 1990	1.1	0.08	3	
Bridgeman et al., 1981	0.29	0.17	2.83	Houston Wave 6
	1.13	0.17	2.83	New Orleans Wave 2
	0.27	0.17	2.83	New Orleans Wave 4
	−0.38	0.17	2.83	New Orleans Wave 5
	1.38	0.17	2.83	New Orleans Wave 6
	0.88	0.17	2.83	Detroit Wave 1
	0.79	0.33	2.67	Birmingham Wave 1
	0.32	0.33	2.67	Birmingham Wave 2
	0.37	0.33	2.67	Birmingham Wave 3
Love et al., 2005	0.09	0.42	1.67	Center only
	0.23	0.42	1.92	Home and center
Kagitcibasi, Sunar, & Bekman, 2001	−0.17	2	2	Home and center
	0.3	2	2	Center only
Deutsch, 1971	0.63	3	.75	Wave 1
	0.69	3	.75	Wave 2
	0.73	3	.75	Wave 3
	0.37	3	.75	Wave 4
Karnes, Hodgins, Stoneburner, Studley, & Teska, 1968	1.43	3	.58	
Average effect size (mixed)	0.45			

Note. Age is the age at which the child began the intervention. Negative age indicates the intervention began before the child was born.

far has yielded disappointing results. In one intervention (Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005), researchers trained 24 four-year-olds on WM tasks for 5 days. These WM tasks consisted of a series of computer games designed to exercise various aspects of attention and inhibition. After the training, these children scored only 3.4 points higher than a control group of 25 four-year-olds who had not received this training, a statistically insignificant difference ($g = 0.227$, $p < .432$). In another study, children were assigned to one of three experimental groups, one of which was a WM training group (Nutley et al., 2011). The 24 children in this study participated in 5 weeks of WM training but did not demonstrate any IQ gains.

Although these efforts failed to produce IQ gains, Nutley et al. (2011) did find that training young children to perform nonverbal reasoning tasks (with items adapted from the Leiter Battery, a test similar to the Ravens' Colored Progressive Matrices, or RCPM) led to their scoring significantly higher on the RCPM ($g = 0.614$). Two other studies (Riding & Powell, 1985, 1986) serve as corroborating evidence for the beneficial effects of training young children to complete reasoning tasks on IQ. However, the high degree of similarity between intervention stimuli and posttest items invites skepticism about whether this improved IQ reflects a genuine increase in the children's intelligence or simple practice effects.

Effortful control is a critical executive function required for attending to pertinent information while also manipulating it to perform mental calculations (Blair & Ursache, 2011). Nineteen 5-year-old preschoolers who were trained on effortful control tasks for over a month did not demonstrate IQ gains immediately after completing the training program; however, when they were tested again 2 months later, their improved IQ revealed a positive effect ($g = 0.178$; Rueda, Checa, & Cómbita, 2012). This delayed impact of the intervention is unexpected and requires replication.

Listening to music

One RCT examined the effects of listening to music on young children's IQ. Forty-one children were randomly assigned to either a phonological skills training program or a music listening program to evaluate potential effects on the phonological ability of the preschoolers. Neither intervention produced IQ gains (Dege & Schwarzer, 2011).

Training mothers to provide cognitively complex environments

Maternal behavior and the environmental complexity of the home vary significantly as a function of SES. Higher SES mothers tend to provide their children with more complex and enriching environments than do lower SES mothers (Neisser et al., 1996). Can providing lower SES mothers with the means and training to provide richer environments for their young children increase these children's IQs? The authors of one study (Karnes, Studley, Wright, & Hodgins, 1968) randomly assigned 26 mothers to either a control group or an 11-week intervention. During this intervention, the experimenters trained mothers to make educational materials for their children, provided them with age-appropriate books and puzzles for their children, and taught them how to help their children learn to speak and identify objects in the home. By the end of the 11 weeks, the children of mothers in the training group had gained 7 IQ points ($g = 0.342$), whereas the IQs of the control group mothers' children remained unchanged.

Narrative talk and elaborate reminiscing

Young children's ability to narrate their own experiences shares strong associations with early school readiness (Cristofaro & Tamis-Lemonda, 2012). This relationship may not be causal; both capacities may be outgrowths of intelligence, linguistic skills, or other important cognitive abilities. Two studies shed light on these connections. In one, 20 three-year-olds were randomly assigned to either a control group or a yearlong intervention in which their mothers were trained to be as elaborative as possible with their children when asking them to narrate past events. Specifically, these mothers were encouraged to talk to their children frequently and at length, to ask many open-ended questions that required more than simple one-word answers, to listen and encourage their children to speak in multiple sentences, and to discuss topics of interest to the children (Peterson, Jesso, & McCabe, 1999). This intervention raised the children's IQ more than 6 points ($g = 0.438$, $p < .01$).

