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

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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, first published in 2008.

Objectives

To assess the effects of household interventions for preventing or reducing lead exposure in children, as measured by improvements in cognitive and neurobehavioural development, reductions in blood lead levels and reductions in household dust lead levels.

Search methods

In May 2016 we searched CENTRAL, Ovid MEDLINE, Embase, nine other databases and two trials registers: the World Health Organization International Clinical Trials Registry Platform (WHO ICTRP) and ClinicalTrials.gov. We also checked the reference lists of relevant studies and contacted experts to find unpublished studies.

Selection criteria

Randomised controlled trials (RCTs) and quasi-RCTs of household educational or environmental interventions, or combinations of interventions to prevent lead exposure in children (from birth to 18 years of age), where investigators reported at least one standardised outcome measure.

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. We assessed the quality of the evidence using the GRADE approach.

Main results

We included 14 studies involving 2643 children: 13 RCTs (involving 2565 children) and one quasi-RCT (involving 78 children). Children in all studies were under six years of age. Thirteen studies took place in urban areas of North America, and one was in Australia. Most studies were in areas with low socioeconomic status. Girls and boys were equally represented in all studies. The duration of the intervention ranged from 3 months to 24 months in 12 studies, while 2 studies performed interventions on a single occasion. Follow-up periods ranged from 6 months to 48 months. Three RCTs were at low risk of bias in all assessed domains. We rated two RCTs and one quasi-RCT as being at high risk of selection bias and six RCTs as being at high risk of attrition bias. For educational interventions, we rated the quality of evidence to be high for continuous blood lead levels and moderate for all other outcomes. For environmental interventions, we assessed the quality of evidence as moderate to low. National or international research grants or governments funded 12 studies, while the other 2 did not report their funding sources.

No studies reported on cognitive or neurobehavioural outcomes. No studies reported on adverse events in children. All studies reported blood lead level outcomes.

We put studies into subgroups according to their intervention type. We performed meta-analyses 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%; 5 studies; N = 815; high quality evidence (log transformed); dichotomous ≥ 10.0 µg/dL (≥ 0.48 µmol/L): risk ratio (RR) 1.02, 95% CI 0.79 to 1.30; I^2 = 0%; 4 studies; N = 520; moderate quality evidence; dichotomous ≥ 15.0 µg/dL (≥ 0.72 µmol/L): RR 0.60, 95% CI 0.33 to 1.09; I^2 = 0%; 4 studies; N = 520; moderate quality evidence). Meta-analysis for the dust control subgroup also found no evidence of effectiveness on blood lead levels (continuous: MD −0.15, 95% CI −0.42 to 0.11; I^2 = 90%; 3 studies; N = 298; low quality evidence (log transformed); dichotomous ≥ 10.0 µg/dL (≥ 0.48 µmol/L): RR 0.93, 95% CI 0.73 to 1.18; I^2 = 0; 2 studies; N = 210; moderate quality evidence; dichotomous ≥ 15.0 µg/dL (≥ 0.72 µmol/L): RR 0.86, 95% CI 0.35 to 2.07; I^2 = 56%; 2 studies; N = 210; low quality evidence). After adjusting the dust control subgroup for clustering in meta-analysis, we found no evidence of effectiveness. We could not pool the studies using soil abatement (removal and replacement) and combination intervention groups in a meta-analysis due to substantial differences between studies, and generalisability or reproducibility of the results from these studies is unknown. Therefore, there is currently insufficient evidence to clarify whether soil abatement or a combination of interventions reduces blood lead levels.

Authors' conclusions

Based on current knowledge, household educational interventions are ineffective in reducing blood lead levels in children as a population health measure. Dust control interventions may lead to little or no difference in blood lead levels (the quality of evidence was moderate to low, meaning that future research is likely to change these results). There is currently insufficient evidence to draw conclusions about the effectiveness of soil abatement or combination interventions. No study reported on cognitive or neurobehavioural outcomes or adverse events. These patient-relevant outcomes would have been of great interest to draw conclusions for practice.

Further trials are required to establish the most effective intervention for preventing 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 low- and middle-income countries and in differing socioeconomic groups in high-income countries.

PLAIN LANGUAGE SUMMARY

Household interventions for preventing domestic lead exposure in children

Why is this review important?

Lead poisoning at high levels can cause anaemia, multi-organ damage, seizures, coma and death in children. At chronic low levels it can lead to cognitive, psychological and neurobehavioural impairment. Researchers have studied many different educational and environmental household interventions to prevent lead exposure in children, such as parental education, removal of lead dust or home remediation work. However, it is not clear if and to what extent these interventions work in preventing lead exposure in children.

Who will be interested in this review?

- Parents and caregivers who want to prevent domestic lead exposure in children.

- Health professionals and decision-makers who are interested in methods to prevent domestic lead exposure in children.

What questions does this review aim to answer?

We wanted to find out if educational or environmental household interventions, or combinations of both, are effective in preventing or reducing domestic lead exposure in children up to 18 years of age. We were interested in looking at improvements in cognitive and neurobehavioural development, reductions in blood lead levels and household lead dust levels.

Which studies were included in the review?

We searched databases up to May 2016 for randomised controlled trials, or RCTs (where participants are randomly assigned to treatment and control groups, in this case with one group that does not receive any intervention and one or more other groups that do) and quasi-RCTs (where children are assigned to groups using methods that are not strictly random). We found 14 studies involving 2643 children that investigated educational or environmental interventions, or a combination of both, to reduce domestic lead exposure in children. Children in all studies were under six years of age. Thirteen studies took place in urban areas of North America, and one was in Australia. Most studies were performed in areas with low socioeconomic status. Boys and girls were equally represented in all studies. The duration of the intervention ranged from 3 months to 24 months in 12 studies, and 2 studies performed an intervention on a single occasion. Follow-up periods ranged from 6 months to 48 months. National or international research grants or governments funded 12 studies, and 2 studies did not report their funding sources.

What does the evidence from the review reveal?

We did not find any studies that evaluated effects on cognitive or neurobehavioural outcomes or adverse events in children. All studies reported on blood lead levels. The included studies found that educational interventions are not effective in reducing blood lead levels of young children; the quality of this evidence was moderate to high. Dust control interventions may lead to little or no difference in blood lead levels; however, future research might change these results because the quality of evidence was moderate to low for these interventions. There is currently insufficient evidence that soil abatement or combination interventions reduce blood lead levels, and further studies need to address this.

What should happen next?

More research is needed to find out what is effective for preventing children's exposure to lead. Studies should be carried out in different socioeconomic groups in high-, middle- and low-income countries to consider how interventions work in contexts shaped by different levels of industrialisation or environmental and occupational health safety regulations.

