

Household interventions for preventing domestic lead exposure in children (Review)

Yeoh B, Woolfenden S, Lanphear B, Ridley GF, Livingstone N



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[Intervention Review]

Household interventions for preventing domestic lead exposure in children

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ABSTRACT

Background

Lead poisoning is associated with physical, cognitive and neurobehavioural impairment in children and trials have tested many household interventions to prevent lead exposure. This is an update of the original review by the same authors first published in 2008.

Objectives

To determine the effectiveness of household interventions in preventing or reducing lead exposure in children as measured by reductions in blood lead levels and/or improvements in cognitive development.

Search methods

We identified trials through electronic searches of CENTRAL (*The Cochrane Library*, 2010, Issue 2), MEDLINE (1948 to April Week 1 2012), EMBASE (1980 to 2012 Week 2), CINAHL (1937 to 20 Jan 2012), PsycINFO (1887 to Dec week 2 2011), ERIC (1966 to 17 Jan 2012), Sociological Abstracts (1952 to 20 January 2012), Science Citation Index (1970 to 20 Jan 2012), ZETOC (20 Jan 2012), LILACS (20 Jan 2012), Dissertation Abstracts (late 1960s to Jan 2012), ClinicalTrials.gov (20 Jan 2012), Current Controlled Trials (Jan 2012), Australian New Zealand Clinical Trials Registry (Jan 2012) and the National Research Register Archive. We also contacted experts to find unpublished studies.

Selection criteria

Randomised and quasi-randomised controlled trials of household educational or environmental interventions to prevent lead exposure in children where at least one standardised outcome measure was reported.

Data collection and analysis

Two authors independently reviewed all eligible studies for inclusion, assessed risk of bias and extracted data. We contacted trialists to obtain missing information.

Main results

We included 14 studies (involving 2656 children). All studies reported blood lead level outcomes and none reported on cognitive or neurobehavioural outcomes. We put studies into subgroups according to their intervention type. We performed meta-analysis of both continuous and dichotomous data for subgroups where appropriate. Educational interventions were not effective in reducing blood lead levels (continuous: mean difference (MD) 0.02, 95% confidence interval (CI) -0.09 to 0.12, $I^2 = 0$ (log transformed); dichotomous $\geq 10\mu\text{g/dL}$ ($\geq 0.48\ \mu\text{mol/L}$): relative risk (RR) 1.02, 95% CI 0.79 to 1.30, $I^2=0$; dichotomous $\geq 15\mu\text{g/dL}$ ($\geq 0.72\ \mu\text{mol/L}$): RR 0.60, 95% CI 0.33 to 1.09, $I^2 = 0$). Meta-analysis for the dust control subgroup also found no evidence of effectiveness (continuous: MD -0.15, 95% CI -0.42 to 0.11, $I^2 = 0.9$ (log transformed); dichotomous $\geq 10\mu\text{g/dL}$ ($\geq 0.48\ \mu\text{mol/L}$): RR 0.93, 95% CI 0.73 to 1.18, $I^2 = 0$; dichotomous $\geq 15\mu\text{g/dL}$ ($\geq 0.72\ \mu\text{mol/L}$): RR 0.86, 95% CI 0.35 to 2.07, $I^2 = 0.56$). When meta-analysis for the dust control subgroup was adjusted for clustering, no statistical significant benefit was incurred. The studies using soil abatement (removal and replacement) and combination intervention groups were not able to be meta-analysed due to substantial differences between studies.

Authors' conclusions

Based on current knowledge, household educational or dust control interventions are ineffective in reducing blood lead levels in children as a population health measure. There is currently insufficient evidence to draw conclusions about the effectiveness of soil abatement or combination interventions.

Further trials are required to establish the most effective intervention for prevention of lead exposure. Key elements of these trials should include strategies to reduce multiple sources of lead exposure simultaneously using empirical dust clearance levels. It is also necessary for trials to be carried out in developing countries and in differing socioeconomic groups in developed countries.

PLAIN LANGUAGE SUMMARY

Household interventions for preventing domestic lead exposure in children

Lead poisoning is associated with adverse effects on the development and behaviour in children. Many educational and environmental household interventions to prevent lead exposure in children have been studied. This review of 14 studies found that educational and dust control interventions are not effective in reducing blood lead levels of young children. There is currently insufficient evidence that soil abatement or combination interventions reduce blood lead levels and further studies need to address this. More research is needed to find out what is effective for preventing children's exposure to lead and studies should be carried out in different socioeconomic groups within developed countries as well as in developing countries as well as developed countries.

SUMMARY OF FINDINGS FOR THE MAIN COMPARISON [\[Explanation\]](#)

Education strategies for preventing domestic lead exposure in children		Relative effect (95% CI)	No of Participants (studies)	Quality of the evidence (GRADE)	Comments
Patient or population: Children Settings: Households Intervention: Education strategies for prevention of domestic lead exposure Comparison: Regular environment					
Outcomes	Illustrative comparative risks* (95% CI)				
	Assumed risk				
	Regular environment Education strategies for prevention of domestic lead exposure				
Blood lead level (continuous) The mean blood lead level (continuous) ranged across control groups from 1.24 to 2.13 µg/dL ^{1,2} Scale from: 0 to 30 Follow-up: 6 to 18 months	The mean blood lead level (continuous) ranged across control groups from 0.02 higher (0.09 lower to 0.12 higher)		814 (5 studies)	⊕⊕⊕⊕ high	
Blood lead level (dichotomous) ≥ 10 µg/dL blood lead level Follow-up: 6 to 18 months	Medium risk population ³ 238 per 1000 ⁴	RR 1.02 (0.79 to 1.3) ⁵	520 (4 studies)	⊕⊕⊕○ moderate ^{4,5}	
Blood lead level (dichotomous) ≥ 15 µg/dL blood lead level Follow-up: 6 to 18 months	Medium risk population ³	RR 0.6 (0.33 to 1.09)	520 (4 studies)	⊕⊕⊕○ moderate ^{4,5}	

	110 per 1000 ⁴	66 per 1000 (36 to 120) ⁴		
Floor dust - hard floor (continuous) Floor dust lead levels Scale from: 0 to 40 Follow-up: 6 to 18 months	The mean floor dust level - hard floor - ranged across control groups from 1.65 to 2.28 µg/ft²	The mean floor dust level - hard floor - in the intervention groups was 0.07 lower (0.37 lower to 0.24 higher)	318 (2 studies)	⊕⊕⊕○ moderate ⁶
Cognitive and neurobehavioural outcomes - not reported	See comment	See comment	-	Not estimable See comment
*The basis for the assumed risk (e.g. the median control group risk across studies) is provided in footnotes. The corresponding risk (and its 95% confidence interval) is based on the assumed risk in the comparison group and the relative effect of the intervention (and its 95% CI). CI: Confidence interval; RR: Risk ratio;				
GRADE Working Group grades of evidence High quality: Further research is very unlikely to change our confidence in the estimate of effect. Moderate quality: Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate. Low quality: Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate. Very low quality: We are very uncertain about the estimate.				

¹ Change in blood lead level

² These are logged values

³ Baseline based on median of control groups

⁴ Total number of events less than 300

⁵ 95% CI around pooled estimate includes no effect and appreciable harm or benefit

⁶ Total population is less than 400

BACKGROUND

Description of the condition

Lead is a metal that has been used since prehistoric times. Over the years, its wide distribution and mobilisation in the environment has resulted in increasing human exposure and uptake (Tong 2000). It has been widely reported that lead poisoning is a serious health hazard with major socioeconomic implications (UNEP-UNICEF 1997). At high levels, lead poisoning in children can cause anaemia, multi-organ damage, seizures, coma and death. At chronic low levels, lead toxicity causes significant cognitive, psychological and neurobehavioural impairment (UNEP-UNICEF 1997; Tong 2000).

In terms of global burden of disease, lead has been shown to account for 0.9% of the total disease burden (Fewtrell 2003). The World Health Organization will soon release their 2005 data on global blood lead levels; however, in the interim, this data has been used to estimate the burden of disease associated with lead exposure in Europe, and amounts to at least 1,053,000 Disability Adjusted Life Years (Braubach 2011). It has been difficult to document lead burden accurately due to the invasive nature of blood lead level monitoring and the diverse manifestations of lead exposure. There are many potential sources of lead in the environment and these include lead industries, mining and smelting; leaded petrol; lead-based paint; water piping, fixtures and solder; as well as consumer products and hobbies that use lead. Lead from these sources is most commonly found in paint, dust, soil or water. Risk factors for lead exposure include socioeconomic disadvantage, living in an area with lead industry, renovation or deterioration of older lead painted houses and living in developing countries where leaded petrol is still used (Tong 2000).

Blood lead levels in the general population of developed countries have fallen significantly over the past 20 years due to phasing out of lead petrol and bans on the use of lead in paints, lead solder used in canned foods and other consumer products (Jacobs 2006). Concern has now grown regarding chronic low level exposure within the environment (Tong 2000). The major source of environmental lead dust exposure in children in developed countries is lead-based paints and other lead hazards in housing. Although lead-based paint is no longer available for domestic use or most industrial use in developed countries, older housing with peeling or flaking paint or current renovations results in increased lead dust levels (EHU 2002).

Occupational and environmental exposures continue to be a serious global problem, especially in developing or rapidly industrialising countries (Tong 2000). Developing country populations, especially children, may have higher levels of lead exposure due to unregulated industrial emission and car emission of leaded petrol; less stringent environmental and occupational health safety regulation, and cottage (domestic) industries such as metal polishing and smelters (UNEP-UNICEF 1997). It is of concern that lead-based paints for household use are still available for purchase in several

developing Asian countries, such as China, India and Malaysia (Clark 2005; Adebawo 2007). In view of rapid industrialisation and persistence of lead in the environment, this is likely to remain a significant public health issue in developing countries for many years (Tong 2000).

Children are at increased risk of lead toxicity. This is due to their increased intake of lead per unit body weight compared with adults and their physiological uptake rate being higher (up to 50% compared with 10% to 15% in adults) (UNEP-UNICEF 1997). Young children often place objects in their mouths resulting in lead-contaminated dust and soil ingestion. Furthermore, a young child's developing body, and in particular the central nervous system, is more vulnerable to the effects of lead.

Urban children in developing countries are considered most at risk and it was estimated in 1994 that "over 80% of those between three and five years of age and 100% under two had average blood lead levels exceeding the threshold of 10 µg/dL (0.48 µmol/L) set by U.S. Centres for Disease Control and Prevention" (UNEP-UNICEF 1997).

There is no evidence of a safe blood lead level below which children are not affected (Wigg 2001) and recent studies show that adverse effects on cognitive function in children are proportionally greater at lower blood lead levels (Canfield 2003; Lanphear 2005a; Kordas 2006). Of further concern, the effects of lead are thought to be largely irreversible so reducing or eliminating lead from the body does not significantly improve the neuropsychological manifestations (Tong 2000). Chelation agents, currently the mainstay of treatment of children with blood lead concentrations > 45 µg/dL, reduce the mortality of severe acute lead encephalopathy but they do not remove the majority (estimated to be 95%) of the body's lead sequestered in bone nor do they reverse neuropsychological effects (Chisolm 2001; Rogan 2001; Dietrich 2004). Due to the higher rate of bone turnover in young children, the average half-life of lead in blood is significantly longer (8 to 11 months with acute exposure and 20 to 38 months with prolonged exposure) than that of adults (15 days) and bone can be a prolonged source of lead in blood (Manton 2000; Chisolm 2001).

It has been estimated that the cost of medical treatment is higher than environmental interventions and is not likely to have significant long-term benefit (Chisolm 2001). In view of this, it is not acceptable to only identify and treat children suffering from toxicity. Prevention of lead-induced effects by controlling lead hazards in the environment should be the primary goal for management of this public health issue (Chisolm 2001).

Primary prevention aims to prevent exposure to lead by eliminating the environmental source both at a community and individual level. Secondary prevention aims to identify children who are at risk from their environment and limit further exposure (Campbell 2000). The best method to identify at-risk individuals is not clearly defined but the AAP recommend screening based on a list of risk criteria. These include children who live in housing built before 1950; live in old housing (pre-1978) undergoing renovation; have

a history of pica; have a history of exposure to lead-containing substances; have a sibling with lead toxicity; have a parent exposed to lead through vocation or hobby, and who were born in countries with high lead prevalence (Campbell 2000). These guidelines provide guidance to public health authorities for developing a screening policy based on local blood lead and housing age data. Hand in hand with effective screening is the need for effective interventions to reduce lead exposure.

Description of the intervention

Environmental and educational interventions have been the main prevention techniques studied. Educational interventions address parental awareness of lead exposure pathways, hygiene and household dust control measures to prevent ingestion of dust and soil (Campbell 2000). Several papers have studied the effectiveness of educational interventions to encourage home cleaning and these studies varied in the extent of cleaning activities and the educational programme. The results have not supported the effectiveness of education alone (Campbell 2000).

Environmental prevention focuses on improvement in risk assessment, development of housing-based standards for lead-based paint hazards, as well as safe and cost-effective lead hazard reduction techniques (Campbell 2000). Several studies have been published regarding various lead reduction techniques and their relative effectiveness and safety. These have studied both abatement (permanent elimination of lead sources through removal of paint and dust, replacement of lead containing structures and covering of lead-contaminated soil) and interim controls pending abatement (specialised cleaning, repairs, maintenance, painting and temporary containment). A variety of environmental lead hazard control interventions to decrease children's blood lead level and home dust lead levels have been tested in randomised controlled trials (RCTs), with most follow-up extending from six months to two years after intervention. Comparison of environmental interventions has been difficult due to variations in intervention type, blood collecting technique, adjustments for age and season, dust lead loading quantification and statistical analyses (Campbell 2000).

Why it is important to do this review

Lead poisoning has long been proven to be associated with physical, cognitive and neurobehavioural impairment in children. Despite efforts to reduce environmental, occupational and industrial lead exposure worldwide, children in many areas with older housing, as well as children living in developing countries with less stringent industrial regulations, continue to show evidence of lead exposure. Many household interventions have been studied in trials and it is important that the effectiveness of these interventions is examined.

This is an update of our original review (Yeoh 2008), which found no evidence of effectiveness for household interventions for education or dust control measures in reducing blood lead levels in children as a population health measure and concluded there was insufficient evidence for soil abatement or combination interventions. Further trials were required to establish the most effective intervention for prevention of lead exposure and hence it is important to update this review looking for any advances in the area.

OBJECTIVES

The aim of this systematic review is to determine if educational and/or environmental household interventions are effective in preventing and/or reducing domestic lead exposure in children. This can be assessed by measuring children's blood lead levels and/or improvements in cognitive and neurobehavioural development in both the short- and long-term.

METHODS

Criteria for considering studies for this review

Types of studies

RCTs (random allocation) or quasi-randomised studies (using a method of allocation that is not truly random; for example, by date of birth, medical record number, or order in which participants are included in the study, such as alternation) where participants were allocated to an intervention or control group.

There are several reasons to rely on RCTs or quasi-randomised trials to test the effect of interventions on children's blood lead levels. First, they account for secular trends in blood lead levels. Children's blood lead levels have declined over the past three decades and studies that attempt to test the effect of interventions in the absence of a control group may overestimate their effect because of the downward trend in blood lead concentrations. Second, children's blood lead levels, which peak at about two years of age, typically decline as they mature, primarily because they no longer exhibit frequent mouthing behaviours. As such, any observational study that enrolls children at 18 months to two years may erroneously conclude that the intervention led to a reduction in blood lead levels even though children's blood lead levels would have declined anyway. Finally, children's blood lead levels peak during summer months; if the intervention does not account for seasonal variation it may under- or over-estimate the effect of an intervention.

Types of participants

Children and adolescents (from birth to 18 years of age) and their parents or carers.

Types of interventions

Interventions that aim to reduce domestic lead exposure compared to no intervention or standard measures/recommendations. In this review, interventions were classified as follows.

- Educational interventions - these address parental awareness of lead exposure pathways, hygiene and household dust control measures to prevent ingestion of dust and soil.
- Environmental (household) interventions - these include specialised cleaning, repairs, maintenance, soil abatement (removal and replacement), painting and temporary containment of lead hazards.
- Combinations of the above interventions.

Interventions involving nutritional supplementation were not included.

Mode of delivery may be by health professionals, paraprofessionals or via written media.

Types of outcome measures

The following outcomes were considered in this review.

1. Cognitive and neurobehavioural outcomes in children. Standardised measures of outcome, such as assessment of a child's intelligent quotient (IQ) (using the Stanford Binet Intelligence Scale (Smith 1989), Wechsler Intelligence Scale for Children (Wechsler 1991), Wechsler Preschool and Primary Scale of Intelligence (Wechsler 1989)); development (for example, Griffiths Mental Development Scales (Griffiths 1954; Griffiths 1970)), or behaviour (for example, Child Behaviour Checklist (Achenbach 1991))

2. Blood lead levels in children (venous blood sample or capillary blood sample) (AAP 1998)

3. Household dust measures

Instruments were confined to those with at least one standardised outcome measure (such as blood lead level) used for intervention and control group. Outcomes for any follow-up duration period (short-term and long-term) were considered.

Data on adverse events and costs, where available, were also reported in the [Results](#) section.