In a second study, researchers replicated the methods of training mothers to elaborate and had mothers require their children to be elaborative when recalling past events (Reese, Leyva, Sparks, & Grolnick, 2010). Thirty-three mothers of Head Start children were randomly assigned to one of three groups: one in which mothers were trained to be elaborative while reminiscing with their children, one in which mothers were trained to be elaborative with their children while co-reading a book during story time, or a control group in which mothers received no training. Children whose mothers had participated in the training groups did not have higher IQs than children whose mothers had been assigned to the control group (E. Reese & D. Leyva, personal communication, November 2, 2011).

Why did one study's elaborative talk training program produce significant IQ gains in young children while another study's did not? These inconsistencies probably stem from the different intensities of the two interventions. In the effective intervention (Peterson et al., 1999), mothers received extensive training, and experimenters followed up with them throughout the yearlong intervention in order to aid and assist them and to ensure fidelity in their implementation of the protocol. In the ineffective intervention (Reese et al., 2010), experimenters provided mothers with a single 45-min training session, then tested their children's IQ 8 months later. Thus, it

is unlikely that the mothers in this unsuccessful intervention received sufficient training, support, and monitoring to ensure faithful adherence to the intervention.

How might elaborative narrative talk increase a young child's intelligence? Parent–child conversations and word use are associated with later intelligence and achievement (Hart & Risley, 1995). Future experiments examining such interventions could help clarify whether such findings reflect the effects of narrative talk in such interactions. However, with only two studies on which to base our assumptions, we can only speculate; but clearly such interventions are promising and merit attempts to replicate.

Interactive reading: A meta-analysis

Children who grow up poor have lower IQs than children who grow up wealthy (Herrnstein & Murray, 1994). One of the mediating mechanisms that explain this gap is differences in cognitive stimulation—specifically, differences in the number of reading interactions a child had with his or her parents and the number of books the child grows up with (Guo & Harris, 2000). Many interventions for altering the reading environment of children focus on remediating effects—for example, treating children with preestablished speech and language difficulties (e.g., Crain-Thoreson & Dale, 1999; Danger & Landreth, 2005). Such studies are not included in this database. A subset of these studies focuses on altering the reading environments of children in the normal range of ability.

The original meta-analysis included two experiments that simply provided books to parents without instructions for developing a more interactive reading style (Chow & McBride-Chang, 2003; Lonigan, Anthony, Bloomfield, Dyer, & Samwel, 1999). Neither of these interventions raised the children's IQ, so we can conclude that the mere presence of books in the home does not raise IQ. In the remaining studies we consider, experimenters provided parents both with books and with a training program for effective reading with their children—teaching them how to ask open-ended questions, encourage their children to read, shadow their children's interests, and so on. This meta-analysis includes eight studies, providing 10 effect sizes across 499 participants (see Appendix).

Reading to a child in an interactive style raises his or her IQ by over 6 points (g = 0.404, 95% CI = .153 to .654). With one exception, interventions that begin after the child is 42 months old do not raise the IQ; however, in the random effects model, age is not a significant moderator. In each of these interventions, children and their parents engage with storybook reading in an interactive way. The child is an active participant in the reading, with the adult encouraging the child to be as elaborate as possible. With one exception, these interventions do not appear to raise the IQ if the child is more than 4 years old (see Table 3). Why might this be the case? It is possible that interactive reading does not raise a young child's intelligence but instead merely accelerates language development, which boosts IQ. If this is the case, once the child's level of language development is more advanced, added demands may no longer act as accelerants.

Preschool and intelligence: A meta-analysis

We located 16 RCT studies, yielding 39 effect sizes based on 7,370 participants in which young children were enrolled in preschool. We found that sending a child to preschool raises the IQ by more than 4 points ($g = 0.307$, 95% $CI_{WV} = .135$ to .479). Preschools that include a specific language development component boost IQ by more than 7 points ($g = 0.512$, $b = .205$, $p < .001$).

As is the case in our earlier analyses, insufficient socioeconomic variation prevented us from testing the reasonable prediction that SES would moderate the effects of preschool. We therefore must caution readers again that these findings cannot be generalized beyond children from low-income homes. In addition, we found that preschool interventions that last longer are no more effective at raising the IQ than preschools that are shorter (see Appendix). We explain this puzzling finding next.