Summary of findings for the main comparison. Education strategies versus no intervention for preventing domestic lead exposure in children

Education strategies versus no intervention for preventing domestic lead exposure in children

Patient or population: children
Settings: households in the USA
Intervention: education strategies for prevention of domestic lead exposure
Comparison: no intervention

Outcomes	Illustrative comparative risks* (95% CI)		Relative effect (95% CI)	Number of participants (studies)	Quality of the evidence (GRADE)	Comments
	Assumed risk	Corresponding risk				
	Regular environment	Educational strategies				
Cognitive and neurobehavioural outcomes	None of the included studies assessed effects on cognitive or neurobehavioural outcomes		—	—	—	—
Adverse events	None of the included studies assessed adverse event outcomes		—	—	—	—
Blood lead level (continuous) Blood lead levels after intervention **Scale**: 0-30 **Follow-up**: 6-18 months	The mean blood lead level (continuous) ranged across control groups from **1.24 to 2.13** [a,b]	The mean blood lead level (continuous) in the intervention groups was **0.02 higher** (0.09 lower to 0.12 higher)[a,b]	—	815 (5 studies)	⊕⊕⊕⊕ **High** [c]	Included studies: Lanphear 1996a; Lanphear 1999; Wasserman 2002; Jordan 2003; Brown 2006
Blood lead level ≥ 10.0 µg/dL (dichotomous) Blood lead level **Follow-up**: 6-18 months	**Medium risk population** [d]		**RR 1.02** (0.79 to 1.30)	520 (4 studies)	⊕⊕⊕⊖ **Moderate** [e]	Included studies: Lanphear 1996a; Lanphear 1999; Wasserman 2002; Brown 2006
	238 per 1000	**243 per 1000** (188 to 309)				
Blood lead level ≥ 15.0 µg/dL (dichotomous) Blood lead level **Follow-up**: 6-18 months	**Medium risk population** [d]		**RR 0.60** (0.33 to 1.09)	520 (4 studies)	⊕⊕⊕⊖ **Moderate** [e]	Included studies: Lanphear 1996a; Lanphear 1999; Wasserman 2002; Brown 2006
	110 per 1000	**66 per 1000** (36 to 120)				
Floor dust - hard floor (continuous) Floor dust lead levels	The mean floor dust level - hard floor - ranged across control groups from **1.65 to 2.28** [b]	The mean floor dust level - hard floor - in the intervention groups was **0.07**	—	318 (2 studies)	⊕⊕⊕⊖ **Moderate** [f]	Included studies: Lanphear 1996a; Lanphear 1999

Scale: 0-40 **Follow-up**: 6-18 months		**lower** (0.37 lower to 0.24 higher)[b]

*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% CI) 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.

[a]Change in blood lead level.
[b]These are logged values.
[c]Although two of five studies had high attrition rates, we did not downgrade for high risk of bias because a sensitivity analysis excluding them showed no relevant difference in the result, and we assessed all other risk of bias domains in all five included studies as being at low risk.
[d]Baseline based on median of control groups.
[e]We downgraded by one level because of imprecision: 95% CI around pooled estimate includes no effect and appreciable harm or benefit and because the total number of events is less than 300.
[f]We downgraded by one level because of imprecision: total population is less than 400.

Summary of findings 2. Environmental strategies (dust control) versus no intervention for preventing domestic lead exposure in children

Environmental strategies (dust control) versus no intervention for preventing domestic lead exposure in children

Patient or population: children
Settings: households in Australia, Canada, USA
Intervention: environmental strategies (dust control)
Comparison: regular environment

Outcomes	**Illustrative comparative risks* (95% CI)**		**Relative effect (95% CI)**	**Number of participants (studies)**	**Quality of the evidence (GRADE)**	**Comments**
	Assumed risk	**Corresponding risk**				
	Control	**Environmental strategies (dust control)**				
Cognitive and neurobehavioural outcomes	None of the included studies assessed effects on cognitive or neurobehavioural outcomes		—	—	—	—
Adverse events	None of the included studies assessed adverse event outcomes		—	—	—	—

Blood lead level (continuous) Blood lead level at end of duration **Scale**: 0-30 **Follow-up**: 6-18 months	The mean blood lead level (continuous) ranged across control groups from **2.4 to 2.9** [a]	The mean blood lead level (continuous) in the intervention groups was **0.15 lower** (0.42 lower to 0.11 higher) [a]	—	298 (3 studies)	⊕⊕⊖⊖ **Low** [b]	Included studies: Hilts 1995; Rhoads 1999; Boreland 2009
Blood lead level ≥ 10.0 µg/dL (dichotomous) Blood lead level **Follow-up**: 6-18 months	**Medium risk population** [c]		**RR 0.93** (0.73 to 1.18)	210 (2 studies)	⊕⊕⊕⊖ **Moderate** [d]	Included studies: Hilts 1995; Rhoads 1999
	573 per 1000	**533 per 1000** (418 to 676)				
Blood lead level ≥ 15.0 µg/dL (dichotomous) Blood lead level **Follow-up**: 6-18 months	**Medium risk population** [c]		**RR 0.86** (0.35 to 2.07)	210 (2 studies)	⊕⊕⊖⊖ **Low** [e]	Included studies: Hilts 1995; Rhoads 1999
	205 per 1000	**176 per 1000** (72 to 424)				

*The basis for the **assumed risk** (for example, 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.

[a] Change in blood lead level and logged values
[b] We downgraded by two levels, one because of imprecision: total population size less than 400; one because of inconsistency: $I^2 = 90\%$.
[c] Baseline based on median of control groups.
[d] We downgraded by one level because of imprecision: total number of events less than 300.
[e] We downgraded by two levels, one because of imprecision: 95% CI around pooled estimate includes no effect and appreciable harm or benefit and total number of events less than 300; one because of inconsistency: $I^2 = 56\%$.

BACKGROUND

Description of the condition

Lead is a metal that has been used since prehistoric times. Over the years, its expansive distribution and mobilisation in the environment has resulted in increasing human exposure and uptake (Tong 2000). Lead poisoning is a serious and recognised health hazard with major socioeconomic implications (UNEP-UNICEF 1997; Attina 2013). At high levels, lead poisoning in children can cause anaemia, multi-organ damage, renal damage, seizures, coma and death. At chronic low levels, lead toxicity causes significant cognitive, psychological and neurobehavioural impairment (UNEP-UNICEF 1997; Tong 2000; Mason 2014).

Lead has been shown to account for 0.6% of the total global disease burden (Lim 2012). The burden of disease associated with lead exposure in Europe amounts to at least 1,053,000 disability adjusted life years (DALYs) per year, of which approximately 66% are due to housing-based exposures (Braubach 2011). There are many potential sources of lead in the environment, including lead industries, mining and smelting; leaded petrol; lead-based paint; water piping, fixtures and solder; and 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; residence in an area with lead industry; renovation or deterioration of older houses containing lead-based paint; and residence in countries where leaded petrol or aviation fuel is still used (Tong 2000; Miranda 2011).

Blood lead levels in the general population of developed countries have fallen significantly over the past 20 years with the phasing out of lead petrol and bans on the use of lead in paints and lead solder used in canned foods and other consumer products (Jacobs 2006). However, 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. Older housing with peeling or flaking paint or current renovations can result in increased lead dust levels (EHU 2002).