Search methods for identification of studies

The aim of the search strategy was for high precision and recall. The search strategies used previously for the review were revised for this update to improve their precision ([Appendix 1](#)). The single search term "lead" was replaced by phrases in which "lead"

occurs in proximity to other relevant terms. The methodology filter used in the MEDLINE strategy was replaced by the 2008 version of the Cochrane highly sensitive search strategy for identifying randomised trials (Lefebvre 2008). Search terms for individual databases were modified as necessary to meet the requirements of any changes to indexing terms or database platforms since the previous searches were executed. There were no language restrictions.

Electronic searches

Relevant trials were identified through searching the following databases, initially in 2006 and then in 2012 for this updated review.

MEDLINE (1948 to Jan Week 1 2012), searched 15 January 2012 ([Appendix 2](#)).

Cochrane Central Register of Controlled Trials (CENTRAL) 2012 (Issue 1), searched 20 January 2012 ([Appendix 3](#)).

EMBASE (1980 to 2012 Week 2), searched 17 January 2012 ([Appendix 4](#)).

PsycINFO (1806 to current), searched 17 January 2012 ([Appendix 5](#)).

CINAHL (1937 to current), searched 20 January 2012 ([Appendix 6](#)).

Sociological Abstracts (1952 to current), searched 20 January 2012 ([Appendix 7](#)).

ERIC (1966 to current) searched 17 January 2012 ([Appendix 8](#))

Science Citation Index (1970 to current), searched 20 January 2012 ([Appendix 9](#)).

ZETOC searched 20 January 2012 ([Appendix 10](#)).

LILACS searched 20 January 2012 ([Appendix 11](#)).

Dissertation Abstracts searched via Dissertation Express January 2012 ([Appendix 12](#)).

ClinicalTrials.gov accessed on 20 January 2012 ([Appendix 13](#)).

Current Controlled Trials accessed on 20 January 2012 ([Appendix 13](#)).

Australian New Zealand Clinical Trials Registry accessed on January 2012 ([Appendix 13](#)).

National Research Register Archive searched January 2012 ([Appendix 13](#)).

We also conducted internet searches, searched conference proceedings and contacted experts to determine if any unpublished or ongoing trials existed. No further studies were identified.

Data collection and analysis

Selection of studies

Two authors (BY, SW) screened titles and abstracts from the search. We resolved disagreement by consensus and in consultation with a third author (GR) and discarded articles that did not fulfil inclusion criteria. We retrieved potentially relevant articles for full-text assessment, where appropriate, and for data extraction.

Data extraction and management

We organised data using Review Manager 5 (Review Manager 2011). We developed data extraction forms a priori and included information regarding methods, participant details, intervention type, administration and outcomes. Two independent authors (BY, SW) completed data extraction forms for each included study and no disagreements arose.

Assessment of risk of bias in included studies

Two of four independent authors (BY, SW, GR, NL) evaluated included studies for risk of bias and relevance. We judged each using the categories of 'low risk of bias', 'high risk of bias', or 'unclear risk of bias', indicating either lack of information or uncertainty over the potential for bias. We assessed six specific domains as listed below.

1. **Sequence generation** describes the method used to generate the allocation sequence to allow an assessment of whether it should produce comparable groups.
2. **Allocation concealment** describes the method used to conceal the allocation sequence in sufficient detail to determine whether intervention allocations could have been foreseen in advance of, or during, enrolment.
3. **Blinding** describes all measures used to blind study participants and personnel from knowledge of which intervention a participant received.
4. **Incomplete outcome data** describes the completeness of outcome data including attrition and exclusions from the analysis.
5. **Selective outcome reporting** considers whether the trialists reported on all relevant and prespecified outcomes.
6. **Other sources of bias** considers any important concerns about bias not addressed in the other domains in the tool. Where there was insufficient information in the published study regarding methodology or results in an extractable form, we contacted authors via email (and fax or phone call if required) on several occasions. We did not score risk of bias on an additive basis.

Measures of treatment effect

Continuous data

Where standardised assessment tools generated a score as the outcome measure, we made comparisons between the means of these scores. We used post-treatment means and standard deviations in all meta-analyses. As blood lead level data are typically positively skewed, log transformation of lead data (presented as geometric means) were often provided by included studies. To prepare data ready for meta-analysis, we performed natural log transformation of all geometric means. We calculated standard deviations from geometric confidence intervals where necessary using the calculation for small sample size (Higgins 2011). If arithmetic means and

standard deviations were provided, we contacted authors to clarify that data were normally distributed and if no clarification was available, we assumed that the data were normally distributed. We then converted arithmetic means and standard deviations to approximate means and standard deviations on the log transformed scale according to Higgins 2008, before including in the meta-analysis. Where raw data were available, we calculated post-treatment means and standard deviations on the log-transformed data.

Binary data

Where outcomes from either standardised instruments or diagnostic evaluations were expressed as proportions, we calculated the relative risk with 95% confidence intervals. For dichotomous data, we performed analysis on the number of children with blood lead levels above two thresholds of $\geq 10\mu\text{g/dL}$ ($0.48\ \mu\text{mol/L}$) and $\geq 15\mu\text{g/dL}$ ($0.72\ \mu\text{mol/L}$).

Incorporation of a cluster-randomised trial for meta-analysis

To determine the impact of possible unit of analysis errors arising from inadequate adjustment for cluster randomisation in published results by Hiltz 1995, we used a range of intraclass correlation coefficients (ICCs) to calculate a design effect to reduce the size of each trial to its 'effective sample size' (Higgins 2011). We then used data generated from this approach in the meta-analysis. We used a range of ICCs (0.001, 0.01, 0.1, 0.2) due to no reliable ICCs being available from cluster trial authors, similar studies or resources that provide examples of ICCs (Ukoununne 1999). We calculated design effects according to the equation: $1+(M-1)ICC$, where $M=6$, the average cluster size of households used in the study (Hiltz 1995). Design effects calculated using an ICC of 0.001 or less resulted in no change in the sample sizes for intervention and control and so we did not use this data in further analysis.

Dealing with missing data

Where some data on trial methods or results were not reported, we contacted trial authors. Where no reply was forthcoming or full data were not made available, we only included data in meta-analysis where possible. There was insufficient data to impute results or conduct sensitivity analysis on a 'best-case/worst case' basis.

Assessment of heterogeneity

We assessed consistency of results visually and by examining I^2 (Higgins 2002), a quantity that describes approximately the proportion of variation in point estimates that is due to heterogeneity rather than sampling error. This was supplemented with a test of homogeneity to determine the strength of evidence that the heterogeneity was genuine.

Assessment of reporting biases

We intended to use funnel plots to investigate relationship between effect size and study precision (closely related to sample size) (Egger 1997). However, due to the small number of included studies, this was not possible.

Data synthesis

When two or more studies reported data that could be combined, we performed a meta-analysis. For any given outcome, mean difference (MD) and relative risk (RR) for dichotomous data, with their 95% confidence intervals (CIs), were calculated for continuous and dichotomous data, respectively, using a random-effects model.

Subgroup analysis and investigation of heterogeneity

Studies were put into subgroups for clinically different interventions as follows.

1. Educational
2. Environmental (household) - dust control and soil abatement
3. Combination - educational and dust control

Due to limited number of studies within each intervention type, there was insufficient data for subgroup analysis for baseline age or baseline blood lead level.

Sensitivity analysis

We conducted a sensitivity analysis to assess the impact of one study (Brown 2006) on the meta-analysis as it had higher baseline blood levels than the other studies within the educational intervention subgroup.

Sensitivity analysis based on risk of bias analysis was planned but due to the studies meta-analysed being of similar low risk of bias, this was not required.

RESULTS

Description of studies

See: [Characteristics of included studies](#); [Characteristics of excluded studies](#).

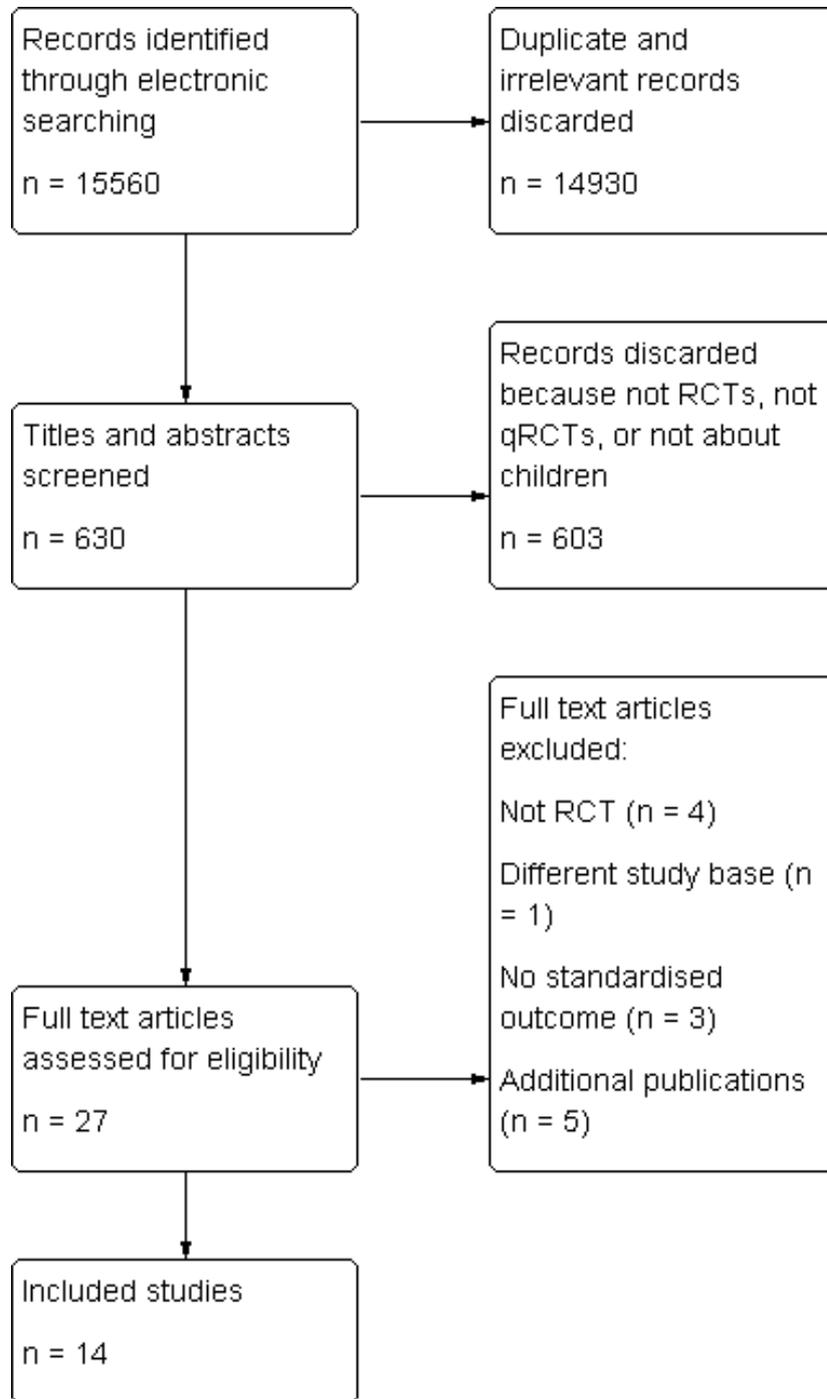
Results of the search

The original literature search for this review was completed at the end of May 2006 and yielded 11,655 titles. We rejected articles at title and abstract stage if they were not primarily about lead exposure in children, were not randomised/quasi-randomised control trials or did not fulfil the inclusion criteria as outlined above. We conducted full text reviews of 25 promising papers and, of these, 20 separate trials were identified (with five papers being additional publications for these trials). From the 20 separate trials identified, we included 12 and excluded eight. We did not identify any unpublished papers or ongoing papers.

An updated search was run at the end of April 2010 and yielded 2951 additional titles. After excluding based on above methods at the title and abstract stage, there was one additional study identified, resulting in 13 trials being included in the review overall.

Another updated search was run end of January 2012 and yielded 954 titles. After excluding based on above methods at the title and abstract stage, there was one additional study identified, resulting in 14 trials being included in the review overall. [Figure 1](#) shows the screening process for the total number of records found for this review to date.

Figure 1. Study flow diagram



Included studies

Please see [Characteristics of included studies](#).

Design

Thirteen RCTs and one quasi-randomised control trial ([Charney 1983](#)) were included in this review and included 2656 children under the age of six years. All studies used a parallel design, with one study ([Weitzman 1993/Aschengrau 1994](#)) also performing the intervention on volunteers from the control group at a later date (phase II). As no control was used, we did not include these phase II results in our review. Another study by [Campbell 2011](#) included a matched control group at the analysis stage. This group had been pre-specified in the study methods but was not part of the randomisation process, therefore could not be included in the results of this review. Twelve studies used individuals (or households) and two studies ([Hilts 1995](#); [Farrell 1998](#)) used clusters (neighbourhoods and blocks of six households, respectively) as the unit of allocation for randomisation.

Sample sizes

Four studies had fewer than 100 participants ([Charney 1983](#); [Aschengrau 1998](#); [Wasserman 2002](#); [Boreland 2009](#)), six had 100 to 200 participants ([Weitzman 1993](#); [Hilts 1995](#); [Lanphear 1996a](#); [Rhoads 1999](#); [Sterling 2004](#); [Brown 2006](#)) and four had more than 200 participants ([Farrell 1998](#); [Lanphear 1999](#); [Jordan 2003](#); [Campbell 2011](#)).

Participants and setting

Thirteen included studies were carried out in urban areas of the USA, with one study performed in Broken Hill, Australia ([Boreland 2009](#)). The majority of studies were performed in areas of lower socioeconomic status, with a significant proportion of participants living in rental accommodation with below average household income levels. More than half of the included studies had significant proportions of people identifying themselves as African-American or Hispanic. Males and females were represented equally in all studies. No measure of the baseline cognitive or neurobehavioural status was available for participants in any included study.

Thirteen studies recruited their participants from routine screening programs, medical clinics, previous lead studies or community volunteers and excluded children who had clinical symptoms, were having treatment for lead toxicity (for example, chelation) or had high blood lead levels requiring intervention (> 20 to 24 $\mu\text{g}/\text{dL}$; 0.97 to 1.16 $\mu\text{mol}/\text{L}$). [Charney 1983](#) recruited participants

from a lead poisoning clinic and 15% of children were reported to have had previous treatment for lead toxicity.

Baseline mean blood lead levels varied across studies with five studies reporting low levels (<10 $\mu\text{g}/\text{dL}$; 0.48 $\mu\text{mol}/\text{L}$) ([Lanphear 1996a](#); [Lanphear 1999](#); [Wasserman 2002](#); [Jordan 2003](#); [Campbell 2011](#)); five reporting low to moderate levels (10 to 14 $\mu\text{g}/\text{dL}$; 0.48 to 0.68 $\mu\text{mol}/\text{L}$) ([Weitzman 1993](#); [Hilts 1995](#); [Farrell 1998](#); [Rhoads 1999](#); [Sterling 2004](#)); three reporting moderate levels (15 to 19 $\mu\text{g}/\text{dL}$; 0.72 to 0.92 $\mu\text{mol}/\text{L}$) ([Aschengrau 1998](#); [Brown 2006](#); [Boreland 2009](#)), and one reporting high levels (> 20 $\mu\text{g}/\text{dL}$; 0.97 $\mu\text{mol}/\text{L}$) ([Charney 1983](#)) ([Table 1](#)).

With regards to the age at baseline, the children in three studies had a mean age of less than 12 months ([Lanphear 1999](#); [Jordan 2003](#); [Campbell 2011](#)); four studies had mean ages between 12 and 24 months ([Lanphear 1996a](#); [Rhoads 1999](#); [Wasserman 2002](#); [Brown 2006](#)); three studies had mean ages between 24 and 36 months ([Weitzman 1993](#); [Hilts 1995](#); [Aschengrau 1998](#)), and three studies had mean ages greater than 36 months ([Charney 1983](#); [Sterling 2004](#); [Boreland 2009](#)). One study did not report mean age; the age range was six months to six years ([Farrell 1998](#)) ([Table 2](#)).

Interventions

The interventions used in the studies were either educational, environmental or a combination of these. In studies using educational interventions, three studies used education alone ([Wasserman 2002](#); [Jordan 2003](#); [Brown 2006](#)) and two studies used education with supply of cleaning products ([Lanphear 1996a](#); [Lanphear 1999](#)). Of the studies using environmental interventions, two studies used soil abatement ([Weitzman 1993](#); [Farrell 1998](#)) and three used dust control interventions ([Hilts 1995](#); [Rhoads 1999](#); [Boreland 2009](#)). Four studies used a combination of lead dust control, education and/or hazard reduction interventions ([Charney 1983](#); [Aschengrau 1998](#); [Sterling 2004](#); [Campbell 2011](#)) ([Table 2](#)).

Intervention integrity

We contacted trial authors to provide additional information about intervention integrity. Authors reported general difficulties in providing consistent environmental and educational interventions in a community setting and inconsistent compliance with recommended housekeeping practices. Measures of compliance were not performed.

Control

One study used a placebo attention-control group in which participants received an accident prevention intervention and were given

home safety items (Rhoads 1999). Thirteen studies did not use any placebo intervention. Seven studies (Charney 1983; Aschengrau 1998; Farrell 1998; Lanphear 1999; Wasserman 2002; Brown 2006; Campbell 2011) gave the control groups lead educational information, dust control and/or hazard reduction available to the general community with no additional input from the researchers. In three studies (Hilts 1995; Lanphear 1996a; Boreland 2009), both intervention and control groups received basic educational brochures or information about reduction of lead hazards separate to the intervention. In two studies (Jordan 2003; Sterling 2004), both groups received home lead assessment and feedback and in one study (Weitzman 1993), both groups received internal lead hazard reduction with the intervention group also receiving the intervention of interest in the study (soil abatement).