Our meta-analysis indicates that sending a disadvantaged child to preschool raises his or her IQ by as much as 7 points

Table 3. Studies Used in the Meta-Analysis on the Effects of Interactive Reading on IQ.

Study	*g*	Age (years)	Notes
Whitehurst et al., 1988	0.58	2.33	
Huebner, 2000	0.27	2.39	
Arnold, Lonigan, Whitehurst, & Epstein, 1994	0.36	2.42	Trained directly
	0.57	2.42	Trained by video
Valdez-Menchaca & Whitehurst, 1992	1.21	2.59	
Whitehurst, Arnold, Epstein, & Angell, 1994	0.15	3.46	School reading
	0.24	3.46	School and home reading
Lonigan, Anthony, Bloomfield, Dyer, & Samwel, 1999	0.03	3.76	
Lamb, 1986	−0.01	4	
Van Kleeck, Vander-Woude, & Hammett, 2006	1.41	4.17	
Chow & McBride-Chang, 2003	−0.18	5.32	
Effect size (random)	0.4		

Table 4. Studies Used in the Meta-Analysis on the Effects of Preschool Attendance on IQ.

Study	*G*	Duration (years)	Notes
No language development component			
Rainey, 1968	0.33	0.5	
Di Lorenzo, Salter, & Brady, 1969	0.52	0.75	Cortland, no language
	–.05	0.75	Greenburgh
	–0.54	0.75	Hempstead
	–0.02	0.75	Long Beach
	0.16	0.75	Spring Valley
	.47	0.75	Yonkers
Karnes, Zehrbach, & Teske, 1974	0.48	0.75	
Erickson, McMillan, Bonnell, Hofmann, & Callahan, 1969	0.67	0.75	Enrichment program
Average effect size (mixed)	0.31		
Language development component			
Abbott-Shim, Lambert, & McCarty, 2003	0.08	0.17	Summer program
Skeels, Ruth, Wellman, & Williams, 1938	0.32	0.32	1–199 days
Blank & Solomon, 1968	1.50	0.33	5 days per week
	0.28	0.33	3 days per week
Peta, 1973	0.52	0.34	
Edwards & Stern, 1970	0.20	0.46	Cohort 1
	0.07	0.46	Cohort 2
Ametjian, 1965	0.87	0.5	
Klaus & Gray, 1968	0.73	0.5	2-year program
	0.59	0.5	3-year program
Dawe, 1942	0.96	0.6	
Di Lorenzo & Salter, 1969	0.52	0.75	Cortland, language
	0.36	0.75	Mt. Vernon
	0.41	0.75	Schenectady
Abbott-Shim et al., 2003	0.24	0.75	School year
	0.32	0.75	School year with posttest delay
Erickson et al., 1969	0.77	0.75	Bereiter-Englemann program
Skeels et al., 1938	0.81	0.77	200–399 days
Weikart, 1966	0.56	1	Wave 0
	0.83	1	Wave 3
Puma, Bell, Cook, & Heid, 2010	0.11	1	4-year-olds
Deutsch, 1971	0.81	1.5	Wave 1
	0.01	1.5	Wave 2
	0.77	1.5	Wave 3
	0.35	1.5	Wave 4
Skeels et al., 1938	1.10	1.65	400+ days
Weikart, 1966	0.55	2	Wave 1
	0.96	2	Wave 2
Herzog, Newcomb, & Cisin, 1974	0.66	2	
Puma et al., 2010	0.07	2	3-year-olds
Average effect size (mixed)	.51		

if it includes a specific language-development component. Schooling is known to both increase and maintain intelligence (Ceci, 1991), though the specific mechanisms remain unknown. Many intelligence tests tap knowledge of information and vocabulary. Although there are specific mechanisms responsible for the recall, comprehension, and retrieval of information and vocabulary words, the most important factor in a young child's success or failure with these test items may be exposure. Young children cannot define a word that they have not encountered, nor can they identify a picture of an item that they have never seen. Therefore, preschool may raise intelligence test performances merely by exposing young children to the information and vocabulary words included on these tests. If exposure is the mechanism by which preschool

boosts IQ test performance, then these improved scores do not indicate genuine gains in intelligence.