Occupational and environmental exposures continue to be a serious global problem, especially in low- and middle-income countries, which may be rapidly industrialising (Tong 2000). People in these settings, especially children, may have higher levels of lead exposure due to unregulated industrial emission, weak environmental and occupational health safety regulations, and cottage (domestic) industries such as metal polishing and smelting (UNEP-UNICEF 1997). Many countries have implemented or proposed legally binding restrictions on lead in paints for domestic use, and consequently this will become a less important source of exposure over time. Nevertheless, lead-based paints for household use are still available for purchase in several low- and middle-income countries such as Argentina, China, Ethiopia, Ghana, India, Malaysia and Tunisia (EHU 2002; Clark 2005; Adebanowo 2007; UNEP 2013). In view of rapid industrialisation and the persistence of lead in the environment, this is likely to remain a significant public health issue in these countries for many years (Tong 2000).

Children are at higher risk of lead toxicity. This is due to their increased intake of lead per unit of body weight compared with adults and their higher rate of physiological uptake (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 (Bellinger 2008; Mason 2014).

Dust is an important residential media for lead exposure and the strongest predictor of blood lead levels. Floor dust exceeding 0.431 mg/m^2 (40.0 µg/ft^2) is currently recognised as hazardous (Dixon 2009).

In 1991, the Centers for Disease Control and Prevention (CDC) defined blood lead levels of 10.0 µg/dL or more as a "blood lead level of concern" for children aged one to five years (CDC 1991). Recent studies, however, show that adverse effects on cognitive function in children are proportional at even lower blood lead levels (Canfield 2003; Lanphear 2005a; Kordas 2006; Evens 2015), suggesting that there is no safe level of blood lead for children (CDC 2005; Grandjean 2010; CDC 2012). Therefore, in 2012 the CDC followed the advice of the Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) to replace the term 'level of concern' with an upper reference interval value, which they defined as the 97.5th percentile of blood lead levels (currently 5.0 µg/dL) from the National Health and Nutrition Examination Survey (NHANES), which includes US children aged one to five years (Wheeler 2013).

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 in children with blood lead concentrations greater than 45.0 µg/dL, reduce the mortality of severe acute lead encephalopathy, but they do little to remove the lead sequestered in bone (> 94% of the body burden in adults, 70% of the body burden in children (O'Flaherty 1995)), 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 months to 11 months with acute exposure and 20 months to 38 months with prolonged exposure) than that of adults (15 days), and bone can be a prolonged source of lead in the blood (Manton 2000; Chisolm 2001).

Chisolm 2001 estimated that the cost of chelation therapy in children who were previously exposed to lead is higher than environmental interventions and is not likely to have significant long-term benefit. The ultimate goal for the management of this public health issue, then, should be to prevent toxicity in the first place by controlling lead hazards in the environment (Chisolm 2001).

Description of the intervention

This review focuses on interventions for secondary prevention in children that are already exposed to lead sources. It includes interventions that aim to reduce existing lead exposure or prevent further lead exposure. Most research has focused on environmental and educational preventive interventions. Educational interventions address parental awareness by imparting knowledge 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 educational programmes. 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). Different randomised controlled trials (RCTs) have tested a variety of environmental lead hazard control interventions to decrease children's blood lead level and home dust lead levels, with most follow-up extending from six months to two years postintervention. Comparison of environmental interventions has been difficult due to variations in intervention types, blood collection techniques, adjustments for age and season, dust lead loading quantification and statistical analyses (Campbell 2000).

How the intervention might work

Removal of sources of lead, specialised cleanings, repairs and maintenance around the house (environmental interventions) aim to reduce exposure to domestic lead and lead dust. Educational interventions focused on parents aim to raise parental awareness of lead hazards and motivate them to reduce lead hazards for their children. Through education, parents should also learn about lead exposure pathways and how to clean their home to keep it in lead-safe condition. Enabling parents by educational means aims to reduce exposure to domestic lead and lead dust, thereby decreasing the risk of lead ingestion and ultimately lead poisoning.

Why it is important to do this review

Lead poisoning has long been linked with physical, cognitive and neurobehavioural impairment in children. Despite efforts to reduce environmental, occupational and industrial lead exposure worldwide, children living in areas with older housing and in low- and middle-income countries with weak industrial regulations continue to show evidence of lead exposure. There has been research on many household interventions, and it is important to examine their effectiveness.

This is an update of an original review by 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. The original review concluded there was insufficient evidence for soil abatement or combination interventions, and that further trials were required to establish the most effective intervention for the prevention of lead exposure. Hence, it is important to update this review looking for any advances in the area.

OBJECTIVES

To assess the effects of household interventions for preventing or reducing lead exposure in children, as measured by improvements in cognitive and neurobehavioural development, reductions in blood lead levels and reductions in household dust lead levels.

METHODS

Criteria for considering studies for this review

Types of studies

All RCTs or quasi-RCTs (which use 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).

The main reason to focus on such study designs is to 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. Furthermore, children's blood lead levels, which peak at about two years of age, typically decline as they mature primarily because they no longer exhibit as many mouthing behaviours. Thus, any observational study that enrolls children aged 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 overestimate the effect of an intervention.

Types of participants

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

Types of interventions

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

1. Educational interventions. One or more educational sessions for parents that aim to raise parental awareness of lead exposure pathways and the dangers lead can have on their children as well as teaching them how to keep their home in lead-safe condition and how to prevent ingestion of dust and soil. Eligible educational interventions had to provide more than standard information via, for example, a brochure.
2. Environmental (household) interventions. These include specialised cleaning, repairs, maintenance, soil abatement (removal and replacement), painting, and temporary containment of lead hazards.
3. Combinations of the above interventions.

We excluded interventions involving nutritional supplementation.

Types of outcome measures

We included the outcomes described below in this review.

Primary outcomes

1. Cognitive and neurobehavioural outcomes in children, assessed with standardised measures of outcome such as assessment of a child's intelligence quotient (IQ), development or behaviour. Suitable IQ measures were the Stanford Binet Intelligence Scale (Smith 1989), the Wechsler Intelligence Scale for Children (WISC) (Wechsler 1991), and the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) (Wechsler 1989). An example of a suitable

development measure is the Griffiths Mental Development Scales (Griffiths 1954; Griffiths 1970), and for behaviour, the Child Behaviour Checklist (Achenbach 1991).

2. Adverse events of the intervention in children (e.g. injuries or poisoning through cleansing agents).

Secondary outcomes

1. Blood lead levels in children (venous blood sample or capillary blood sample; AAP 1998).
2. Household dust measures of lead exposure (e.g. lead loading of household floor dust).
3. Cost of intervention (e.g. cost of cleaning supplies, soil abatement or education).

Instruments were confined to those with at least one standardised outcome measure (such as blood lead level) used for intervention and control groups. We considered outcomes for any follow-up duration period (short term: 6 months to 18 months; long-term: longer than 18 months).