Intervention duration

The duration of intervention for twelve studies ranged between three months and 24 months. In the two studies that used soil abatement intervention (Farrell 1998, Weitzman 1993), the intervention was performed on a single occasion within the study duration.

Outcomes

Blood lead level was the standardised outcome reported in all studies. No studies used any standardised cognitive and neurobehavioural outcomes. Environmental outcomes, including household dust and lead loading, were reported in nine studies (Weitzman 1993; Hilts 1995; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Rhoads 1999; Sterling 2004; Brown 2006; Campbell 2011) (Table 3).

Both continuous and dichotomous blood lead level data were available from seven studies (Charney 1983; Hilts 1995; Lanphear 1996a; Lanphear 1999; Rhoads 1999; Wasserman 2002; Brown 2006). Five studies provided only continuous data (Weitzman 1993; Aschengrau 1998; Jordan 2003; Boreland 2009; Campbell 2011); one study provided only dichotomous data (Sterling 2004), and one study reported results in terms of 'total effect' (Farrell 1998). Additionally, raw data were available for three studies (Lanphear 1996a; Lanphear 1999; Wasserman 2002).

For continuous data, seven of the twelve studies reported geometric means (Hilts 1995; Lanphear 1996a; Lanphear 1999; Jordan 2003; Brown 2006; Boreland 2009; Campbell 2011) and five studies reported arithmetic means (Charney 1983; Weitzman 1993; Aschengrau 1998; Rhoads 1999; Wasserman 2002). Data from Aschengrau 1998 were reported as having normal distribution. As no clarification was available for remaining studies providing arithmetic means, it was assumed that the data were normally distributed.

Limited data detailing study costs were available for six studies (Hilts 1995; Farrell 1998; Wasserman 2002; Sterling 2004;

Brown 2006; Boreland 2009). Information on adverse outcomes was available for five studies (Hilts 1995; Farrell 1998; Rhoads 1999; Wasserman 2002; Brown 2006) and none of these reported significant adverse events.

Follow-up duration

The period of follow-up ranged from six months to 48 months from baseline with the majority of studies reporting blood lead levels measured from three to 12 months post-intervention. Two studies provided longer follow-up (Lanphear 1999; Jordan 2003). Lanphear 1999 collected data up to 18 months post intervention with a follow-up publication at 48 months follow-up (Lanphear 2000). Jordan 2003 had follow-up data reported at four-monthly intervals up to three years post intervention.

Short-term post intervention data were used from the two long-term studies (six months for Lanphear 1999 and 18 months for Jordan 2003) in our meta-analysis to enable a more comparable follow-up period to other included studies. With regard to household dust level outcomes, six-month follow-up data were used for the two studies with available data (Lanphear 1996a; Lanphear 1999).

Excluded studies

Please see [Characteristics of excluded studies](#).

Of the eight excluded papers, three studies used retrospective or historical controls without randomisation (EPA 1996; Taha 1999; Pollak 2002); one study reported long-term follow up for an included trial but did not use controls (Aschengrau 1994); one study compared two groups from different study bases (Omidpanah 1998), and three studies did not report any standardised measure in children as an outcome (with Boreland 2006 reporting on environmental measures, Dugbatey 2005 reporting on maternal blood levels and Marlowe 2001 reporting hair lead levels).

Risk of bias in included studies

Thirteen studies were randomised and one was a quasi-randomised controlled trial in which alternate clinic numbers determined allocation to groups (Charney 1983). We received responses from all authors when we contacted them to provide missing information on methodology or results but, in many instances, some of the information requested for methodology and/or results was not available.

Allocation

Participation

The participation rate varied significantly between studies. In two studies where patients were recruited from medical clinics,

participation rate was 100% (Charney 1983; Wasserman 2002). Nine studies used population-based enrolment. Of these, six studies reported a 60% to 80% participation rate (Weitzman 1993; Hiltz 1995; Lanphear 1999; Rhoads 1999; Sterling 2004; Brown 2006) and three studies reported less than 60% (Lanphear 1996a; Aschengrau 1998; Boreland 2009). For the three remaining studies, authors were unable to specify participation rate for two studies due to the use of a community/outreach recruitment process in which volunteers were recruited from community posters and/or door knocks (Farrell 1998; Jordan 2003). In another, the participation rate could not be determined as it was not clear how many children attended the outpatient practices where recruitment took place (Campbell 2011).

Sequence generation

Of the 13 RCTs, methods of randomisation were available for 12 RCTs and remained unclear in one study (Sterling 2004). The quasi-randomised study in which alternate clinic numbers determined allocation to groups was not included in this section (Charney 1983). Method of randomisation was adequate for the twelve studies with available information. Eight studies used random number generators, tables or lists; two studies used coin toss; one study used numbered slips of paper, and one study used permuted blocks of varying length.

Allocation concealment

Of the 13 RCTs, eight studies had adequate allocation concealment by the use of sealed envelope or a central office and allocation concealment remained unclear in two studies (Sterling 2004; Campbell 2011). Three studies did not report adequate concealment (Weitzman 1993; Aschengrau 1998; Boreland 2009).

Blinding

In some studies, not all participants or study personnel were blinded. Blinding for all outcome assessors for dust and blood samples was performed in all studies except one where no information on blinding was provided (Campbell 2011).

Incomplete outcome data

Eight studies had more than 80% follow-up (Weitzman 1993; Hiltz 1995; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Rhoads 1999; Brown 2006; Boreland 2009); three studies had 60% to 80% follow-up (Charney 1983; Wasserman 2002; Jordan 2003), and three studies had less than 60% follow-up (Farrell 1998; Sterling 2004; Campbell 2011) and were not included in meta-analysis. Summarised for each intervention subgroup: education subgroup follow-up ranged from 60% to 90%; dust control subgroup follow-up ranged from 85% to 95%; soil abatement subgroup ranged from 45% to 95%, and combination subgroup

ranged from 35% to 90%. The most common reasons reported for withdrawal were that families had moved out of the area or were no longer contactable.

We contacted authors to determine if participants were analysed in the groups to which they were randomised (intention to treat). Complete measure of all participants' outcomes (full intention to treat analysis) was not possible in any study due to loss of contact with some participants in all studies. Seven studies analysed data based on available participants' outcomes (available case analysis) (Weitzman 1993; Hiltz 1995; Lanphear 1996a; Lanphear 1999; Brown 2006; Boreland 2009; Campbell 2011). The review authors were unable to determine if data from all available participants was used without correction in five studies (Charney 1983; Rhoads 1999; Wasserman 2002; Jordan 2003; Sterling 2004). In two studies (Aschengrau 1998; Farrell 1998, participants were excluded from analysis if non-study interventions (such as any lead hazard reduction measures performed independently of study intervention) occurred during the study.

Selective reporting

Although it was difficult gathering all required information to accurately assess this, information from authors suggest that published reports include all expected outcomes, including those that were pre-specified. This applied to all except for Campbell 2011, where the data on blood lead levels in children at 12 months of age, for prespecified intervention and control groups, were not reported separately but as a combined group that was compared with a matched control group which had not been included in the initial randomisation process.

Other potential sources of bias

Unit of allocation

Twelve studies used individual children or households as their unit of allocation for intervention and analysis and two studies used cluster allocation. One of these used neighbourhood clusters (Farrell 1998) and it was unclear how analysis was performed as data were not available. The other study used clusters of six households (Hiltz 1995) but used individuals as unit for analysis and therefore introduced a unit of analysis error.

Effects of interventions

See: [Summary of findings for the main comparison Education strategies for preventing domestic lead exposure in children](#); [Summary of findings 2 Environmental strategies \(dust control\) for preventing domestic lead exposure in children](#)

We present results sequentially by intervention type, by outcome measure and by type of data, i.e. continuous and dichotomous.

The 14 studies were put in subgroups based on type of intervention as combining these significantly different types of intervention would not be clinically appropriate.

1. Education (Lanphear 1996a; Lanphear 1999; Wasserman 2002; Jordan 2003; Brown 2006).

2. Environmental

a) Dust control (Hilts 1995; Rhoads 1999; Boreland 2009).

b) Soil abatement (Weitzman 1993; Farrell 1998).

3. Combination - education and dust control (Charney 1983; Aschengrau 1998; Sterling 2004; Campbell 2011).

Education

Cognitive and neurobehavioural outcomes

None of the included studies measured cognitive or neurobehavioural outcomes of their participants.

Blood lead level outcomes

Five studies of educational interventions were available for meta-analysis (Lanphear 1996a; Lanphear 1999; Wasserman 2002; Jordan 2003; Brown 2006). Geometric means were readily available from all authors except Wasserman 2002 who provided raw data.

Continuous data

Meta-analysis of log transformed summary data showed no evidence of a treatment effect (MD 0.02, 95% CI -0.09 to 0.12, $I^2 = 0$; Analysis 1.1). Exponentiation of the result produced a treatment effect of 1.02, 95% CI 0.91 to 1.13. The mean age for all studies was less than two years of age and baseline blood level of all except in Brown 2006 was low ($< 10 \mu\text{g/dL}$; $0.48 \mu\text{mol/L}$). As the baseline blood lead level for Brown 2006 was in the moderate range (15 to $19 \mu\text{g/dL}$; 0.72 to $0.92 \mu\text{mol/L}$), a sensitivity analysis was performed to assess the effect of clinical heterogeneity. When Brown 2006 was excluded, there was still no evidence of a treatment effect (MD -0.01, 95% CI -0.13 to 0.11; $I^2 = 0$). Exponentiation of the result produced a treatment effect of 0.99, 95% CI 0.88 to 1.12.

Dichotomous data

We performed meta-analysis of dichotomous data for four studies as dichotomous outcomes were not available for Jordan 2003. Meta-analysis for numbers of children with blood lead level $\geq 10 \mu\text{g/dL}$ ($0.48 \mu\text{mol/L}$) showed no evidence of a treatment effect (RR 1.02, 95% CI 0.79 to 1.30, $I^2=0$; Analysis 1.2). Meta-analysis of data reported as numbers of children with blood lead level $\geq 15 \mu\text{g/dL}$ ($0.72 \mu\text{mol/L}$) showed a trend supporting the intervention

but no statistically significant effect (RR 0.60, 95% CI 0.33 to 1.09, $I^2 = 0$; Analysis 1.3).

Household floor dust outcomes

Continuous data

Two studies (Lanphear 1996a; Lanphear 1999) of the five had log transformed summary data available on hard floor dust lead levels for this intervention. The meta-analysis of the log transformed summary data showed no evidence of treatment effect (MD -0.07, 95% CI -0.37 to 0.24; Analysis 1.4). Exponentiation of the result produced a treatment effect of 0.93, 95% CI 0.69 to 1.27.

Environmental

Cognitive and neurobehavioural outcomes

None of the included studies measured cognitive or neurobehavioural outcomes in their participants.

Blood lead level outcomes

Dust control

Continuous data

Three studies (Hilts 1995; Rhoads 1999; Boreland 2009) used dust control interventions. Hilts 1995 and Boreland 2009 reported log transformed summary data while Rhoads 1999 reported arithmetic means and standard deviations. The meta-analysis of log transformed summary data showed no evidence of a treatment effect (MD -0.15, 95% CI -0.42 to 0.11; Analysis 2.1). Exponentiation of the result produced a treatment effect of 0.86, 95% CI 0.66 to 1.12.

Dichotomous data

We performed meta-analysis of dichotomous data for two studies (Hilts 1995; Rhoads 1999). Meta-analysis for numbers of children with blood lead level $\geq 10 \mu\text{g/dL}$ ($0.48 \mu\text{mol/L}$) showed no evidence of a treatment effect (RR 0.93, 95% CI 0.73 to 1.18, $I^2=0$; Analysis 2.2) as was also the case for children with blood lead levels $\geq 15 \mu\text{g/dL}$ ($0.72 \mu\text{mol/L}$) (RR 0.86, 95% CI 0.35 to 2.07, $I^2 = 56$; Analysis 2.6).

Impact of clustering and unit of analysis errors

Effective sample sizes were calculated for the cluster-randomised trial (Hilts 1995) for a range of ICCs before incorporating into meta-analysis. For blood lead levels ≥ 10 $\mu\text{g/L}$ (0.48 $\mu\text{mol/L}$), there was no statistically significant treatment benefit when meta-analysis was adjusted for clustering: ICC 0.01 (RR 0.93, 95% CI 0.73 to 1.18, $I^2=0$; Analysis 2.3); ICC of 0.1 (RR 0.95, 95% CI 0.72 to 1.24, $I^2=0$; Analysis 2.4); ICC of 0.2 (RR 0.97, 95% CI 0.72 to 1.29, $I^2=0$; Analysis 2.5). For blood lead levels ≥ 15 $\mu\text{g/dL}$ (0.72 $\mu\text{mol/L}$), there was no statistically significant treatment benefit when meta-analysis was adjusted for clustering: ICC 0.01 (RR 0.82, 95% CI 0.37 to 1.81, $I^2=45$; Analysis 2.7); ICC 0.1 (RR 0.83, 95% CI 0.34 to 2.03, $I^2=48$; Analysis 2.8); ICC 0.2 (RR 0.75, 95% CI 0.34 to 1.66, $I^2=25$; Analysis 2.9). Thus, correcting for unit of analysis errors did not alter the overall outcome.

Soil abatement

Two studies (Weitzman 1993; Farrell 1998) performed soil abatement interventions. As no blood lead level data was available in a usable form from one study (Farrell 1998) and follow up was less than 60%, comparison was not possible. Farrell 1998 reported results as 'total effect' showing no statistical significance and no data was available for our analysis. Weitzman 1993 reported a statistically significant effect from intervention. Difference in mean change scores between the intervention group and control group A (loose interior dust abatement and paint removal) was -1.53 $\mu\text{g/dL}$ (standard deviation (SD): 4.9) and between the intervention group and control group B (loose interior paint removal only) was -1.92 $\mu\text{g/dL}$ (SD 5.0). No measure of variance was available for post-treatment means or mean change scores so further analysis was not possible in our review.

Household floor dust outcomes

Dust control

One study (Hilts 1995) provided household carpet lead measures for dust control interventions. No clinically significant treatment effect was reported with geometric means for post-treatment for dust lead level being 0.36 mg/m^2 (SD 3.38) in the intervention group and 0.23 mg/m^2 (SD 3.29) in the control group.

Soil abatement

No studies reported household dust lead levels for this intervention.

Combination

Cognitive and neurobehavioural outcomes

None of the included studies measured cognitive or neurobehavioural outcomes in their participants.

Blood lead level outcomes

Of the four studies that used a combination of interventions, two (Aschengrau 1998; Campbell 2011) reported continuous data, but for one study only baseline blood lead levels were reported (Campbell 2011). One study reported dichotomous data (Sterling 2004), and the fourth (Charney 1983) was clinically very different being a quasi-randomised trial with high mean baseline blood lead levels (> 30 $\mu\text{g/dL}$ (1.44 $\mu\text{mol/L}$)) and older participants (mean age 3.5 years). It was therefore not possible or appropriate to combine any of these studies.

Aschengrau 1998 reported arithmetic means for post-treatment blood lead levels as 11.5 $\mu\text{g/dL}$ (SD 3.22) in intervention group and 10.4 $\mu\text{g/dL}$ (SD 3.12) in control group. An analysis of these post-treatment scores performed in our review failed to reach statistical significance with a mean difference of 1.10 (95% CI -1.45 to 3.65). Sterling 2004 reported dichotomous data with four out of 10 (40%) in intervention group one; six out of 14 (43%) in intervention group two, and six out of 15 (40%) in control group having blood lead levels < 10 $\mu\text{g/dL}$ (0.48 $\mu\text{mol/L}$) post treatment but this study had small numbers and less than 40% follow up. An analysis of this data performed in our review, reported as numbers of children with blood lead levels ≥ 10 $\mu\text{g/dL}$ (0.48 $\mu\text{mol/L}$), showed no evidence of treatment effect (intervention group one (newsletters and education): RR 1.0, 95% CI 0.52 to 1.92; intervention group two (newsletters, education and specialised cleaning): RR 0.95, 95% CI 0.52 to 1.76). Charney 1983 reported a significant effect favouring treatment with arithmetic means for post-treatment blood lead levels of 31.7 $\mu\text{g/dL}$ (SD 2.6) in the intervention group and 37.8 $\mu\text{g/dL}$ (SD 7.9) in the control group. Campbell 2011 reported geometric means for baseline blood lead levels only for the intervention group (2.6 $\mu\text{g/dL}$) and control group (2.7 $\mu\text{g/dL}$). A post-treatment analysis is planned at two years follow-up.

Household floor dust outcomes

One study (Aschengrau 1998) provided continuous data of hard floor dust lead levels for this intervention subgroup. No treatment effect was found with median changes for dust lead level being -0.15 $\mu\text{g/ft}^2$ (SD 0.81) in the intervention group and 0.03 $\mu\text{g/ft}^2$ (SD 0.23) in the control group. A second study (Campbell 2011) provided dichotomous data with no significant difference observed in the number of households with positive dust lead levels (floor > 40 $\mu\text{g/sq.ft.}$; window > 250 $\mu\text{g/sq.ft.}$) between the intervention (17/59) and control (11/51) groups at 12 months post-treatment.