A more optimistic explanation of preschool's causal role in boosting young children's intelligence is that the cognitive complexity inherent to the preschool environment and experience leads to increases in underlying intelligence. Attending preschool provides lower SES children with the opportunity to engage with novel stimuli, to practice complex problem solving, to navigate social interactions, and to confront other cognitive challenges they do not face in their home environments. This explanation is consistent with the positive results of extensive maternal training and early educational interventions and provides additional support for the argument that increasing the cognitive demands of a young child's environment causes him or her to adapt and become more intelligent. Yet why do we fail to find a positive relationship between duration of preschooling and IQ gains? Recall that preschool programs of longer durations were no more effective than shorter ones. We suspect that the reason for this seeming contradiction is that as the program wears on, preschool may become less challenging and complex.

For example, consider a physically weak young boy who cannot do more than one or two push-ups. Our goal is to increase the number of push-ups he can do, so we enroll him in a push-up intervention. This intervention involves daily push-up exercises of up to 15 push-ups per day. After participating in this intervention for 6 weeks, our formerly weak young boy can reliably perform 15 push-ups on demand. If the intervention continues for another 12 weeks and the boy still has to do only 15 push-ups a day, we would not see another increase in the young boy's strength. Why? Because the daily demands of the intervention did not change. Preschoolers may have similar experiences. If a young child gains intelligence after attending preschool for 1 year, for example, and the preschool curriculum's demands do not improve, the child may not be adequately challenged to further increase his or her intelligence if the preschool's demands on intelligence remain constant. As a result, his or her intellectual progress may stagnate.

Conclusion

The present work has been a systematization and synthesis of as much of the available knowledge on raising young children's intelligence as we could locate. Our analysis allows us to draw the following conclusions:

- Supplementing the diets of pregnant women and neonates with LC-PUFA raises the children's IQ in young childhood. Providing preschool-aged children with iron supplements may boost their IQ, but giving these supplements to infants does not.
- Enrolling a lower SES infant in an intense early educational intervention will raise his or her IQ in young childhood. Enrolling him or her in such a program at a younger age has no additional benefits for his or her IQ. The more complex the intervention is, the greater these gains will be.
- Reading interactively with young children raises their IQ. The earlier the interactive reading takes place, the larger the benefits.
- Attending preschool increases a young child's IQ. If the preschool program includes a specific language-development component, these gains are even larger.

We currently lack sufficient data to determine whether any of the other interventions we examined are effective. Using the most scientifically rigorous and conservative standards available, we will continue to compile and synthesize all of the available scientific evidence on raising intelligence. Our current findings strengthen earlier conclusions (e.g., that complex environments build intelligence), cast doubt on others (e.g., that earlier interventions are always most effective), and give rise to tantalizing new questions for future research (e.g., can essential fatty acids increase intelligence?).

Database Activity

We made every effort to include every intervention in our study. However, we 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 DORI will remain an active database, updated 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 DORI's inclusion criteria to send their data. Please contact the corresponding author with any research you may have to contribute.

Appendix

What follows is the statistical methods and procedures we used to come to our conclusions, including all models run and the results from those models. Across all four meta-analyses, we included multiple experiments from the same study. Though a more conservative approach is to remove studies with multiple interventions and only keep one, we find this method troublesome as the choice of which experiment to keep can be too subjective and leave open the possibility for bias. To examine whether experiments that came from the same study could bias our results, we tested whether effect sizes were dependent on groups of studies. In each of the meta-analyses reported here, no such grouping was found at any significant level (Card, 2011). All confidence intervals are given using the Weighted-Variance method (Sidik & Jonkman, 2003).

Statistical issues on the meta-analysis of LC-PUFA supplementation and intelligence

There was no evidence of publication bias in our data ($p_{\text{Begg}} < .158$, where Begg is the metric of publication bias). We

originally ran the data in STATA (StataCorp, 2011) using a weighted regression and clustering the covariance matrix to be able to take into account longitudinal data. Two effect sizes were outliers in the data set, both from studies with the same authors (Carlson & Werkman, 1996; Werkman & Carlson, 1996; both greater than three standard deviations above). In both studies, the intervention included giving infants formula supplemented with 0.2% docosahexaenoic acid (DHA, the most common form of supplemented LC-PUFA), one for 2 months (Carlson & Werkman, 1996) and the other for 9 months (Werkman & Carlson, 1996). Two other studies (Auestad et al., 2003; Birch et al., 2007) included a third intervention group (both included DHA plus arachidonic acid). We removed these studies from the analysis and present them separately.