Blood lead levels in children from venous and capillary blood samples were assessed together, as one outcome of blood lead levels.

We used cognitive and neurobehavioural outcomes, adverse events, blood lead levels in children and household dust measures to populate Summary of findings for the main comparison and Summary of findings 2.

Search methods for identification of studies

The previous version of this review included studies up to January 2012. For this update, we revised the search strategies used in the original review (Appendix 1) by introducing additional search strings in which 'lead' was found in proximity to other terms (rather than searching for 'lead' as a single term), and we ran this revised strategy from 2012 to 2016. In addition, we added the Conference Proceedings Citation Index - Science (CPCI-S) as a substitute for searching for conference papers in ZETOC, two additional databases (Cochrane Database of Systematic Reviews (CDSR) and Database of Abstracts of Reviews of Effects (DARE)), and two trial registers (ClinicalTrials.gov and World Health Organization International Clinical Trials Registry Platform (WHO ICTRP)), which we searched from inception up to 2016 (see Differences between protocol and review).

Electronic searches

The Information Specialist of the Cochrane Developmental, Psychosocial and Learning Problems Group (CDPLPG) searched the databases and trials registers listed below on 3 May 2016. The search strategies used for this update and the previous update are in Appendix 2. We report further search details, including the search dates and numbers of records retrieved by individual databases, in Appendix 3.

1. Cochrane Central Register of Controlled Trials (CENTRAL; 2016, Issue 4) in the Cochrane Library, which includes the CDPLPG Specialised Register.
2. Ovid MEDLINE (1946 to April week 3 2016).
3. Embase Ovid (1980 to 2016 week 18).
4. PsycINFO EBSCO (1806 to April week 4 2016).
5. CINAHL EBSCO (Cumulative Index to Nursing and Allied Health Literature; 1937 to 3 May 2016).
6. Sociological Abstracts ProQuest (1952 to 3 May 2016).
7. ERIC EBSCO (Education Resources Information Center; 1966 to 3 May 2016).
8. Science Citation Index Web of Science (SCI; 1970 to 2 May 2016).
9. CPCI-S Web of Science (CPCI-S; 1990 to 2 May 2016).
10. CDSR (2016, Issue 5) in the Cochrane Library.
11. DARE (2015, Issue 2) in the Cochrane Library.
12. LILACS (Latin American and Caribbean Health Science Information database; bases.bireme.br; searched 3 May 2016).
13. WHO ICTRP (apps.who.int/trialsearch; searched 3 May 2016).
14. ClinicalTrials.gov (clinicaltrials.gov; searched 3 May 2016).

Searching other resources

We examined the reference list of relevant studies, and contacted experts to determine whether any unpublished or ongoing trials existed. We did not identify any further studies.

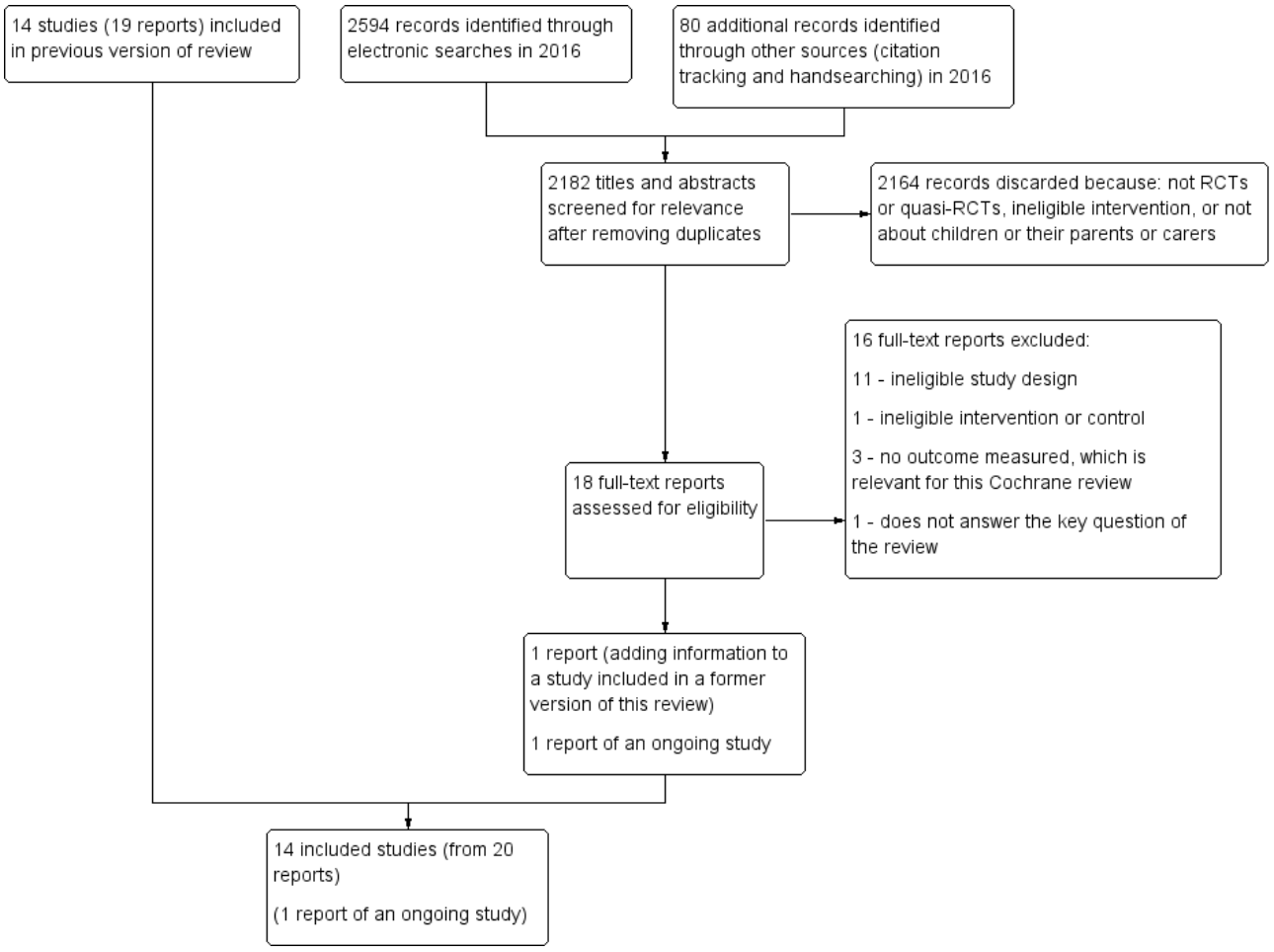
Data collection and analysis

Selection of studies

For this update, we screened records using AbstrackR 2015 software. After pilot-testing the screening-process, two review authors screened titles and abstracts from the search independently (BY and SW prior to 2016; BNS, LMP, LKB, SKL, SL and GG in 2016). We resolved disagreements by consensus and in consultation with a third author (GR prior to 2016; UG in 2016) and discarded records that did not fulfil our inclusion criteria (see Criteria for considering studies for this review). We retrieved potentially relevant reports for full-text assessment. Pairs of review authors (BY and SW prior to 2016; BNS and SKL, LMP and LKB, SL and GG in 2016) independently screened full-text reports for eligibility. We resolved disagreements by consensus and in consultation with a third author (GR prior to 2016; UG in 2016).