Adverse events

Few studies reported adverse events. We contacted each author to obtain further data. No significant adverse effects were reported by five studies (Hilts 1995; Farrell 1998; Rhoads 1999; Wasserman 2002; Brown 2006). Nine studies (Charney 1983; Weitzman 1993; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Jordan 2003; Sterling 2004; Boreland 2009; Campbell 2011) did not collect data about adverse events.

Cost data

Six studies provided cost data for their intervention or study. Large variations in costs were reported depending on the type of intervention and types of cost data collected. The costs of researcher and educators were often not included in the calculation. With regard to educational interventions, Brown 2006 noted that comparison families on average spent \$108.78 and intervention families spent

\$43.01 on cleaning supplies. Wasserman 2002 reported that Medicaid paid for medical check-ups and researchers spent \$11 per blood test. With dust control interventions, Hilts 1995 reported that the entire study cost approximately \$200,000 but no detailed costs for intervention was available. Boreland 2009 reported that the average cost per household was \$5,000 (Australian dollars in 1994) but ranged from \$1000 to \$20,000. For soil abatement, Farrell 1998 estimated that the average cost per household was \$1,700, with the entire study costing \$5,000,000. For combination intervention, Sterling 2004 reported average cost per quarterly cleaning at \$500 per household and Campbell 2011 reported median costs of Lead Hazard Control or remediation work over a 12month period of \$4,656 for 42 control households and \$5,512 for 36 intervention households. No cost data was available for seven studies (Charney 1983; Weitzman 1993; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Rhoads 1999; Jordan 2003).

ADDITIONAL SUMMARY OF FINDINGS [Explanation]

Environmental strategies (dust control) for preventing domestic lead exposure in children						
Patient or population: Children Settings: Households Intervention: Environmental strategies (dust control) Comparison: Regular environment						
Outcomes	Illustrative comparative risks* (95% CI)		Relative effect (95% CI)	No of Participants (studies)	Quality of the evidence (GRADE)	Comments
	Assumed risk	Corresponding risk				
	Control	Environmental strategies (Dust Control)				
Blood lead level (continuous) Blood lead level at end of duration. Scale from: 0 to 30 Follow-up: 6 to 18 months	The mean blood lead level (continuous) ranged across control groups from 2.4 to 2.9 µg/dL ¹	The mean blood lead level (continuous) in the intervention groups was 0.15 lower (0.42 lower to 0.11 higher)		298 (3 studies)	⊕⊕⊕⊕ moderate ²	
Blood lead level (dichotomous ≥ 10 µg/dL) blood lead level Follow-up: 6 to 18 months	Medium risk population ³ 573 per 1000 ⁴	533 per 1000 (418 to 676) ⁴	RR 0.93 (0.73 to 1.18)	210 (2 studies)	⊕⊕⊕⊕ moderate ⁴	
Blood lead level (dichotomous ≥ 15 µg/dL) blood lead level Follow-up: 6 to 18 months	Medium risk population ³		RR 0.86 (0.35 to 2.07) ⁵	210 (3 studies)	⊕⊕⊕⊕ moderate ^{4,5}	

	205 per 1000⁴	176 per 1000 (72 to 424) ⁴	
Cognitive and neurobehavioural outcomes - not reported	See comment	See comment	See comment
		Not estimable	-

*The basis for the **assumed risk** (e.g. the median control group risk across studies) is provided in footnotes. The **corresponding risk** (and its 95% confidence interval) is based on the assumed risk in the comparison group and the **relative effect** of the intervention (and its 95% CI).

CI: Confidence interval; **RR:** Risk ratio;

GRADE Working Group grades of evidence

High quality: Further research is very unlikely to change our confidence in the estimate of effect.

Moderate quality: Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.

Low quality: Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.

Very low quality: We are very uncertain about the estimate.

¹ Change in blood lead level

² Total population size less than 400

³ Baseline based on median of control groups

⁴ Total number of events less than 300,

⁵ 95% CI around pooled estimate includes no effect and appreciable harm or benefit

DISCUSSION

Prevention of lead toxicity in children is an important issue globally, particularly for disadvantaged and developing nation populations. There are many different types of educational, environmental and combination interventions that have been developed to attempt to reduce lead exposure in children and these interventions can involve large resources in terms of costs, time and personnel.

The results of this systematic review suggest that educational and dust control interventions are not effective in reducing children's blood lead levels. However, a trend (although not statistically significant) towards treatment effect for educational interventions was noted in preventing the numbers of children exceeding a threshold blood lead level of 15 µg/dL (0.72 µmol/L). Further studies on populations with substantial proportions of children that have or are at risk of these moderate blood lead levels are needed to clarify this possible benefit. For the soil abatement and combination interventions, two of the included studies reported statistically significant reductions in blood lead level for treatment groups. These results were not able to be meta-analysed as studies used clinically distinct intervention types (soil abatement and combination).

A previous review limited to low-cost lead hazard control interventions and including four trials, reported no substantial effect on mean blood lead concentration but noted treatment effect with dichotomous data for reducing the number of participants with blood lead levels ≥ 15 µg/dL (Haynes 2002). Haynes 2002 differed from our review in that it combined the results of different types of interventions in a meta-analysis. Our review did not find a statistically significant effect for participants with blood lead levels ≥ 15 µg/dL, although a positive trend was noted.

One study showed a statistically significant treatment effect with a combined (education and dust control) intervention (Charney 1983). As this was a quasi-randomised controlled trial and had participants with high baseline blood lead levels (> 30 µg/dL), it was clinically distinct from other included studies. The significant blood lead level reduction after intervention is consistent with previous findings that interventions are likely to have more benefit in children who had higher baseline blood lead levels (Charney 1983; Haynes 2002). This finding requires further research to assess whether or not preventive interventions are better aimed at particular populations of children. Weitzman 1993 estimated intervention effects on blood lead levels of 1.5 to 1.9 µg/dL (0.07 to 0.09 µmol/L). The clinical significance of this on an individual level is likely to be minimal but at a population level may be important. However, the generalisability or reproducibility of the results from these studies is not known. Therefore, as meta-analysis of studies is not available, there is currently insufficient evidence to clarify whether soil abatement or combination interventions reduce blood lead levels.

Meta-analysis was not possible for all interventions or outcomes due to clinical diversity of trials, use of different outcome measures and different forms of data reported. No more than five studies used a similar intervention and even within these intervention subgroups, the reported intervention varied significantly, for example, type of education, duration of intervention, study setting and whether or not supplies were provided. In addition, there were variations in baseline lead levels and mean age.

These issues of clinical diversity, inconsistent participant compliance with household cleaning practices, lower than optimal recruitment numbers and loss to follow-up that reduces study power may all be contributing to the lack of clear effect demonstrated in a meta-analysis of study results. The effectiveness of other more intensive interventions or interventions performed over a longer duration than those available to date is not yet known. Also, the trials in this review largely focus on participants from lower socio-economic status in the USA in rental housing and as such, results may not be generalisable to different populations.

As interventions evaluated were not able to eliminate all ongoing environmental lead sources and were limited to household interventions, it is possible that recontamination occurred during the trial period. Thus, while reduction in lead-contaminated house dust may be needed to reduce or prevent childhood lead exposure, it is not sufficient. It may be necessary to eliminate the ongoing source of lead exposure by removing or eliminating ongoing contamination from lead-based paint and other residential lead hazards. Furthermore, other sources of lead contamination outside the home may have limited the possible benefit of interventions. Another reason for lack of treatment effect may be that the majority of included studies had a follow-up period of 12 months or less and the long half-life of lead in children may contaminate short-term outcomes.

On the surface, these results may appear to be in conflict with observational studies that reported a reduction in dust lead loadings and, on average, a decrease in children's blood lead levels (Clark 2004). But the key question is whether the interim lead hazard controls or partial abatement led to a significant reduction or increase among at-risk (i.e. younger) children who exhibit mouthing behaviours. The observational data actually show that household interventions led to a significant increase in blood lead concentration for young children, especially six-month old infants. Compared with children over 40 months of age, the odds of having an increase in blood lead levels of 5 µg/dL or higher following abatement was 11.2 (95% CI 2.8 to 44.2) for six-month old infants; 3.69 (95% CI 1.68 to 8.09) for 12-month old infants; 1.79 (95% CI 1.07 to 2.99) for 18-month old infants and; 1.18 (95% CI 0.79 to 1.76) for 24-month old infants. These results indicate that the floor clearance levels used by the HUD grantees (< 100 or 200 µg/ft²) were insufficient to protect children. This is not surprising; there is considerable evidence that dust lead levels < 10 µg/ft² are associated with a large increase in the risk of children

having a blood lead level > 10 µg/dL (Lanphear 1996b; Lanphear 1998; Lanphear 2005b; Dixon 2009). Thus, while lead hazard controls or renovation activities can most likely be done safely if empirically-derived dust clearance standards are required, they may actually increase young children's blood lead concentrations if we rely on obsolete standards.

It was noted that none of the studies reviewed used a standardised cognitive or neurobehavioural outcome measure despite this being one of the main adverse outcomes of lead exposure. However, in view of the magnitude of the blood lead level reductions reported in the studies with significant treatment effect, and the known correlation between blood lead level and cognition, no significant improvement in cognitive outcomes would be anticipated even if they had they been used.

Although no significant adverse outcomes were reported in the five studies with available information, nine did not report any collection of adverse events data. Future trials need to better examine and report adverse effects and ensure that sample sizes are sufficiently large to allow this. The societal impact of reducing children's blood lead concentration is considerable. If effective, residential lead hazard controls would be cost-beneficial. Gould 2009 showed that for every dollar invested in lead hazard control, society would benefit by \$17 to \$220. This cost-benefit ratio is better than that for vaccines in developed countries. When reported, the cost data showed large variability between studies but detailed data on costs of interventions would be useful to determine potential cost-effectiveness of various types of interventions.

AUTHORS' CONCLUSIONS

Implications for practice

Based on review of the current research, there is evidence that educational and dust control interventions are not effective in reduc-

ing blood lead levels in children. It is difficult to support the use of the interventions examined in this review as a general population health measure, given their cost and the lack of data showing positive reductions in blood lead levels. There is currently insufficient evidence that soil abatement or combination interventions reduce blood lead levels.

Implications for research

Further trials are required to establish the most effective intervention for prevention of lead exposure in children. Key elements for these trials should include collecting data over longer time periods (36 to 48 months), more intensive interventions that simultaneously reduce multiple sources of lead exposure, different populations, stratification of participants based on baseline blood lead levels, measures of intervention compliance and loss to follow-up. Studies using neurodevelopmental outcomes would also be useful.

Trials that look at suitable interventions in developing countries are also urgently required, as are studies of children in more affluent areas where lead exposure is often due to renovation rather than poor maintenance and may be more short-term and more easily prevented.

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CHARACTERISTICS OF STUDIES

Characteristics of included studies [ordered by study ID]

Aschengrau 1998

Methods	RCT Blinding of outcome assessors for both blood and dust lab analysers 63/402 (16%) enrolled with 41 randomised and 22 subjects at high risk automatically assigned intervention 32/63 blood levels analysed (seven no blood samples, 24 excluded as non-study interventions undertaken) Overall, 24/41 from randomised groups analysed (11 intervention and 13 control) Power calculation performed to determine number of participants (required number not recruited)
Participants	63 children (41 randomised) under four years from Boston located from screening program, mean age 24.5 months, BL blood lead level 16.9 µg/dL
Interventions	Intervention - Low technology lead hazard reduction: -remove lead dust -loose paint chips -HEPA vacuum -Parent education re: cleaning Control - universal outreach and educational activities for both
Outcomes	Blood lead level six months from baseline (Environmental dust levels)
Notes	Blood lead level and dust levels dropped in both intervention groups compared to control (crude and adjusted) but no statistical significance Control group and both intervention groups different baseline characteristics and small sample size Inconsistent parental compliance with housekeeping Several participants had non-study interventions and were excluded from analysis in the article

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "an open list of random numbers"
Allocation concealment (selection bias)	High risk	Quote: "open list"
Blinding (performance bias and detection bias) Blood lead level	Low risk	Quote: "lab analysers were blinded"

Aschengrau 1998 (Continued)

Blinding (performance bias and detection bias) Household dust	Low risk	Quote: “lab analysers were blinded”
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Unclear risk	Insufficient information
Other bias	Low risk	The study appears to be free from other sources of bias

Boreland 2009

Methods	RCT Blinding of outcome assessors for both blood lab analysers 117/365 (32%) enrolled with 103 eligible children randomised and 90 matched by age and blood lead level range (13 were unable to be adequately matched). 88/90 blood levels analysed (two: no blood samples) Intention to treat (available case analysis) Power calculation performed to determine number of participants (required number not recruited)
Participants	90 children aged 1.5 to 5.8 years with blood lead level 15 to 29µg/dL on routine screening in Broken Hill, Australia, mean age 3.5 years, mean blood lead level 19.4 µg/dL
Interventions	Intervention - Home remediation work was performed on intervention households and varied depending on assessment of need to provide each house with a “similar level of lead safety”. Work may have included: ceiling dust removal, sealing of ceilings, paint stabilisation, replacement of floor coverings/windows and cleaning Control - Universal information about minimising lead hazards was provided to both groups
Outcomes	Blood lead level six months from baseline (Environmental dust levels)
Notes	Control group received remediation after completion of study To examine dose response effect, indoor dust levels were measured to examine the extent in which indoor lead levels were associated with changes in blood lead level No significant change in blood lead level between groups.

Risk of bias

Boreland 2009 (Continued)

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from author, "Children were matched in pairs and then a coin tossed to see which would be the 'case' and have their home re-mediated first"
Allocation concealment (selection bias)	Unclear risk	Insufficient information
Blinding (performance bias and detection bias) Blood lead level	Low risk	Nurses collecting blood samples were involved but lab analysers were blinded
Blinding (performance bias and detection bias) Household dust	Low risk	Technical officers collecting dust samples and lab analysers were blinded
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Low risk	The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified and confirmed by investigator
Other bias	Low risk	The study appears to be free from other sources of bias

Brown 2006

Methods	RCT Blinding of outcome assessors 175/241 (73%) enrolled 145/175 (83%) analysed Intention-to-treat (available case analysis) Power calculation performed to determine number of participants (required number recruited)
Participants	175 children under 28 months with blood lead level 15 to 19 µg/dL on routine screening in Rhode Island, mean age 19 months, mean blood lead level 16.5 µg/dL

Interventions	Intervention - Parental Education (with nursing care plan) via five home visits during one year period Control - one to two educational visits by outreach worker available for both
Outcomes	Blood lead level 12 months from baseline (Environmental dust levels) (Questionnaires on lead exposures) (Parental-infant interaction scale)
Notes	blood lead level decrease overall over study in participants but no statistical significance Dust levels decreased for intervention groups but not statistically significant Questionnaire showed significant improvements in reported housekeeping practices in intervention group Parent-Infant scale significantly improved in intervention group

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "Random numbers table was used to assign cases to either the intervention or the comparison group, sequentially"
Allocation concealment (selection bias)	Low risk	Quote: "Group assignments were sealed into envelopes and unknown to either study personnel or the families until after parental consent was obtained"
Blinding (performance bias and detection bias) Blood lead level	Low risk	Quote: "The nurses who provided follow up to comparison group children were blinded and nurses that provided care to intervention group were not blinded" and outcome (laboratory) assessors were blinded
Blinding (performance bias and detection bias) Household dust	Low risk	Quote: Laboratory analysers were "unaware of group assignment."
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups

Brown 2006 (Continued)

Selective reporting (reporting bias)	Low risk	The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified and confirmed by investigator
Other bias	Low risk	The study appears to be free of other sources of bias.