All of the studies involved supplementing either pregnant or breast-feeding mothers or neonates, except one study (Ryan & Nelson, 2008) in which 4-year-old children received 400 mg of LC-PUFA supplements daily for 4 months. To determine whether LC-PUFA supplementation in the first years of life raises intelligence while trying to reduce the heterogeneity in the sample, we ran the analysis both with and without this study; removing this study from the analysis did not substantively change the results.

We found nine other studies on LC-PUFA supplementation and young children's intelligence, yielding 13 effect sizes. There was significant heterogeneity in the sample, $Q(12) = 184.748$, $p < .001$, so it was necessary to look for moderators, remove outliers, or run a random- or mixed-effects analysis on the data. We thought it best to remove the two outliers from the data first; doing so reestablished normality to the data set's distribution ($p_{K\text{-}S} < .2$; K-S = Kolmogorov–Smirnov normality test).

To determine the role of LC-PUFA supplementation on subsequent intelligence, we restricted longitudinal investigations to the last time point at which participants were still taking supplements and had completed an intelligence test. In doing so, we removed five of our effect sizes, reducing the final sample to 10 effect sizes across 844 participants. Although in some ($n = 4$) of the studies researchers tested children's intelligence in toddlerhood, in others ($n = 6$) researchers tested children's intelligence in infancy by using the Fagan Test of Infant Intelligence (FTII; Fagan & Shepard, 1987). Test type (FTII vs. tests for older children, such as the Stanford-Binet or Wechsler Intelligence Scale for Children) did moderate the results. As there was still significant heterogeneity, we used a mixed-effects analysis[4] (Overton, 1998) with whether the FTII was used as our moderator. We reached convergence (defined as $\Delta\tau^2 < 10^{-10}$; Erez, Bloom, & Wells, 1996) in seven iterations.

Statistical issues on the meta-analysis of intense early educational interventions and intelligence

There were no outliers in the early educational intervention data. Effect sizes were normally distributed ($p_{K\text{-}S} < .2$), but there was a grouping problem with effect sizes coming from the same study being more similar than others. We found no evidence for publication bias in the data ($p_{Begg} < .832$).

In order to determine the maximum effect of each intervention while minimizing the amount of delay between the intervention and the posttest, we restricted the data to time points immediately following the interventions, reducing the sample to 43 effect sizes (see Table 2). By doing so, we eliminated the aforementioned grouping issue, $F(1, 12) = .397$. However, there was significant heterogeneity in the sample, $Q(42) = 558.713$, $p < .001$, so further analyses were in order.

To provide the most complete list of theoretically appropriate moderators, we coded the following variables in the studies: socioeconomic status (SES) of the family, age of the child when the intervention started, duration (years) of the intervention, whether the child's diet was supplemented, and whether the intervention occurred in the children's home, in a special development center, or both. We removed studies including a specific nutritional component from the analysis because of insufficient variability.

Initial analyses indicated significant heterogeneity in the sample, $Q(42) = 558.713$, $p < .001$, so we identified characteristics of the interventions that might moderate the effects. With 43 effect sizes we had enough statistical power to test only the two most important moderators: child's age at the intervention's inception and the inclusion of a center-based education component (e.g., nursery school or program at another specially designed development center that delivered developmentally appropriate and cognitively demanding activities). Both of these moderators were statistically significant.

Because the effect sizes were still grouped within studies, we tested for heteroskedasticity in the errors (Dickens, 1990), as possible effect size errors are correlated with group level errors; heteroskedasticity existed, Breusch–Pagan $\chi^2(1) = 1203.87$, $p < .001$. Combining the statistical issues of heteroskedastic errors from data grouping and heterogeneity across the samples, we conducted the analysis again, this time using a mixed-effects model (Overton, 1998) in R. We first tested the moderators under a nonweighted situation and found that interventions that included a center-based education component were more effective at boosting young children's IQ, but interventions that began earlier in a child's life were no more effective than those that started later. Therefore, in the subsequent mixed-effects analysis, we included the presence of a center-based education component as the sole moderator. We reached convergence (defined as $\Delta\tau^2 < 10^{-10}$; Erez et al., 1996) in 20 iterations.