Figure 1 shows how many full-text publications we excluded and why.

Figure 1. Study flow diagram.



Data extraction and management

We stored records yielded by the electronic searches in reference management software (EndNote 2012). We recorded and managed the results of abstract and full-text screening, including information on the reasons for exclusion at full-text assessment, in the Endnote database. We stored and managed records from trial registries in an Excel spreadsheet. We organised data using Review Manager (RevMan) version 5 (RevMan 2014). We developed and piloted data extraction forms a priori, extracting the information described below.

1. Methods: study design, study location or setting, recruitment, follow-up, intention-to-treat, power calculation.
2. Participants: eligibility criteria, participation rate, reason for non-participation, numbers analysed, number of dropouts/ withdrawals, reasons for dropouts/withdrawals, baseline characteristics (sex, mean age, mean blood lead levels for each treatment group).
3. Interventions: brief descriptions of intervention (including frequency and duration of intervention events) and usual care provided.
4. Outcomes: timing of follow-up events, outcomes assessed and scales used.
5. Notes: information on funding, conflicts of interests and further information to aid understanding of the study.

Two authors (BY and SW prior to 2016; BNS and SL in 2016) independently completed data extraction forms for each study (14 included studies and 1 ongoing study). No disagreements arose.

Assessment of risk of bias in included studies

In the previous version of this review (Yeoh 2014), two review authors (of BY, SW, GR and NL) assessed the risk of bias of included studies. For this update, two review authors (BNS and UG) assessed the 'blinding' domain in accordance with the updated methodological criteria in the*Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011a), as 'blinding of participants and personnel' and 'blinding of outcome assessment'.

For each included study, we rated the following domains as being at high, low or unclear risk of bias.

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 investigators or participants could have foreseen intervention allocations before or during enrolment.

3. **Blinding of participants and personnel** describes all measures used, if any, to blind study participants and personnel from knowledge of which intervention a participant received.
4. **Blinding of outcome assessment** describes all measures used, if any, to blind outcome assessors from knowledge of which intervention a participant received.
5. **Incomplete outcome data** describes the completeness of outcome data, including attrition and exclusions from the analysis.
6. **Selective outcome reporting** considers whether trialists reported on all relevant and pre-specified outcomes.
7. **Other sources of bias** considers any important concerns about bias not addressed by the other domains (listed above) in the tool.

For cluster-randomised studies, we additionally looked at the risk of recruitment bias, baseline imbalance, loss to follow-up of clusters, and unit of analysis bias.

Where there was insufficient information in the published study regarding methodology or results in an extractable form, we (BNS and BY) contacted authors via email (and fax or phone call if required). If we did not receive a reply after the first contact, we sent one reminder. We did not score risk of bias on an additive basis.

Measures of treatment effect

Dichotomous data

Where outcomes from either standardised instruments or diagnostic evaluations were expressed as proportions, we calculated the risk ratio (RR) with 95% confidence intervals (CIs). We chose to calculate the RR over the odds ratio (OR), because the OR is more difficult to interpret correctly and potentially misleading to the reader.

For dichotomous data, we performed the analysis on the number of children with blood lead levels at or above two thresholds: 10.0 µg/dL (0.48 µmol/L) and 15.0 µg/dL (0.72 µmol/L). We chose these cut-off points because most primary research to date has used them, although the recently updated CDC reference value of 5.0 µg/dL suggests that a lower threshold is indicated (CDC 2012). We did not calculate risk differences because they strongly depend on baseline risks and are not as stable as RRs (Higgins 2011b).

For additional methods archived for future updates of this review, please see Yeoh 2006 and Appendix 4.

Continuous data

Where standardised assessment tools generated a score as the outcome measure, we conducted comparisons between the means of these scores. We used post-treatment means and standard deviations (SD) in all meta-analyses. We used the mean difference (MD) of post-treatment means as the outcome measure of choice because all studies reported outcomes on the same scale. As blood lead level data are typically positively skewed, included studies often provided log transformation of lead data (presented as geometric means). To prepare data for meta-analysis, we performed a natural log transformation of all geometric means. We calculated SDs from geometric confidence intervals (CIs), where necessary, using the calculation for small sample size (Higgins 2011c), to integrate it in the meta-analysis. If trials provided arithmetic means and SDs, we contacted authors to clarify that the data were normally distributed, and if no clarification was available, we assumed that this was the case. We then converted arithmetic means and SDs to approximate means and SDs on the log transformed scale according to Higgins 2008 before including them in the meta-analysis. Where raw data were available, we calculated post-treatment means and SDs on the log-transformed data. We also performed exponentiation of the results.

For additional methods archived for future updates of this review, please see Yeoh 2006 and Appendix 4.

Unit of analysis issues

Cluster-randomised trials

To determine the impact of possible unit of analysis errors arising from inadequate adjustment for cluster randomisation in published results by Hilts 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 2011d). 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 provided examples of ICCs (Ukoumunne 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 (Hilts 1995). We calculated design effects using an ICC of 0.001 or less, resulting in no change in the sample sizes for intervention and control groups, so we did not use these data in further analyses.

Studies with multiple treatment groups

We reported the results of each treatment group narratively because we could not integrate the only study consisting of multiple treatment groups into a meta-analysis (Sterling 2004).

For methods archived for future updates of this review, please see Yeoh 2006 and Appendix 4.

Dealing with missing data

Where some data on trial methods or results were not available in the study reports, we contacted trial authors. Where no reply was forthcoming or full data were not made available, we included available data only in the meta-analysis, where possible.

For each study we assessed the participation rate (enrolled/eligible). We also stated the number of participants who were in the final analysis as a proportion of all randomised participants in each study and presented reasons for missing data (please see Characteristics of included studies tables for more information).

For additional methods archived for future updates of this review, please see Yeoh 2006 and Appendix 4.

Assessment of heterogeneity

We assessed consistency of results visually and by examining I^2 (Higgins 2002), a quantity that describes the approximate proportion of variation in point estimates that is due to heterogeneity rather than sampling error. In addition, we used the Chi^2 test to assess the statistical significance of the heterogeneity. We considered a P value less than 0.10 as statistically significant. We also reported Tau^2, an estimate of the between-study variance in a random-effects meta-analysis.

We examined clinical heterogeneity by comparing PICO (patient/population/problem, intervention, comparator, outcome) definitions of included studies. We assessed methodological heterogeneity by comparing study designs.