Campbell 2011

Methods	RCT Blinding of outcome assessors not clear Children: 314/314 (100%) enrolled 279/314 (88.9%) blood lead levels analysed at 12 months of age Households: 310/310 (100%) enrolled 110/310 (35.5%) Households evaluated at 12 months Intention-to-treat (not performed) Power calculation performed to determine number of participants (required number recruited)
Participants	314 newborn children from outpatient practices in low income neighbourhoods of Philadelphia (no history of elevated blood lead levels)
Interventions	Intervention - Standard lead poisoning prevention education plus additional extensive education regarding maintaining home in lead-safe condition and visits from trained staff at baseline, 6 and 12 months. Cleaning materials provided Control- Standard lead poisoning prevention education
Outcomes	blood lead levels at 12 months Housing lead dust levels Parental Knowledge Assessment
Notes	No significant difference in blood lead levels between groups at 12 months (baseline reading). A two-year follow-up is planned for blood lead levels A matched comparison group was included in results, receiving community standard for prevention of elevated blood lead levels. This group was not part of the randomisation process Number of households with positive dust wipe results (>40 µg/sq.ft. and window >250µg/sq.ft.) not significantly different. Large number households (65%) lost to follow-up Study did not demonstrated an impact on parental knowledge in the children's first blood lead levels at 12 months of age

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "Randomised blocks using computer-generated random numbers"
Allocation concealment (selection bias)	Unclear risk	Quote: "Study coordinator selected next card in the random sequence to randomise that family"
Blinding (performance bias and detection bias) Blood lead level	High risk	No information provided on blinding
Blinding (performance bias and detection bias) Household dust	High risk	No information provided on blinding
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Majority of children enrolled (88.9%) had blood lead levels measured at 12months. Not clear if missing outcome data was balanced in numbers across groups
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups (59/149 treatment and 51/157 Control)
Selective reporting (reporting bias)	Low risk	blood lead levels outcome data for the Intervention vs Control groups as pre-specified in the methods not fully included in text. Comparison of blood lead levels at analysis stage was between intervention and control groups combined and a matched comparison group not included in initial randomisation process. This analysis however was pre-specified in the Methods

Charney 1983

Methods	Quasi-RCT (even/odd clinic no. assignment) Blinding of outcome assessors 78/78 (100%) enrolled 49/78 (63%) analysed Intention-to-treat (unclear) Power calculation performed to determine number of participants (unclear if required number recruited)
Participants	78 children 15 to 72 months from lead poisoning clinic with blood lead level 30 to 49 µg/dL, mean age 43 months, mean blood lead level 38.6 µg/dL
Interventions	Intervention - Dust control team to wet mop all rooms twice per month and parental education to clean more frequently over 12 months period Control - Routine advice dust control by mopping given at clinic. Paint stabilisation for both groups
Outcomes	Vblood lead level 12 months from baseline (Environmental dust levels in intervention homes only)
Notes	Significant change in blood lead level in intervention gp No significant change in blood lead level in control gp No persistent improvement in dust lead levels in intervention group No significant relation between dust level and child's blood lead level

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	High risk	Allocation method alternate based on "even or odd clinic number"
Allocation concealment (selection bias)	High risk	Not used
Blinding (performance bias and detection bias) Blood lead level	Low risk	Blinding of outcome assessors (laboratory)
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Unclear risk	Insufficient information
Other bias	Unclear risk	Insufficient information

Farrell 1998

Methods	Cluster RCT by neighbourhoods Blinding of outcome assessors Participation rate N/A as community recruitment 182 / 408 (111/263 households) (45%) analysed No intention-to-treat analysis due to those with non-study interventions excluded Power calculation performed to determine number of participants (required number recruited)
Participants	408 children living in the included neighbourhoods of Baltimore six months to six years, mean blood lead level 11 µg/dL
Interventions	Intervention - Soil abatement Control - External paint stabilisation for all in study
Outcomes	Blood lead level two-year from baseline (Soil lead levels)
Notes	Blood lead level decreased in both groups at follow up but no statistical significance Soil lead levels significant decrease in intervention group at one week but re-accumulated in two-year follow up Baseline soil lead levels lower than hypothesised with 54% >1000ppm No internal household interventions Adjacent properties not abated

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote "Coin toss"
Allocation concealment (selection bias)	Unclear risk	Insufficient information
Blinding (performance bias and detection bias) Blood lead level	Unclear risk	Quote: "Specimen collectors and laboratory personnel were blinded to group allocation and analyses were done by the State laboratory which had no interest in the outcome of the study"
Incomplete outcome data (attrition bias) Blood lead level	Unclear risk	Reasons for missing data not provided
Selective reporting (reporting bias)	Unclear risk	Insufficient information
Other bias	Unclear risk	Insufficient information

Hilts 1995

Methods	Cluster RCT (households in blocks of six stratified by area and blood lead level) Blinding of outcome assessors 122/176 (69%) enrolled 111/122 (91%) analysed Intention-to-treat (available case analysis) Power calculation performed to determine number of participants (required number recruited)
Participants	122 households with children under six years in high risk areas (active smelter) identified by 1992 blood screen, mean age 32 months, mean blood lead level 11.6 µg/dL
Interventions	Intervention- HEPA vacuuming (seven times in a ten-month period) Control - Routine advice regarding maintenance and general lead education provided to both groups
Outcomes	Blood lead level ten months from baseline (Hand lead and floor dust and lead levels)
Notes	No statistical or clinical significant change in blood lead level even with multiple regression analysis for baseline blood lead level and area No clinical significance in dust and lead levels Potential for unit of analysis error

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "Drew concealed slips of paper numbered 1-6 without replacement" and assigned blocks and then "coin toss" determined that "odds would be treatment blocks"
Allocation concealment (selection bias)	Low risk	Quote: "Done in central office"
Blinding (performance bias and detection bias) Blood lead level	Low risk	Quote: "blood specimen collector and lab personnel did not know of group assignments"
Blinding (performance bias and detection bias) Household dust	Low risk	Quote: "Lab personnel analysing the carpet dust samples were not aware of group assignment"
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups (55/61 treatment and 56/61 control)

Hilts 1995 (Continued)

Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Low risk	The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified and confirmed by investigator
Other bias	Low risk	The study appears to be free of other sources of bias.

Jordan 2003

Methods	RCT Blinding of outcome assessors Participation rate N/A as community recruitment 378/607 (62%) analysed Intention-to-treat (unclear) Power calculation performed to determine number of participants (unclear if required number recruited)
Participants	594 pregnant women and mothers of children zero to three years in neighbourhood of Phillips, Minneapolis, yielding 607 children recruited by door knocking and community information, mean blood lead level <10 µg/dL
Interventions	Intervention - 20 biweekly culturally specific educational session by peer leaders provided individually and three-monthly boosters until child = three years Control - Routine state health brochures and home lead assessment and feedback to both groups
Outcomes	blood lead level (capillary until 12 months, venous >12 months) three years from baseline
Notes	No statistically significant change in blood lead level Dichotomous data All subjects given financial incentive

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: Random number generator"
Allocation concealment (selection bias)	Low risk	Quote: "Central office"

Jordan 2003 (Continued)

Blinding (performance bias and detection bias) Blood lead level	Low risk	Outcome assessors “laboratory” blinded according to author
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across groups, with similar reasons for missing data across groups and “no evidence that a missing data pattern that differed by randomization group”
Selective reporting (reporting bias)	Low risk	The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified and confirmed by investigator
Other bias	Low risk	The study appears to be free of other sources of bias.

Lanphear 1996a

Methods	<p>RCT</p> <p>Blinding of outcome assessors</p> <p>104/205 (50%) enrolled (no significant difference in those refused)</p> <p>96/104 (91%) analysed</p> <p>Intention to treat (available case analysis)</p> <p>Power calculation not performed to determine number of participants</p>	
Participants	<p>104 children 12 to 31 months recruited from Lead in Dust Study in Rochester, NY, mean age 20 months, mean blood lead level 6.7 µg/dL</p>	
Interventions	<p>Intervention - trained interviewer provided brief lead reduction information, cleaning products to families, demonstration on cleaning and instructions on frequency for household cleaning</p> <p>Control - Brochures on lead poisoning provided to both</p>	
Outcomes	<p>Blood lead level seven months from baseline</p> <p>(Household dust samples)</p>	
Notes	<p>No statistical significant difference noted in change in median blood lead level at follow up</p> <p>No statistical significant change in dust lead levels between groups</p>	
<i>Risk of bias</i>		
Bias	Authors’ judgement	Support for judgement

Lanphear 1996a (Continued)

Random sequence generation (selection bias)	Low risk	Quote: "Computer random number generator"
Allocation concealment (selection bias)	Low risk	Quote: "Sealed opaque envelopes"
Blinding (performance bias and detection bias) Blood lead level	Low risk	Quote: "Yes, blood lead specimen collectors and analysers were blinded to group allocation."
Blinding (performance bias and detection bias) Household dust	Unclear risk	Author was unable to recall this information
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups and small numbers (8/104)
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups;
Selective reporting (reporting bias)	Low risk	The study protocol is available and all of the study's pre-specified outcomes reported in the pre-specified way
Other bias	Low risk	The study appears to be free of other sources of bias.

Lanphear 1999

Methods	RCT (also non-study control to rule out Hawthorne effect) Blinding of outcome assessors 275/429 (64%) enrolled 245/275 (89%) and 189/275 (69%) analysed at 24 and 48 months, respectively Intention-to-treat (available case analysis) Power calculation performed to determine number of participants (required number recruited)
Participants	275 children, six months in Rochester area identified by birth data from five urban hospitals, mean age 6 months, mean blood lead level 2.8ug/dL
Interventions	Intervention - Up to eight visits by dust control advisors, cleaning equipment and supplies in 24 month period Control - Baseline four home visits to both groups
Outcomes	Vblood lead level measured at 6- (baseline), 12-, 18-, 24-, 36- and 48-months (Household dust levels)

Lanphear 1999 (Continued)

Notes	No statistical significant difference in blood lead level at 24 or 48 months between intervention and control groups or percentage of children with raised blood lead level between groups. Decreased levels of dust in both groups at 24 months but no significant difference between groups	
Risk of bias		
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: Random number generator"
Allocation concealment (selection bias)	Low risk	Quote: "Sealed opaque envelopes"
Blinding (performance bias and detection bias) Blood lead level	Low risk	Quote: "Blood lead specimen collectors and analysers were blinded to group allocation".
Blinding (performance bias and detection bias) Household dust	Low risk	Quote: "Environmental technicians and interviewers blind to group assignment"
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Low risk	The study protocol is available and all of the study's pre-specified outcomes reported in the pre-specified way
Other bias	Low risk	The study appears to be free of other sources of bias

Rhoads 1999

Methods	RCT Blinding of outcome assessors 113/147(77%) enrolled 99/113 (87%) analysed Intention-to-treat (unclear) Power calculation performed to determine number of participants (required number not recruited)
Participants	113 children, six to 36 months in Jersey City, NY who responded to posters or referred from community clinics, mean age 20 months, mean blood lead level: 11 to 12 µg/dL
Interventions	Intervention - Biweekly assistance with household cleaning (HEPA vacuum and wet mopping) by community staff members for one year Control - Accident Prevention group given household safety items. Both groups offered education sessions
Outcomes	Blood lead level 12 months from baseline (Household dust and lead levels) (Maternal lead knowledge)
Notes	Statistical significant decrease in blood lead level in intervention group compared to controls Intervention subgroup analysis (39/46 analysed) - statistical significant decrease (26%) in blood lead level in uncarpeted homes compared with no statistical change in carpeted homes Statistical significant decrease in dust and lead levels between groups Statistical significant increase in maternal knowledge in intervention group at follow-up compare with baseline and control

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "Permutated blocks of varying length"
Allocation concealment (selection bias)	Low risk	Quote: "Sealed envelopes"
Blinding (performance bias and detection bias) Blood lead level	Low risk	Outcome assessors blinded
Blinding (performance bias and detection bias) Household dust	Low risk	Outcome assessors blinded
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Reasons for missing data not available but there were relatively small numbers missing with 99/113 analysed

Rhoads 1999 (Continued)

Incomplete outcome data (attrition bias) Household dust	Unclear risk	Numbers and reasons for missing data not available
Selective reporting (reporting bias)	Unclear risk	Insufficient information
Other bias	Unclear risk	Insufficient information

Sterling 2004

Methods	RCT Blinding of outcome assessors 101/132 (76%) enrolled 39/101 (39%) analysed Intention to treat (unclear) Power calculation performed to determine number of participants (unclear if required number recruited)
Participants	101 children, six months to six years in Missouri Superfund area identified through community clinics and screening, mean age three years, mean blood lead level 12.7 µg/dL
Interventions	Intervention 1) Three quarterly educational home visit by nurse and six personalised newsletters over nine month period 2) As above plus three quarterly professional cleans with wet mopping, HEPA and carpet shampooing Control - a standard health education session, baseline home environment assessment and generic brochures for all
Outcomes	Blood lead level three-monthly until nine months from baseline (Household lead dust levels)
Notes	Overall decrease in blood lead level in all groups but no statistical difference in blood lead level between groups No statistical significant change in dust levels between groups No assessment of variation between subjects lost to follow up and those completing study

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	Method of randomisation unknown
Allocation concealment (selection bias)	Unclear risk	Unknown

Sterling 2004 (Continued)

Blinding (performance bias and detection bias) Blood lead level	Low risk	Outcome analysers (laboratory) blinded
Blinding (performance bias and detection bias) Household dust	Low risk	Outcome analysers (laboratory) blinded
Incomplete outcome data (attrition bias) Blood lead level	Unclear risk	Numbers and reasons for missing data not available
Incomplete outcome data (attrition bias) Household dust	Unclear risk	Numbers and reasons for missing data not available
Selective reporting (reporting bias)	Unclear risk	Insufficient information
Other bias	Unclear risk	Insufficient information

Wasserman 2002

Methods	<p>RCT</p> <p>Blinding of outcome assessors</p> <p>63/63 (100%) enrolled</p> <p>50/63 (79%) analysed</p> <p>Intention-to-treat (unclear)</p> <p>Power calculation not performed to determine number of participants</p>	
Participants	<p>Caregivers of 63 children 12 to 36 months of age selected from clients enrolled in Broward County MediPass (Medicaid) who selected Children's Diagnostic and treatment Centre as their health care provider, mean age 22.5 months, mean blood lead level 3 to 4 µg/dL</p>	
Interventions	<p>Intervention - education session at clinic consisting of module, video and brochure at first clinic</p> <p>Control - education session at second clinic</p>	
Outcomes	<p>Blood lead level three to four months from baseline</p> <p>(Parental knowledge - Chicago Lead Knowledge Test)</p>	
Notes	<p>No significant difference in blood lead level at follow up between groups</p> <p>Intervention group had slight decrease in blood lead level versus control group with increase in blood lead level</p> <p>Statistical significant increase in parental knowledge in intervention group post intervention</p>	
<i>Risk of bias</i>		
Bias	Authors' judgement	Support for judgement

Wasserman 2002 (Continued)

Random sequence generation (selection bias)	Low risk	Quote: "Random list of numbers"
Allocation concealment (selection bias)	Low risk	Assigned by central office
Blinding (performance bias and detection bias) Blood lead level	Low risk	Blinding of outcome assessors (laboratory)
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Low risk	The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified and confirmed by investigator
Other bias	Low risk	The study appears to be free of other sources of bias.

Weitzman 1993

Methods	RCT Blinding of outcome assessors 152/236 (64%) enrolled 149/152 (98%) analysed Intention-to-treat (available case analysis) Power calculation performed to determine number of participants (required number recruited)
Participants	152 children under four years from high lead risk areas of Boston with finger prick blood lead level 10 to 20 µg/dL identified on screening in 1989, mean age 31.6 months, mean blood lead level 12 to 13 µg/dL
Interventions	Phase I only Intervention - soil abatement from yard, interior dust abatement, loose interior paint removal Control A - interior dust abatement, loose interior paint removal Control B - loose interior paint removal All received lead education from study staff
Outcomes	Venous blood lead levels 11 months from baseline (Environmental measures)

Weitzman 1993 (Continued)

Notes	Phase I and phase II of Boston Lead-In-Soil trial performed but phase II excluded as no controls At 11 months post soil abatement, small but significant decrease in blood lead level in intervention groups compared with controls	
Risk of bias		
Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote: "Computer based random number generator"
Allocation concealment (selection bias)	High risk	Allocation performed by one staff member but not actively concealed from other investigators enrolling participants
Blinding (performance bias and detection bias) Blood lead level	Low risk	Outcome assessors (laboratory analysers) blinded
Blinding (performance bias and detection bias) Household dust	Low risk	Outcome assessors (laboratory analysers) blinded
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups
Incomplete outcome data (attrition bias) Household dust	Low risk	Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups
Selective reporting (reporting bias)	Unclear risk	Insufficient information
Other bias	Unclear risk	Insufficient information

Characteristics of excluded studies [ordered by study ID]

Study	Reason for exclusion
Aschengrau 1994	No control group utilised for Phase II
Boreland 2006	Outcome only environmental measures before and after intervention
Dugbatey 2005	Outcome (blood lead levels) measured in mothers rather than children, data not in useable form

(Continued)

EPA 1996	Retrospective data collection on two groups not randomly assigned
Marlowe 2001	Outcome measured using hair lead levels
Omidpanah 1998	Control and Intervention groups from two different study bases
Pollak 2002	Historical control group with no randomisation used
Taha 1999	Retrospective control with no randomisation used

DATA AND ANALYSES

Comparison 1. Education

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Blood lead level (continuous)	5	815	Mean Difference (IV, Random, 95% CI)	0.02 [-0.09, 0.12]
2 Blood lead level (dichotomous) ≥ 10 $\mu\text{g/dL}$	4	520	Risk Ratio (M-H, Random, 95% CI)	1.02 [0.79, 1.30]
3 Blood lead level (dichotomous) ≥ 15 $\mu\text{g/dL}$	4	520	Risk Ratio (M-H, Random, 95% CI)	0.60 [0.33, 1.09]
4 Floor dust - hard floor	2	318	Mean Difference (IV, Random, 95% CI)	-0.07 [-0.37, 0.24]