Statistical issues on the meta-analysis of interactive reading and intelligence

Data were normally distributed ($p_{K\text{-}S} < .114$). There was no indication of publication bias ($p_{Begg} < .335$). Under a fixed-effects analysis, reading to a child raises his or her IQ by over

2 points ($g = 0.159$, 95% CI = .129 to .189); however there was significant heterogeneity in this model, $Q(15) = 100.586$, $p < .001$, so we looked further into the data. We first removed longitudinal data points so that results would reflect only the immediate result of the intervention. After doing so, we found that reading to a young child raises his or her IQ by over 3 points ($g = 0.204$, 95% CI = .169 to .239), but we also found heterogeneity in the data, $Q(11) = 105.846$, $p < .001$. Because we had enough power to run only one moderator in our attempt to remove this heterogeneity, we examined our three most informed moderators: age when the intervention began (centered on age 2), SES of the parents, and duration of the intervention. The younger the child was when the intervention began, the better the results ($b = -.168$, $p < .001$); however, the interventions did not differ in effectiveness as a function of SES ($p < .963$). Because multiple effect sizes came from the same studies, errors may not be independent; indeed, we found that to be the case under both models (all Breusch–Pagan ps $< .001$). Two studies included two experimental conditions, one a dialogic reading condition (see Whitehurst et al., 1988) and another a regular reading condition in which parents received books without specific instructions or training regarding how to read with their children. Neither of these interventions raised the IQ of children, and they were removed from the analysis. Continuing with this analysis changes it from a meta-analysis on reading to your child to a meta-analysis on interactive reading. Clustering the standard errors for nonindependence in the fixed effects model did not substantively change the results; further models were therefore run without clustering. Because of significant heterogeneity, $Q(9) = 69.345$, $p < .001$, we ran the final model as a random-effects model with no moderators. We then explored whether the age at which the intervention began moderated the results.

Statistical issues in the meta-analysis of preschool and intelligence

There were no outliers in the preschool data, effect sizes were normally distributed ($p_{K\text{-}S} < .2$), and there was no evidence of publication bias ($p_{Begg} < .25$).

The first problem with the preschool analysis was grouping. We coded effect sizes by the study from which we obtained them. Studies with multiple sites or multiple waves of an intervention presented in a single article were coded as such, and we investigated to ensure that effect sizes from the same studies did not differ significantly from others, which would represent a problem of grouping. Effect sizes were dependent on groups of studies, $F(1, 15) = 6.942$, $p < .001$.

Removing the longitudinal data points and keeping only those that represented the longest duration of preschool while minimizing the delay between the end of preschool and the posttest removed the grouping problem, $F(1, 15) = 1.87$, $p < .086$, and reduced the number of effect sizes to 39 across 7,370 participants. Because doing so did not eliminate the amount of heterogeneity in the sample, $Q(38) = 510.64$, $p < .001$, we then examined characteristics that moderate a preschool's effectiveness at raising a young child's IQ.

Although a host of predictors could help identify the most effective components of a preschool program, we coded the following possible moderators as part of our meta-analysis: children's families' SES, age at which children started preschool, duration of the preschool (in years), number of days per week preschool was attended, number of hours per day preschool was attended, presence of a nutritional component, maternal involvement (either through home visits or presence at the preschool), presence of a specific language-development component, presence of a specific cognitive-skills component, and measure of intelligence used as posttest.

Our initial analyses indicated significant heterogeneity existed in the sample so we looked for characteristics of the interventions that could moderate the effects of preschool on intelligence.

Limited statistical power permitted the testing of only two critical moderators: duration of preschool and inclusion of a specific language-development component. Although both moderators were significant in the analysis, there was still significant heterogeneity in the sample, $Q(36) = 465.795$, $p < .001$; and heteroskedasticity of the errors, Breusch-Pagan $\chi^2(1) = 38.09$, $p < .001$. With correlated error components within groups, significant heterogeneity, and moderators of interest, we conducted a mixed-effects model on the data (Overton, 1998) in R to obtain a more accurate estimate of the effects. To ensure the acceptability of both moderators, we ran the model again, this time without the weighting; in this analysis, preschools that included a specific language-development component were found to be somewhat more effective at raising IQ ($p < .099$), but program duration did not moderate the effectiveness of a preschool program ($p < .651$). As a result, in the final analysis, we included only the presence of a specific language-development component. We reached convergence (defined as $\Delta\tau^2 < 10^{-10}$; Erez et al., 1996) in 19 iterations.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Notes

1. LC-PUFA are frequently referred to as omega-3 fatty acids.
2. All point values are the effect sizes multiplied by a standard deviation of 15. These represent conservative values as many of the studies have smaller distributions, so the actual point values would be higher. These values are given for ease of interpretation only.
3. ARA—or omega-6—is a nonessential fatty acid.
4. A mixed-effects analysis is a random-effects meta-analytic model with a fixed moderator. See Overton (1998) for a full explanation.

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