Data synthesis

When two or more studies reported data that could be combined, we performed a meta-analysis. For any given outcome, we calculated the mean difference (MD) and risk ratio (RR) with 95% CIs for continuous and dichotomous data, respectively, using both the random-effects (DerSimonian and Laird) and fixed-effect (Mantel-Haenszel) models. We reported the results of the random-effects models because we assumed that the effects of secondary prevention are not identical across different populations and settings. The results of the random-effects and fixed-effect models, in general, were similar. We analysed data from RCTs separately from quasi-RCTs.

Subgroup analysis and investigation of heterogeneity

We organised studies into subgroups for clinically different interventions as described below.

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

Please see Yeoh 2006 and Appendix 4 for additional subgroup analyses archived for future updates of this review.

Sensitivity analysis

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

Please see Yeoh 2006 and Appendix 4 for additional sensitivity analyses archived for future updates of this review.

Summary of findings table

With the exception of cost of intervention, we assessed the quality of evidence for each outcome deemed critical for decision-making, using the GRADE approach (Guyatt 2011). As recommended by GRADE, we constructed a 'Summary of findings' table for the main interventions: education strategies for preventing domestic lead exposure in children, and environmental strategies (dust control) for preventing domestic lead exposure in children. We presented the results from the meta-analyses in the 'Summary of findings' tables (Summary of findings for the main comparison; Summary of findings 2). To judge the quality of evidence, we assessed risk of bias, inconsistency, indirectness, imprecision, and publication bias of the evidence base for each outcome. The judgement of quality of evidence was based on GRADE's four categories: high quality, when further research is very unlikely to change our confidence in the estimate of effect; moderate quality, when further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate; low quality, when 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; or very low quality, when we are very uncertain about the estimate of the effect (Balshem 2011; Guyatt 2011).

RESULTS

Description of studies

Results of the search

We completed the literature search for the original version of this review at the end of May 2006, retrieving 12,308 titles. We rejected records at title and abstract stage if they were not primarily about lead exposure in children, were not RCTs or quasi-RCTs, or did not fulfil the inclusion criteria as outlined in Criteria for considering studies for this review. We retrieved and assessed the full-texts of 25 promising papers and of these, we identified 20 unique trials (with 5 reports being additional publications for these trials). Of the 20 separate trials identified, we included 12 and excluded 8. We did not identify any unpublished papers or ongoing papers (Yeoh 2008).

We updated the searches at the end of April 2010 and again at the end of January 2012. After excluding reports based on above methods at title and abstract stage, we identified two additional studies, bringing the included trials to 14 in the present review (see Yeoh 2014).

For the purposes of this update, we revised our original search strategy by introducing additional search strings in which 'lead' was found in proximity to other terms and reran our revised search from 2012 to 2016. We also added CPCI-S as a substitute for searching for conference papers in ZETOC, two additional databases (CDSR and DARE) to identify other reviews, and two trials registers (ClinicalTrials.gov and WHO ICTRP), which we searched using our revised strategy for all available years (see Differences between protocol and review). This strategy yielded 2594 records from electronic searches and 80 records from citation tracking and searches of grey literature. After removing duplicates, we screened 2182 titles and abstracts for eligibility. We rejected 2164 records at title and abstract stage because they did not fulfil the inclusion criteria as outlined in Criteria for considering studies for this review. We retrieved and assessed the full-texts of 18 promising reports, and of these we excluded 16 (see Excluded studies). We identified one paper reporting additional information on a previously included study (Campbell 2011) and one report of an ongoing study (the results of which will probably be available by the end of 2017) that met our eligibility criteria (Figure 1).

Included studies

Please see Characteristics of included studies.

Design

This review includes 13 RCTs (Weitzman 1993; Hilts 1995; Lanphear 1996a; Aschengrau 1998; Farrell 1998; Lanphear 1999; Rhoads 1999; Wasserman 2002; Jordan 2003; Sterling 2004; Brown 2006; Boreland 2009; Campbell 2011), along with 1 quasi-RCT (Charney 1983). The trials correspond to 20 records and involved 2643 children under six years of age. All studies used a parallel-group design, with one study also performing the intervention on volunteers from the control group at a later date (Weitzman 1993). As there was no parallel control group in this second phase, we did not include these results in our review. Another study by Campbell 2011 included a matched control group in addition to the two randomised arms at the analysis stage. The study methods had pre-specified this group, but it was not part of the randomisation process so we could not include the results

of this review. As a consequence, we included data only from the two randomised study arms (maintenance education group = treatment arm, and standard education group = control arm). Twelve studies used individuals or households (Charney 1983; Weitzman 1993; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Rhoads 1999; Wasserman 2002; Jordan 2003; Sterling 2004; Brown 2006; Boreland 2009; Campbell 2011), and two studies used clusters (neighbourhoods and blocks of six households; Hilts 1995; Farrell 1998 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 studies had 100 to 200 participants (Weitzman 1993; Hilts 1995; Lanphear 1996a; Rhoads 1999; Sterling 2004; Brown 2006), and four studies had more than 200 participants (Farrell 1998; Lanphear 1999; Jordan 2003; Campbell 2011).

Participants and setting

Thirteen included studies took place in urban areas of North America; one study was performed in Broken Hill, Australia (Boreland 2009). Most studies were performed in areas of low 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 involved significant proportions of people identifying themselves as African-American or Hispanic. Boys and girls were equally represented in all studies.

Thirteen studies recruited participants from routine screening programmes, medical clinics, previous lead studies or community volunteers, and they excluded children who had clinical symptoms, were receiving treatment for lead toxicity (e.g. chelation) or had high blood lead levels requiring intervention (> 20.0 µg/dL to 24.0 µg/dL; 0.97 µmol/L to 1.16 µmol/L) (Weitzman 1993; Hilts 1995; Lanphear 1996a; Aschengrau 1998; Farrell 1998; Lanphear 1999; Rhoads 1999; Wasserman 2002; Jordan 2003; Sterling 2004; Brown 2006; Boreland 2009; Campbell 2011). 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 levels below 10.0 µg/dL (0.48 µmol/L) (Lanphear 1996a; Lanphear 1999; Wasserman 2002; Jordan 2003; Campbell 2011), five reporting levels between 10.0 µg/dL and 14.0 µg/dL (0.48 µmol/L to 0.68 µmol/L) (Weitzman 1993; Hilts 1995; Farrell 1998; Rhoads 1999; Sterling 2004), three reporting levels between 15.0 µg/dL and 19.0 µg/dL (0.72 µmol/L to 0.92 µmol/L) (Aschengrau 1998; Brown 2006; Boreland 2009), and one reporting levels above 20.0 µg/dL (0.97 µmol/L) (Charney 1983). See Table 1 for more information.

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

Interventions

The interventions used in the studies were either educational, environmental or a combination of both (for detailed information on the interventions used see Characteristics of included studies). In studies using educational interventions, three studies used education alone (Wasserman 2002; Jordan 2003; Brown 2006), and two studies used education plus provision of cleaning products (Lanphear 1996a; Lanphear 1999). Of the studies using environmental interventions, two studies used soil abatement (Weitzman 1993; Farrell 1998), and three studies 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). See Table 2 for more information.