Comparison 2. Environmental - Dust control

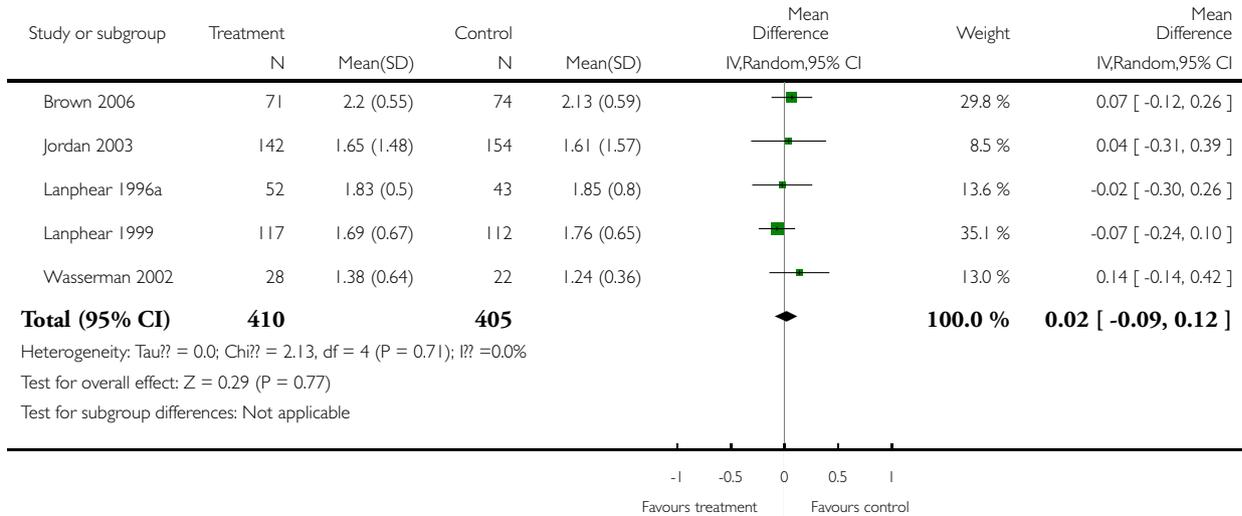
Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Blood lead level (continuous)	3	298	Mean Difference (IV, Random, 95% CI)	-0.15 [-0.42, 0.11]
2 Blood lead level (dichotomous) ≥ 10 $\mu\text{g/dL}$	2	210	Risk Ratio (M-H, Random, 95% CI)	0.93 [0.73, 1.18]
3 Blood lead level (dichotomous) ≥ 10 $\mu\text{g/dL}$ ICC 0.01	2	204	Risk Ratio (M-H, Random, 95% CI)	0.93 [0.73, 1.18]
4 Blood lead level (dichotomous) ≥ 10 $\mu\text{g/dL}$ ICC 0.1	2	173	Risk Ratio (M-H, Random, 95% CI)	0.95 [0.72, 1.24]
5 Blood lead level (dichotomous) ≥ 10 $\mu\text{g/dL}$ ICC 0.2	2	155	Risk Ratio (M-H, Random, 95% CI)	0.97 [0.72, 1.29]
6 Blood lead level (dichotomous) ≥ 15 $\mu\text{g/dL}$	2	210	Risk Ratio (M-H, Random, 95% CI)	0.86 [0.35, 2.07]
7 Blood lead level (dichotomous) ≥ 15 $\mu\text{g/dL}$ ICC 0.01	2	204	Risk Ratio (M-H, Random, 95% CI)	0.82 [0.37, 1.81]
8 Blood lead level (dichotomous) ≥ 15 $\mu\text{g/dL}$ ICC 0.1	2	173	Risk Ratio (M-H, Random, 95% CI)	0.83 [0.34, 2.03]
9 Blood lead level (dichotomous) ≥ 15 $\mu\text{g/dL}$ ICC 0.2	2	155	Risk Ratio (M-H, Random, 95% CI)	0.75 [0.34, 1.66]

Analysis 1.1. Comparison 1 Education, Outcome 1 Blood lead level (continuous).

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 1 Education

Outcome: 1 Blood lead level (continuous)

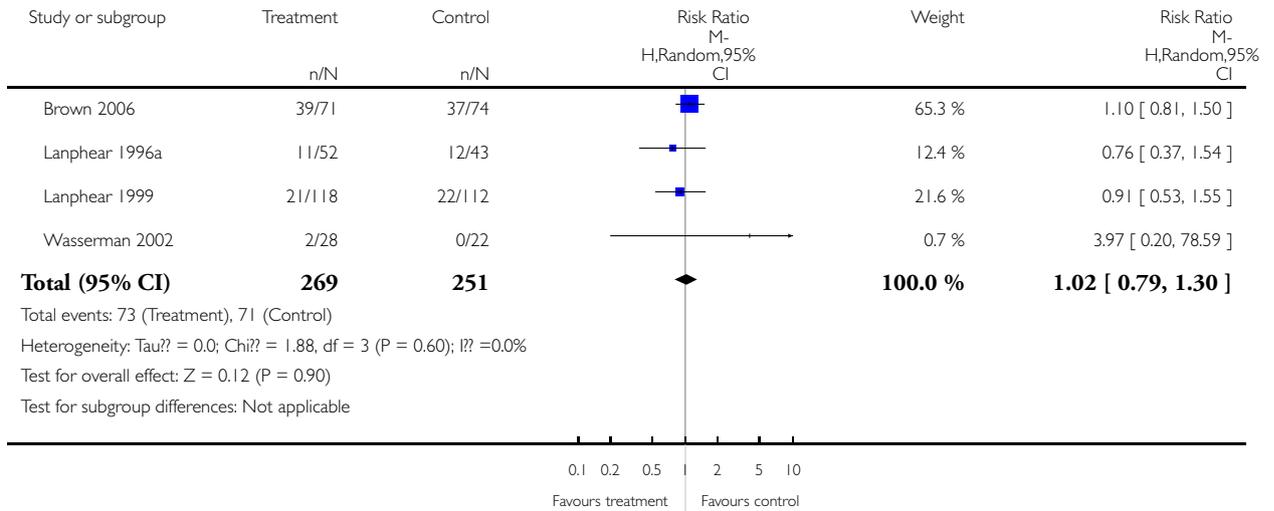


Analysis 1.2. Comparison 1 Education, Outcome 2 Blood lead level (dichotomous) $\geq 10 \mu\text{g/dL}$.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 1 Education

Outcome: 2 Blood lead level (dichotomous) $\geq 10 \mu\text{g/dL}$

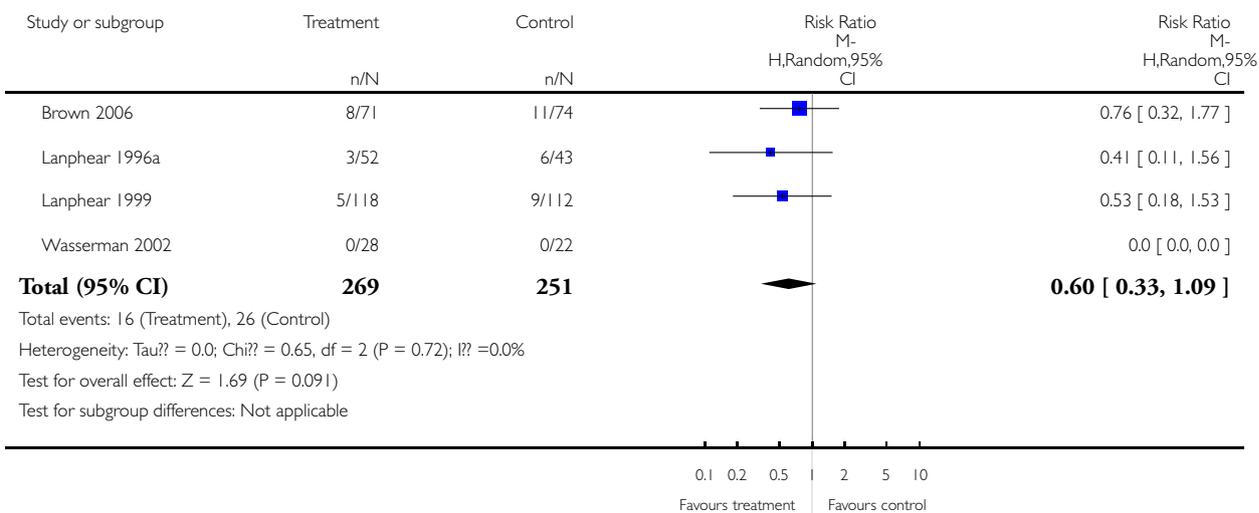


Analysis 1.3. Comparison 1 Education, Outcome 3 Blood lead level (dichotomous) $\geq 15 \mu\text{g/dL}$.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 1 Education

Outcome: 3 Blood lead level (dichotomous) $\geq 15 \mu\text{g/dL}$

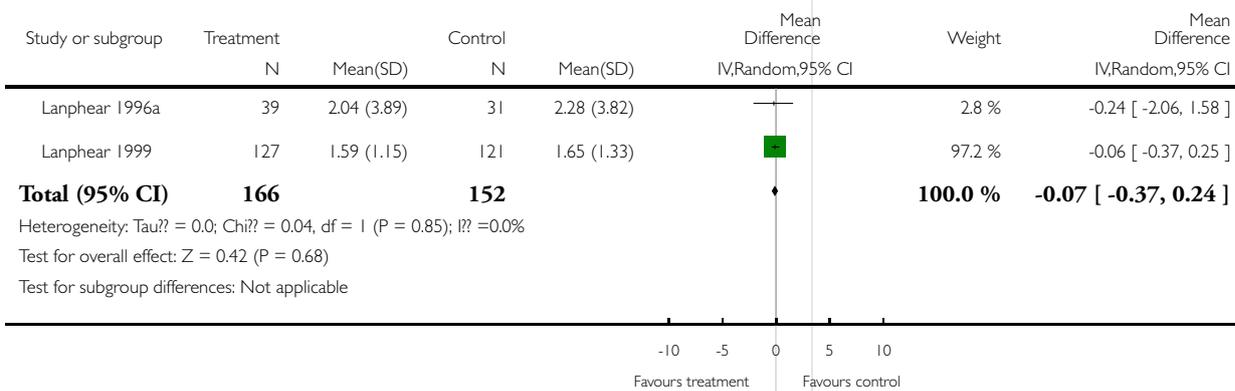


Analysis 1.4. Comparison 1 Education, Outcome 4 Floor dust - hard floor.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 1 Education

Outcome: 4 Floor dust - hard floor

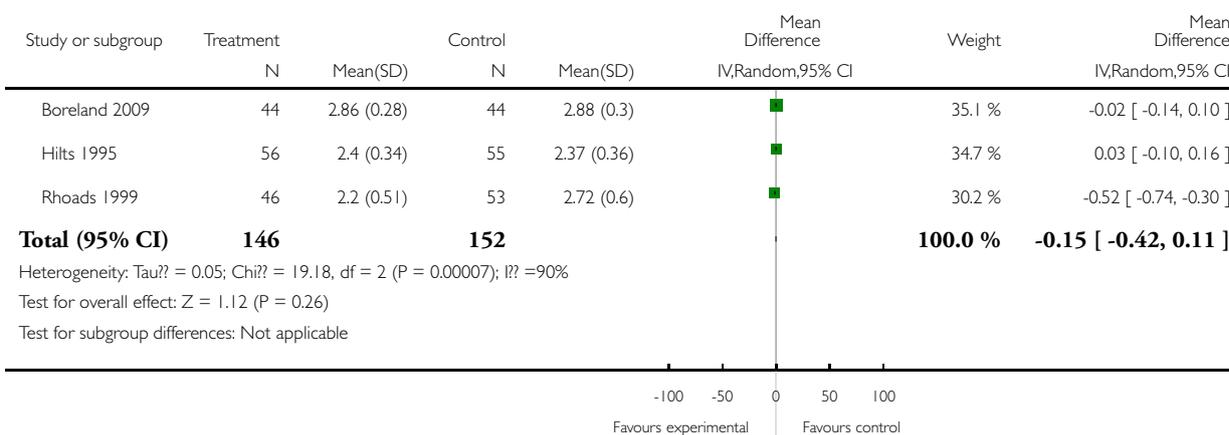


Analysis 2.1. Comparison 2 Environmental - Dust control, Outcome 1 Blood lead level (continuous).

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 1 Blood lead level (continuous)

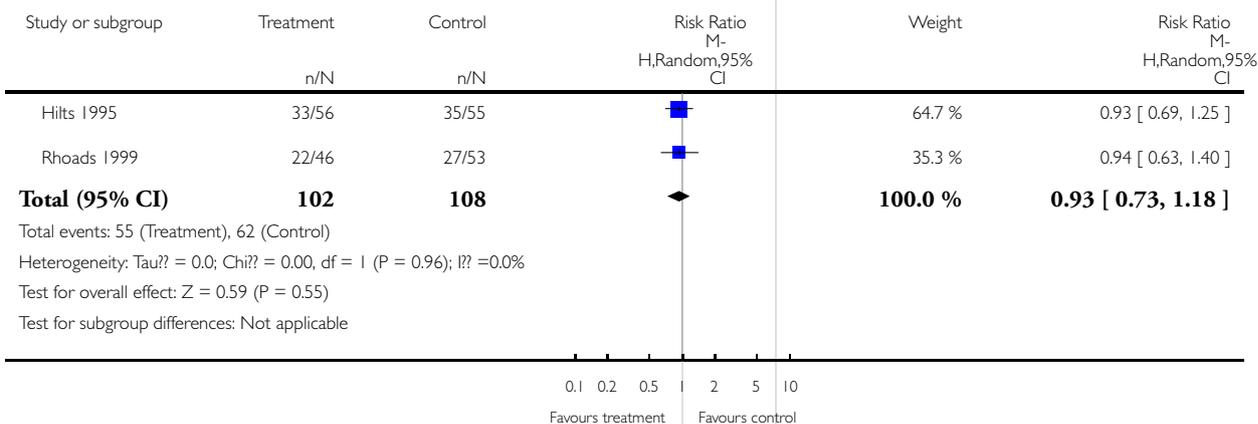


Analysis 2.2. Comparison 2 Environmental - Dust control, Outcome 2 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$).

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 2 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$)

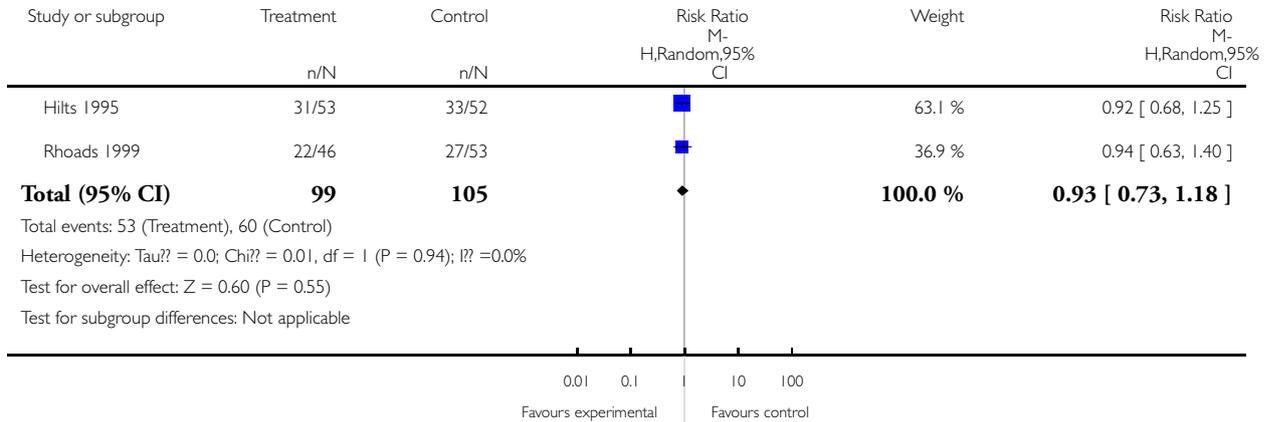


Analysis 2.3. Comparison 2 Environmental - Dust control, Outcome 3 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$) ICC 0.01.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 3 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$) ICC 0.01

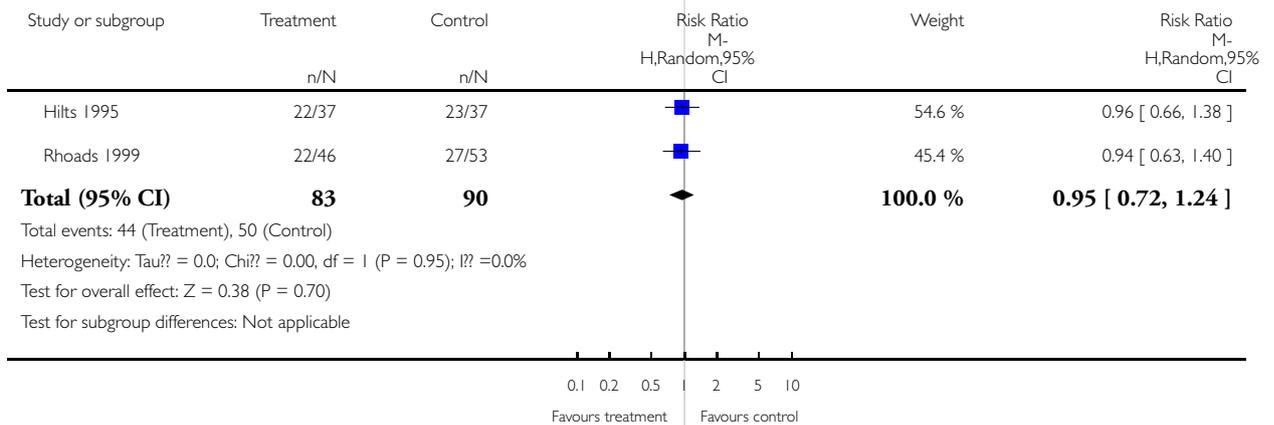


Analysis 2.4. Comparison 2 Environmental - Dust control, Outcome 4 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$) ICC 0.1.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 4 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$) ICC 0.1