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 adherence to recommended housekeeping practices. Investigators did not measure adherence. We contacted all authors of included studies, and all responded.

Control

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

Intervention duration

For 12 studies the duration of the intervention ranged between 3 months and 24 months. In the two studies that used soil abatement intervention, the intervention was performed on a single occasion during the study (Weitzman 1993; Farrell 1998).

Outcomes

No studies used any standardised cognitive and neurobehavioural outcomes or gathered standardised information on adverse events in children.

Blood lead level was the standardised outcome reported in all studies. Five studies reported household floor dust (Hilts 1995;

Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Campbell 2011; see 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, 7 of the 12 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). Aschengrau 1998 reported data as having a normal distribution. As no clarification was available for the remaining studies providing arithmetic means, we 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).

Follow-up duration

The period of follow-up from baseline ranged from 6 months to 48 months, with most studies reporting blood lead levels at 3 months to 12 months postintervention. Three studies provided data at longer time points (Lanphear 1999; Jordan 2003; Campbell 2011). Lanphear 1999 collected data up to 18 months postintervention with a follow-up publication at 48 months (Lanphear 2000). Jordan 2003 had follow-up data reported at four-month intervals up to three years postintervention. Campbell 2011 reported the blood lead levels after 24 months follow-up.

We used short-term postintervention data from the two long-term studies in our meta-analysis (6 months for Lanphear 1999 and 18 months for Jordan 2003) to enable a more comparable follow-up period to other included studies. With regard to household dust level outcomes, we used six-month follow-up data for the two studies with available data (Lanphear 1996a; Lanphear 1999).

Ongoing studies

We identified one ongoing study, which is due to be completed in September 2017 (NCT00129324). This study enrolled pregnant women and their children, comparing a lead hazard control intervention to injury hazard control intervention and no intervention. Outcomes of interest will be blood lead levels and neurobehavioral outcomes in children.

Excluded studies

Please see Characteristics of excluded studies.

Across all versions of the review (Yeoh 2008; Yeoh 2012; Yeoh 2014), including this update, we assessed 45 full-texts reports for eligibility. Of these, we included 20 reports (14 studies) in the review, identified 1 ongoing study and excluded 24 reports (reasons for exclusion are provided in the Characteristics of excluded studies table). We excluded 8 of these studies in the previous version (Yeoh 2014) and 16 in this update.

For this update, we excluded 11 studies because of ineligible study design: five were observational studies (Farfel 1990; Malcoe 2004; Dixon 2012; NCT00000104; NCT00011674), two used qualitative research methods (Thomas 2013; Feit 2014), two used a before-and-after design without a comparison group (Phoenix 2013; Wilson 2015), one study was a systematic review (a former version of this Cochrane Review; Yeoh 2014), and one was a cross-sectional study (Whitehead 2014). We also excluded one study because it used an ineligible, historical control without randomisation (EPA 1997). We excluded a further three studies because they did not measure an outcome that was relevant for this Cochrane review: Butterfield 2011 measured parent's self-efficacy and precaution adoption, Zimmermann 2006 investigated iron fortification as the intervention, and Maharaj 2007 consisted of a discussion paper about the link between lead and asthma. Finally, we excluded one study because it did not answer a key question of the review: Untimanon 2012 did not focus on preventing lead exposure but on contamination modes.

In the previous version of this review, we excluded three studies because they used retrospective or historical controls without randomisation (EPA 1996; Taha 1999; Pollak 2002), one study because it reported long-term follow-up for an included trial but did not use controls (Aschengrau 1994), one study because it compared two groups from different study bases (Omidpanah 1998), and three studies because they did not measure any outcomes relevant for this Cochrane review: Boreland 2006 measured environmental measures, Dugbatey 2005 measured maternal blood levels, and Marlowe 2001 measured hair lead levels.

Risk of bias in included studies

This review includes 13 RCTs and one quasi-RCT in which alternate clinic numbers determined allocation to groups (Charney 1983). We received responses from all 14 corresponding authors of the included studies when we contacted them to provide missing information on methodology or results, but in many instances, some of the requested information was not available.

Figure 2 and Figure 3 show the risk of bias of each domain for all included studies.

Figure 2. Risk of bias graph: review authors' judgements about each risk of bias item presented as percentages across all included studies.