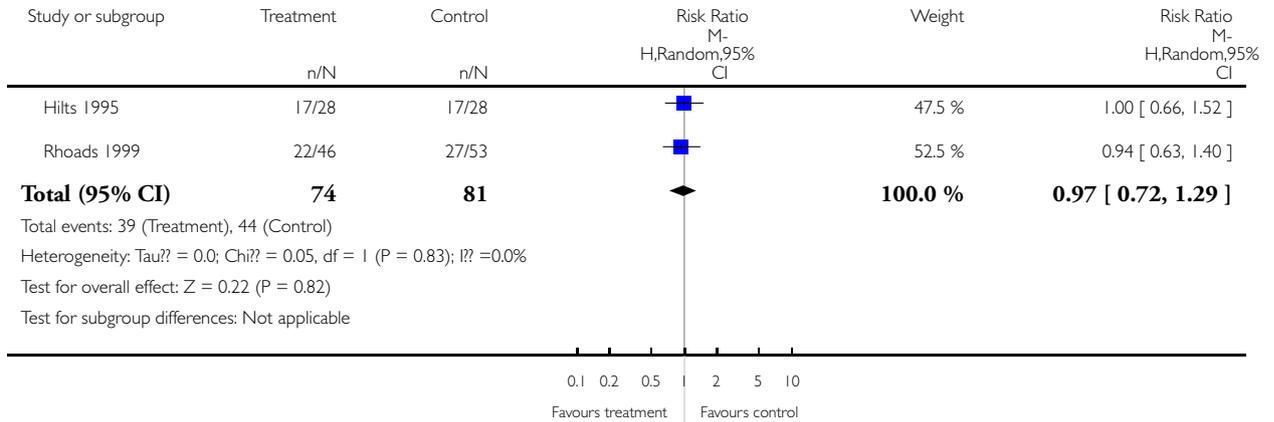


Analysis 2.5. Comparison 2 Environmental - Dust control, Outcome 5 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$) ICC 0.2.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 5 Blood lead level (dichotomous ≥ 10 $\mu\text{g/dL}$) ICC 0.2

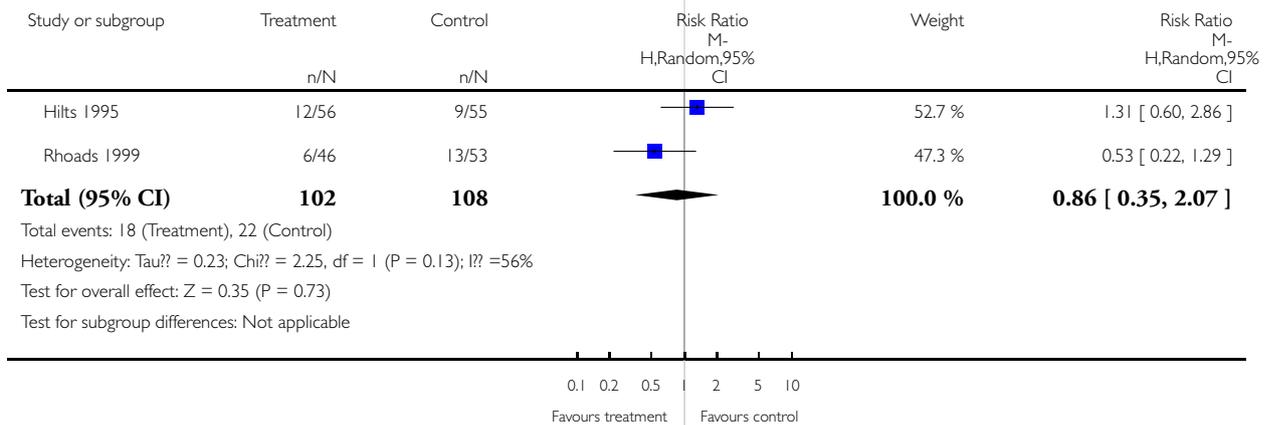


Analysis 2.6. Comparison 2 Environmental - Dust control, Outcome 6 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$).

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 6 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$)

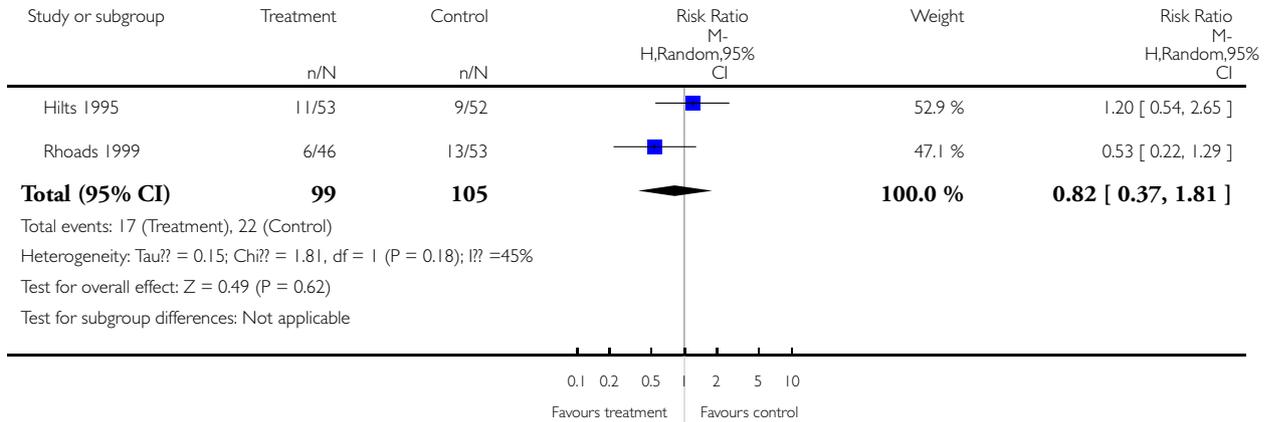


Analysis 2.7. Comparison 2 Environmental - Dust control, Outcome 7 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$) ICC 0.01.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 7 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$) ICC 0.01

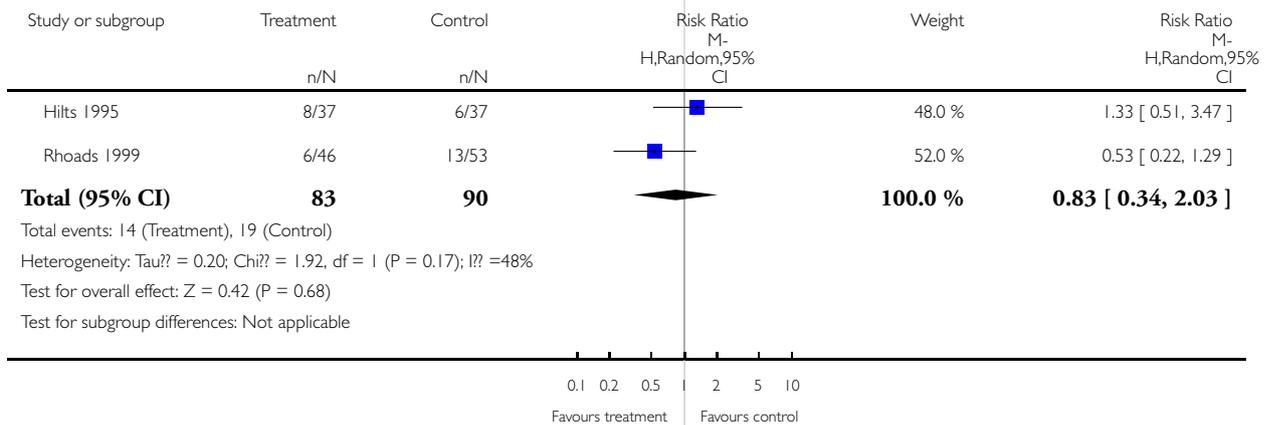


Analysis 2.8. Comparison 2 Environmental - Dust control, Outcome 8 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$) ICC 0.1.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 8 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$) ICC 0.1

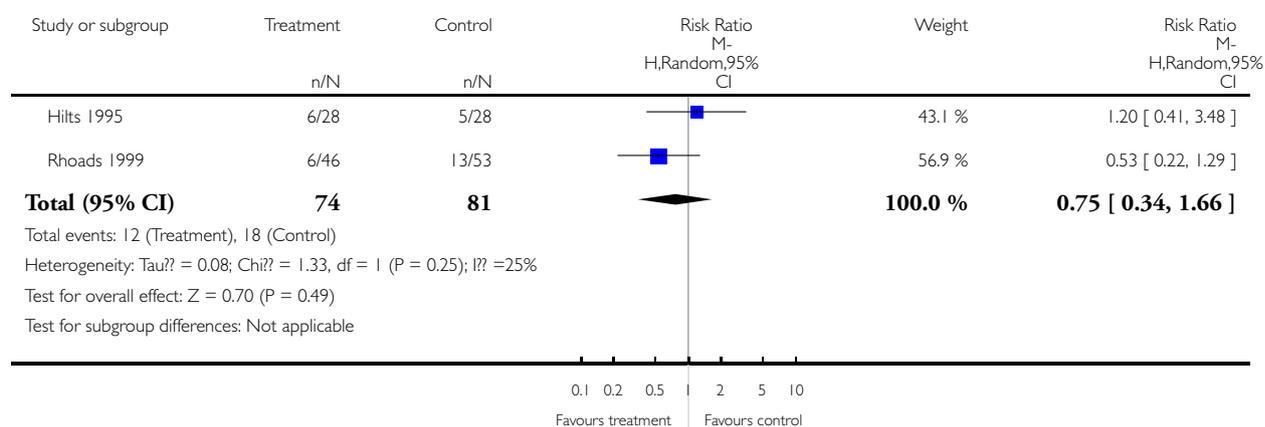


Analysis 2.9. Comparison 2 Environmental - Dust control, Outcome 9 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$) ICC 0.2.

Review: Household interventions for preventing domestic lead exposure in children

Comparison: 2 Environmental - Dust control

Outcome: 9 Blood lead level (dichotomous ≥ 15 $\mu\text{g/dL}$) ICC 0.2



ADDITIONAL TABLES

Table 1. Mean blood lead level and age at baseline

Study ID	As-chen-grau 1998	Bore-land 2009	Brown 2006	Camp-bell 2011	Char-ney 1983	Far-rell 1998	Hilts 1995	Jor-dan 2003	Lan-phear 1996	Lan-phear 1999	Rhoads 1999	Ster-ling 2004	Wasser-man 2002	Weitz-man 1993
Mean blood lead level at baseline ($\mu\text{g/dL}$)	15-19	15-19	15-19	2.6-2.7	>20	10-14	10-14	<10	<10	<10	10-14	10-14	<10	10-14
Mean age at baseline (months)	24-36	> 36	12-24	8-14	> 36	6-72	24-36	<12	12-24	<12	12-24	> 36	12-24	24-36

Table 2. Intervention type by study

Study ID	Education	Dust control	Soil abatement	Combination
Aschengrau 1998				Yes
Boreland 2009		Yes		
Brown 2006	Yes			
Campbell 2011				Yes
Charney 1983				Yes
Farrell 1998			Yes	
Hilts 1995		Yes		
Jordan 2003	Yes			
Lanphear 1996	Yes			
Lanphear 1999	Yes			
Rhoads 1999		Yes		
Sterling 2004				Yes
Wasserman 2002	Yes			
Weitzman 1993			Yes	

Table 3. Outcome measures by study

Study ID	Blood lead - continuous	Blood lead - dichotomous	Hard floor lead	Carpet lead	Other
Aschengrau 1998	Yes		Yes		
Boreland 2009	Yes				
Brown 2006	Yes	Yes			Parent-Child Interaction scale
Campbell 2011	Yes		Yes		Chicago Parents Knowledge Test
Charney 1983	Yes	Yes			
Farrell 1998					Total effect (blood lead levels)

Table 3. Outcome measures by study (Continued)

Hilts 1995	Yes	Yes		Yes	
Jordan 2003	Yes				
Lanphear 1996	Yes	Yes	Yes	Yes	
Lanphear 1999	Yes	Yes	Yes	Yes	
Rhoads 1999	Yes	Yes			Maternal knowledge lead poisoning
Sterling 2004		Yes			
Wasserman 2002	Yes	Yes			Chicago Parents Knowledge Test
Weitzman 1993	Yes				

APPENDICES

Appendix I. Search strategies used in previous version of the review

MEDLINE search strategy

1. Lead Poisoning, Nervous System, Childhood/ or Lead Poisoning/ or Lead/ or lead.mp. or Lead Poisoning, Nervous System/ or Lead Radioisotopes/
2. lead poisoning.mp.
3. lead exposure.mp.
4. lead blood level.mp.
5. lead reduction.mp.
6. 1 or 2 or 3 or 4 or 5
7. randomized controlled trial.pt.
8. controlled clinical trial.pt.
9. randomized controlled trials/
10. random allocation/
11. double blind method/
12. single blind method/
13. or/7-12
14. animal/ not (animal/ and human/)
15. 13 not 14
16. clinical trial.pt.
17. exp clinical trials/
18. (clinic\$ adj25 trial\$).ti,ab.
19. cross-over studies/

20. (crossover or cross-over or cross over).tw.
21. ((singl\$ or doubl\$ or trebl\$ or tripl\$) adj25 (blind\$ or mask\$)).ti,ab.
22. placebos/
23. placebo\$.ti,ab.
24. random\$.ti,ab.
25. research design/
26. or/16-25
27. 26 not 14
28. 15 or 27
29. adolescent/ or child/ or infant.mp. [mp=title, original title, abstract, name of substance word, subject heading word]
30. (child\$ or boy\$ or girl\$ or baby or babies or infant\$ or toddler\$ or teen\$ or adolescen\$).tw.
31. 29 or 30
32. 28 and 31
33. limit 28 to (“newborn infant (birth to 1 month)” or “infant (1 to 23 months)” or “preschool child (2 to 5 years)” or “child (6 to 12 years)” or “adolescent (13 to 18 years)”)
34. 32 or 33
35. 6 and 34

CENTRAL search strategy

- #1 LEAD POISONING NERVOUS SYSTEM CHILDHOOD
- #2 LEAD POISONING
- #3LEAD
- #4 (lead near poison*)
- #5 (#1 or #2 or #3 or #4)
- #6 CHILD
- #7 INFANT
- #8 (child* or baby or babies or infant* or preschool* or (pre next school* or boy* or girl*))
- #9 (#6 or #7 or #8)
- #10 (#5 and #9)

EMBASE search strategy

1. LEAD 203/ or LEAD 212/ or LEAD/ or LEAD POISONING/ or lead.mp. or LEAD 210/ or LEAD BLOOD LEVEL/
2. lead poisoning.mp.
3. lead exposure.mp.
4. lead reduction.mp.
5. lead control.mp.
6. 1 or 2 or 3 or 4 or 5
7. exp clinical trial/
8. comparative study/
9. drug comparison/
10. major clinical study/
11. randomization/
12. crossover procedure/
13. double blind procedure/
14. single blind procedure/
15. placebo/
16. prospective study/
17. ((clinical or controlled or comparative or placebo or prospective or randomi#ed) adj3 (trial or study)).ti,ab.
18. (random\$ adj7 (allcat\$ or allot\$ or assign\$ or basis\$ or divid\$ or order\$)).ti,ab.
19. ((singl\$ or doubl\$ or trebl\$ or tripl\$) adj7 (blind\$ or mask\$)).ti,ab.

20. (cross?over\$ or (cross adj1 over\$)).ti,ab.
21. ((allocat\$ or allot\$ or assign\$ or divid\$) adj3 (condition\$ or experiment\$ or intervention\$ or treatment\$ or therap\$ or control\$ or group\$)).ti,ab.
22. or/7-16
23. or/17-21
24. 22 or 23
25. (baby or babies).mp. [mp=title, abstract, subject headings, heading word, drug trade name, original title, device manufacturer, drug manufacturer name]
26. youth.mp.
27. child\$.mp.
28. adolescen\$.mp.
29. teenage\$.mp.
30. or/25-29
31. limit 24 to (infant or child or preschool child <1 to 6 years> or school child <7 to 12 years> or adolescent <13 to 17 years>)
32. 24 and 30
33. 31 or 32
34. 6 and 33

PsycINFO search strategy

1. exp LEAD POISONING/ or exp "LEAD (METAL)"/ or lead.mp.
2. lead poisoning.mp.
3. lead control.mp.
4. lead reduction.mp.
5. lead exposure.mp.
6. lead blood level.mp.
7. 1 or 2 or 3 or 4 or 5 or 6
8. random\$.af.
9. (random\$ adj25 (alloc\$ or assign\$ or divid\$)).mp.
10. (random\$ adj25 (trial\$ or study or studies)).mp.
11. ((control\$ or clinic\$ or prospectiv\$) adj25 (trial\$ or stud\$)).mp.
12. ((alloc\$ or assign\$ or divi\$) adj25 (condition\$ or experiment\$ or treatment\$ or control\$ or group\$)).mp.
13. ((singl\$ or doubl\$) adj (blind\$ or mask\$)).mp.
14. "CROSS?OVER".mp.
15. exp placebo/
16. (compar\$ adj25 (trial\$ or stud\$)).mp.
17. or/8-16
18. child\$.af.
19. adolesc\$.af.
20. teenage\$.af.
21. or/18-20
22. limit 17 to (100 childhood or 120 neonatal or 140 infancy or 160 preschool age or 180 school age or 200 adolescence)
23. 17 and 21
24. 22 or 23
25. 7 and 24

5 CINAHL search strategy

CINAHL searched 1982 to March 2006

1. LEAD POISONING/ or LEAD/ or lead.mp.
2. lead poisoning.mp.
3. lead reduction.mp.
4. lead control.mp.

5. lead exposure.mp.
6. lead blood level.mp.
7. 1 or 2 or 3 or 4 or 5 or 6
8. experimental studies/
9. exp clinical trials/
10. ((control\$ or clinic\$ or prospectiv\$) adj25 (trial\$ or study or studies)).tw.
11. ((allocat\$ or assign\$ or divid\$) adj25 (condition\$ or experiment\$ or treatment\$ or control\$ or group\$)).tw.
12. cross?over\$.tw.
13. placebo\$.tw.
14. (comp\$ adj25 (trial\$ or study or studies)).mp.
15. exp clinical research/
16. exp Comparative Studies/
17. exp evaluation research/
18. exp "control (research)"/
19. exp Random Assignment/
20. exp prospective studies/
21. random\$.tw.
22. or/8-21
23. child\$.tw.
24. adolescenc\$.tw.
25. teenage\$.tw.
26. exp child/
27. or/23-26
28. 22 and 27
29. limit 22 to (newborn infant or infant <1 to 23 months> or preschool child <2 to 5 years> or child <6 to 12 years> or adolescence <13 to 18 years>)
30. 28 or 29
31. 7 and 30

6 Sociofile search strategy

Sociofile searched 1963 to March 2006

Lead (KY) or lead (De) or lead poisoning (De)

7 ERIC search strategy

Lead poisoning.mp and Lead poisoning (subject heading)

8 Science Citation Index search strategy

TS=(lead same poison*) AND TS=(child* or baby or babies or infant* or preschool* or boy* or girl*)

9 ZETOC search strategy

This was searched using the term "childhood lead poisoning prevention"

10 LILACS search strategy

lead poison\$ and (child\$ or baby or babies or infant\$ or preschool\$ or girl\$ or boy\$)

11 Dissertation Abstracts search strategy

Searched using the term “childhood lead poisoning prevention”

12 Terms used for search other databases and websites

Terms used for searching ClinicalTrials.gov, Current Controlled Trials, Australian Clinical Trials Registry and National Research Register (all searched 22/03/06)

These four titles were searched using the terms “lead” and “children”.

Appendix 2. MEDLINE search strategy

- 1 Lead Poisoning, Nervous System, Childhood/ or Lead Poisoning/ or Lead/ or Lead Poisoning, Nervous System/ or Lead Radioisotopes/
- 2 lead.rn. or Pb.tw.
- 3 (lead adj5 poison\$).tw.
- 4 (lead adj5 expos\$).tw.
- 5 (lead adj5 blood\$).tw.
- 6 (lead adj5 reduc\$).tw.
- 7 (lead adj5 toxic\$).tw.
- 8 (lead adj5 environ\$).tw.
- 9 (lead adj5 hazard\$).tw.
- 10 (lead adj5 control\$).tw.
- 11 (lead adj5 (domestic\$ or home\$ or hous\$)).tw.
- 12 (lead adj5 contamin\$).tw.
- 13 (lead adj5 pollut\$).tw.
- 14 or/1-13
- 15 exp Infant/
- 16 Adolescent/
- 17 exp Child/
- 18 15 or 16 or 17
- 19 (child\$ or baby or babies or toddler\$ or boy\$ or girl\$ or preschool\$ or pre-school\$ or pre school\$ or teen\$ or adolescen\$).tw.
- 20 18 or 19
- 21 randomized controlled trial.pt.
- 22 controlled clinical trial.pt.
- 23 randomized.ab.
- 24 placebo.ab.
- 25 drug therapy.fs.
- 26 randomly.ab.
- 27 trial.ab.
- 28 groups.ab.
- 29 or/21-28
- 30 exp animals/ not humans.sh.
- 31 29 not 30
- 32 14 and 20 and 31

Appendix 3. CENTRAL search strategy

- #1 MeSH descriptor Lead, this term only
- #2 MeSH descriptor Lead Poisoning, Nervous System, Childhood, this term only
- #3 MeSH descriptor Lead Poisoning, this term only
- #4 lead near poison*
- #5 lead near expos*
- #6 lead near blood*
- #7 lead near toxic*
- #8 lead near environ*
- #9 lead near reduc*
- #10 lead near hazard*
- #11 lead near control*
- #12 lead near pollut*
- #13 lead near contamin*
- #14 lead near (domestic* or home* or hous*)
- #15 (#1or#2 OR #3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10 OR #11 OR #12 OR #13 OR #14)
- #16 MeSH descriptor Infant explode all trees
- #17 MeSH descriptor Child explode all trees
- #18 MeSH descriptor Adolescent, this term only
- #19 child* or baby or babies or toddler* or boy* or girl* or preschool* or pre-school* or (pre next school*) or teen* or adolescen*
- #20 (#16 OR #17 OR #18 OR #19)
- #21 (#15 AND #20)

Appendix 4. EMBASE search strategy

- 1 lead chloride/ or lead sulfide/ or lead 212/ or lead chromate/ or lead oxide/ or lead 210/ or lead nitrate/ or lead acetate/ or lead 203/ or lead/ or "pb".tw.
- 2 lead poisoning/
- 3 lead blood level/
- 4 (lead adj5 poison\$).tw.
- 5 (lead adj5 expos\$).tw.
- 6 (lead adj5 blood\$).tw.
- 7 (lead adj5 reduc\$).tw.
- 8 (lead adj5 toxic\$).tw.
- 9 (lead adj5 environ\$).tw.
- 10 (lead adj5 hazard\$).tw.
- 11 (lead adj5 control\$).tw.
- 12 (lead adj5 (domestic\$ or home\$ or hous\$)).tw.
- 13 (lead adj5 contamin\$).tw.
- 14 (lead adj5 pollut\$).tw.
- 15 or/1-14
- 16 exp child/
- 17 exp adolescent/
- 18 (child\$ or baby or babies or toddler\$ or boy\$ or girl\$ or preschool\$ or pre-school\$ or pre school\$ or teen\$ or adolescen\$).tw.
- 19 or/16-18)
- 20 crossover procedure/
- 21 exp double blind procedure/
- 22 single blind procedure/
- 23 random\$.tw.
- 24 randomized controlled trial/
- 25 factorial\$.tw.
- 26 (crossover\$ or cross over\$ or cross-over\$).tw.

- 27 placebo\$.tw.
- 28 doubl\$ blind\$.tw.
- 29 singl\$ blind\$.tw.
- 30 assign\$.tw.
- 31 allocat\$.tw.
- 32 volunteer\$.tw.
- 33 or/20-32
- 34 15 and 19 and 33
- 35 limit 34 to yr="2006 -Current"

Appendix 5. PsycINFO search strategy

- S34 S16 and S20 and S30
- S33 S16 and S20 and S30
- S32 S16 and S20 and S30
- S31 S16 and S20 and S30
- S30 S21 or S22 or S23 or S24 or S25 or S26 or S27 or S28 or S29
- S29 AB (placebo*) or TI (placebo*)
- S28 AB (randomly) or TI (randomly)
- S27 AB (randomi?ed) or TI (randomi?ed)
- S26 AB (trebl* blind* or trebl* mask* or tripl* blind* or tripl* mask*) or TI (trebl* blind* or trebl* mask* or tripl* blind* or tripl* mask*)
- S25 AB (double* blind* or doubl* mask*) or TI (double* blind* or doubl* mask*)
- S24 AB (singl* blind* or singl* mask*) or TI (singl* blind* or singl* mask*)
- S23 AB (clinic* trial*) or TI (clinic* trial*)
- S22 MR Quantitative Study
- S21 MR Treatment Outcome/Clinical Trial
- S20 S17 or S18 or S19
- S19 TI (baby or babies or infant* or toddler* or child* or pre school* or preschool* or pre-school* or teen* or adolescen* or boy* or girl*)
- S18 AB (baby or babies or infant* or toddler* or child* or pre school* or preschool* or pre-school* or teen* or adolescen* or boy* or girl*)
- S17 AG (100 or 120 or 140 or 160 or 180 or 200)
- S16 S1 or S2 or S3 or S4 or S5 or S6 or S7 or S8 or S9 or S10 or S11 or S12 or S13 or S14 or S15
- S15 DE "Lead (Metal)" or DE "Lead Poisoning"
- S14 AB ("Pb") or TI ("Pb")
- S13 AB (lead N5 poison*) or TI (lead N5 poison*)
- S12 AB (lead N5 pollut*) or TI (lead N5 pollut*)
- S11 AB (lead N5 contamin*) or TI (lead N5 contamin*)
- S10 AB (lead N5 hous*) or TI (lead N5 hous*)
- S9 AB (lead N5 home*) or TI (lead N5 home*)
- S8 AB (lead N5 domestic*) or TI (lead N5 domestic*)
- S7 AB (lead N5 control*) or TI (lead N5 control*)
- S6 AB (lead N5 hazard*) or TI (lead N5 hazard*)
- S5 AB (lead N5 environ*) or TI (lead N5 environ*)
- S4 AB (lead N5 toxic*) or TI (lead N5 toxic*)
- S3 AB (lead N5 reduc*) or TI (lead N5 reduc*)
- S2 AB (lead N5 blood*) or TI (lead N5 blood*)
- S1 AB (lead N5 expos*) or TI (lead N5 expos*)

Appendix 6. CINAHL search strategy

S40 S20 and S37 and S38

S39 S20 and S37 and S38

S38 S1 or S2 or S3 or S4 or S5 or S6 or S7 or S8 or S9 or S10 or S11 or S12 or S13 or S14 or S15

S37 S21 or S22 or S23 or S24 or S25 or S26 or S27 or S28 or S29 or S30 or S31 or S32 or S33 or S34 or S35 or S36

S36 AB (placebo*) or TI (placebo*)

S35 (MH "Placebos")

S34 (MH "Quantitative Studies")

S33 TI (random* N5 assign* or random* N5 allocat*)

S32 AB (random* N5 assign* or random* N5 allocat*)

S31 (MH "Random Assignment")

S30 AB (randomi?ed control* trial*) or TI (randomi?ed control* trial*)

S29 TI (trebl* blind* or trebl* mask* or tripl* blind* or tripl* mask*)

S28 AB (trebl* blind* or trebl* mask* or tripl* blind* or tripl* mask*)

S27 TI (double* blind* or doubl* mask*)

S26 AB (double* blind* or doubl* mask*)

S25 TI (singl* blind* or singl* mask*)

S24 AB (singl* blind* or singl* mask*)

S23 AB (clinic* trial*) or TI (clinic* trial*)

S22 PT clinical trial

S21 (MH "Clinical Trials+")

S20 S16 or S17 or S18 or S19

S19 (MH "Adolescence+")

S18 (MH "Child+")

S17 TI (baby or babies or infant* or toddler* or child* or pre school* or preschool* or pre-school* or teen* or adolescen* or boy* or girl*)

S16 AB (baby or babies or infant* or toddler* or child* or pre school* or preschool* or pre-school* or teen* or adolescen* or boy* or girl*)

S15 AB ("Pb") or TI ("Pb")

S14 AB (lead N5 pollut*) or TI (lead N5 pollut*)

S13 AB (lead N5 contamin*) or TI (lead N5 contamin*)

S12 AB (lead N5 hous*) or TI (lead N5 hous*)

S11 AB (lead N5 home*) or TI (lead N5 home*)

S10 AB (lead N5 domestic*) or TI (lead N5 domestic*)

S9 AB (lead N5 control*) or TI (lead N5 control*)

S8 AB (lead N5 hazard*) or TI (lead N5 hazard*)

S7 AB (lead N5 environ*) or TI (lead N5 environ*)

S6 AB (lead N5 toxic*) or TI (lead N5 toxic*)

S5 AB (lead N5 reduc*) or TI (lead N5 reduc*)

S4 AB (lead N5 blood*) or TI (lead N5 blood*)

S3 AB (lead N5 expos*) or TI (lead N5 expos*)

S2 AB (lead N5 poison*) or TI (lead N5 poison*)

S1 (MH "Lead") or (MH "Lead Poisoning")

Appendix 7. Sociological Abstracts search strategy

((DE="lead poisoning") or(KW=(lead within 5 poison*) or (lead within 5 expos*) or (lead within 5 blood*) or (lead within 5 reduc*) or (lead within 5 toxic*) or (lead within 5 environ*) or (lead within 5 hazard*) or (lead within 5 control*) or (lead within 5 domestic*) or (lead within 5 home*) or (lead within 5 house*) or (lead within 5 contamin*) or (lead within 5 pollut*) or "pb")))

AND

((DE=("children" or "adolescents" or "infants")) or(KW=(baby or babies or infant* or child* or toddler* or pre-school* or "pre school*" or pre-school* or boy* or girl* or teen* or adolescen*)))

Appendix 8. ERIC search strategy

((DE="lead poisoning") or(KW=(lead within 5 poison*)or (lead within 5 expos*) or (lead within 5 blood*)or (lead within 5 reduc*)or (lead within 5 toxic*)or (lead within 5 environ*) or (lead within 5 hazard*)or(lead within 5 control*)or (lead within 5 domestic*)or (lead within 5 home*) or (lead within 5 hous*) or (lead within 5 contamin*) or (lead within 5 pollut)or ("Pb")))

AND

((DE=("adolescents" or "children" or "infants" or "toddlers" or "young children")) Or (KW= (baby or babies or infant* or toddler* or child* or preschool* or pre-school* or pre school* or teen* or adolescen* or boy* or girl*)))

Appendix 9. Science Citation Index search strategy

#5 #4 AND #3

#4 TS=(baby or babies or infant* or child* or toddler* or boy* or girl* or preschool* or preschool* or teen* or adolescen*)

#3 #2 OR #1

#2 TS= ("Pb" same (poison* or expos* or blood* or reduc* or toxic* or environ* or hazard* or control* or pollut* or contamin* or domestic* or home* or hous*))

#1 TS= (lead same (poison* or expos* or blood* or reduc* or toxic* or environ* or hazard* or control* or pollut* or contamin* or domestic* or home* or hous*))

Appendix 10. ZETOC search strategy

Lead and child*

Appendix 11. LILACS search strategy

Mh lead or Mh lead poison\$ or TW lead and poison\$ or TW lead and expos\$ or TW lead and toxic\$ or TW lead and contamin\$ or TW lead and blood\$ or tw lead and reduc\$ or TW lead and control\$ or TW lead and pollut\$ or TW lead and hazard\$ or TW lead and hous\$ or TW lead and home\$ or TW lead and domestic\$ or TW lead and environ\$ or tw Pb [Words] and (Mh infant or Mh child or Mh child,preschool or Mh adolescent or tw baby or tw babies or tw infant\$ or tw child\$ or tw preschool or tw pre-school or Tw adolescen\$ or Tw teen\$) AND (PD 2006 or PD 2007 or PD 2008 or PD 2009 or PD 2010) [Words] and ((Pt randomized controlled trial OR Pt controlled clinical trial OR Mh randomized controlled trials OR Mh random allocation OR Mh double-blind method OR Mh single-blind method) AND NOT (Ct animal AND NOT (Ct human and Ct animal)) OR (Pt clinical trial OR Ex E05.318.760.535\$ OR (Tw clin\$ AND (Tw trial\$ OR Tw ensa\$ OR Tw estud\$ OR Tw experim\$ OR Tw investiga\$)) OR ((Tw singl\$ OR Tw simple\$ OR Tw doubl\$ OR Tw doble\$ OR Tw duplo\$ OR Tw trebl\$ OR Tw trip\$) AND (Tw blind\$ OR Tw cego\$ OR Tw ciego\$ OR Tw mask\$ OR Tw mascar\$)) OR Mh placebos OR Tw placebo\$ OR (Tw random\$ OR Tw randon\$ OR Tw casual\$ OR

Tw acaso\$ OR Tw azar OR Tw aleator\$) OR Mh research design) AND NOT (Ct animal AND NOT (Ct human and Ct animal)) OR (Ct comparative study OR Ex E05.337\$ OR Mh follow-up studies OR Mh prospective studies OR Tw control\$ OR Tw prospectiv\$ OR Tw volunt\$ OR Tw volunteer\$) AND NOT (Ct animal AND NOT (Ct human and Ct animal))) [Words]

Appendix 12. Dissertation Abstracts search strategy

This was searched using the term “childhood lead poisoning prevention”

Appendix 13. Terms used for search other databases and websites

Terms used for searching ClinicalTrials.gov, Current Controlled Trials, Australian New Zealand Clinical Trials Registry and National Research Register Archive
“lead” AND “children”.

WHAT'S NEW

Last assessed as up-to-date: 7 March 2012.

Date	Event	Description
17 February 2012	New citation required but conclusions have not changed	Two new included studies. New risk of bias tables. New summary of findings table
20 January 2012	New search has been performed	New search.

HISTORY

Protocol first published: Issue 2, 2006

Review first published: Issue 2, 2008

Date	Event	Description
9 November 2008	Amended	Converted to new review format.

CONTRIBUTIONS OF AUTHORS

BY, SW, GR, NL and BL developed and wrote the text of this review. The search strategy was developed in concert with Jo Abbott and Margaret Anderson, the former and current Trials Search Coordinator of the Cochrane Developmental, Psychosocial and Learning Problems Group.

DECLARATIONS OF INTEREST

Berlinda Yeoh - none known

Susan Woolfenden - none known

Bruce Lanphear - was an investigator in two trials included in this review ([Lanphear 1996a](#); [Lanphear 1999](#))

Greta F Ridley - none known

Nuala Livingstone - none known

SOURCES OF SUPPORT

Internal sources

- No sources of support supplied

External sources

- Financial Markets Foundation for Children, Australia.
- Commonwealth Government Department of Health, Australia.

INDEX TERMS

Medical Subject Headings (MeSH)

*Lead [blood]; Dust [*prevention & control]; Environmental Exposure [adverse effects; *prevention & control]; Environmental Remediation [*methods]; Lead Poisoning [*prevention & control]; Paint [toxicity]; Soil

MeSH check words

Child; Humans