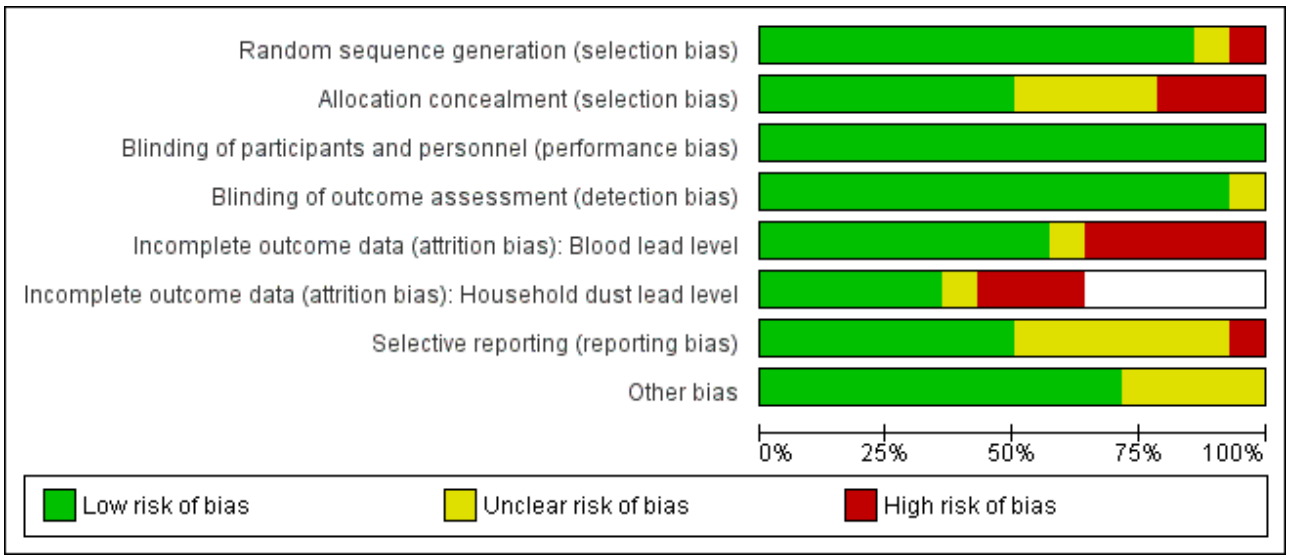
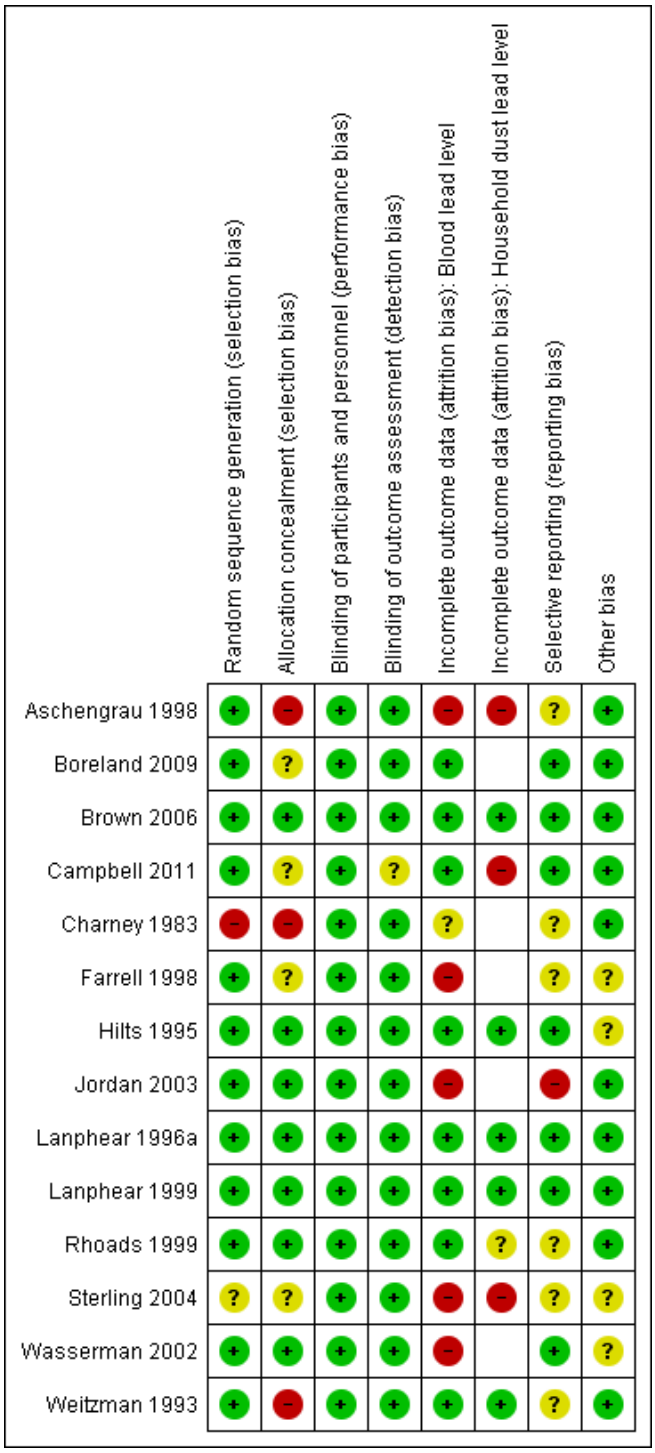


Figure 3. Risk of bias summary: review authors' judgements about each risk of bias item for each included study.



Allocation

Sequence generation

Of the 13 RCTs, the method of randomisation was adequate for 12 studies with available information. Eight studies used random number generators, tables or lists (Weitzman 1993; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Wasserman 2002; Jordan 2003; Brown 2006; Campbell 2011), two studies used coin toss (Farrell 1998; Boreland 2009), one study used numbered slips of paper (Hilts 1995), and one study used permutated blocks of varying length (Rhoads 1999). Methods of randomisation were unclear in one study (Sterling 2004). We rated the quasi-RCT, in which alternate clinic numbers determined allocation to groups, at high risk of bias (Charney 1983).

Allocation concealment

Of the 13 RCTs, 7 used adequate methods of allocation concealment with either sealed envelopes or a central office (Hilts 1995; Lanphear 1996a; Lanphear 1999; Rhoads 1999; Wasserman 2002; Jordan 2003; Brown 2006). Allocation concealment remained unclear in four studies (Farrell 1998; Sterling 2004; Boreland 2009; Campbell 2011). Two of the RCTs did not report adequate concealment (Weitzman 1993; Aschengrau 1998).

We assessed the quasi-RCT as being at high risk of bias because it had no allocation concealment (Charney 1983).

Blinding

Blinding participants and personnel (performance bias)

Although not every study blinded participants and personnel, we rated the risk for performance bias as low because the participants' knowledge on treatment allocation probably had no influence on outcomes like blood lead level and household dust levels.

Blinding outcome assessment (detection bias)

All but one study blinded outcome assessors for dust and blood samples; the personnel collecting dust samples in Campbell 2011 knew the household assignment. It is unclear whether this knowledge could have biased the result, so we rated the risk of detection bias accordingly.

Incomplete outcome data

We rated risk for attrition bias separately for the outcomes of blood lead levels and household dust measures of lead exposure.

For blood lead levels, we rated the risk of attrition bias as low in eight studies because the attrition rate was acceptable and similar in both the intervention and control groups (Weitzman 1993; Hilts 1995; Lanphear 1996a; Lanphear 1999; Rhoads 1999; Brown 2006; Boreland 2009; Campbell 2011). In one study we rated the risk of attrition as unclear, because the overall attrition rate was quite high, but it was the same in both groups (Charney 1983). In the other studies we rated the risk of attrition bias as high. In Aschengrau 1998 the overall attrition rate was 41%; it was 18% points higher in the intervention group than in the control group. In Jordan 2003 the attrition rate was 38%, with no information on the attrition rate in the treatment arms. In Wasserman 2002 the attrition rate was acceptable (21%); however, it was much higher in the control group (30%) than in the intervention group (12%). In Farrell 1998 the attrition rate was 55%, and in Sterling 2004 it was 61%.

Of the nine studies that reported on household dust measures of lead exposure, we rated five as being at low risk of attrition bias because the attrition rate was acceptable and similar in both the intervention and control groups (Weitzman 1993; Hilts 1995; Lanphear 1996a; Lanphear 1999; Brown 2006). We assessed one study as being at unclear risk because numbers and reasons for missing data were not available (Rhoads 1999). In Aschengrau 1998, Campbell 2011 and Sterling 2004, we rated the risk of attrition bias for household dust measures of lead exposure as high, because overall attrition rates were 46%, 64% and 66%, respectively.

The most common reasons reported for withdrawal were that families had moved out of the area or were no longer reachable.

We contacted authors to determine if they had analysed participants in the groups to which they were randomised (intention-to-treat). None of the studies performed complete measures of all participants' outcomes (full intention-to-treat analysis). Seven studies analysed data based on available participants' outcomes (available case analysis; Weitzman 1993; Hilts 1995; Lanphear 1996a; Lanphear 1999; Brown 2006; Boreland 2009; Campbell 2011). We were unable to determine if five studies used data from all available participants (Charney 1983; Rhoads 1999; Wasserman 2002; Jordan 2003; Sterling 2004). In two studies, participants were excluded from analyses if non-study interventions (such as any lead hazard reduction measures performed independently of study intervention) occurred during the study (Aschengrau 1998; Farrell 1998).

Selective reporting

Information from authors suggest that published reports of seven studies included all expected outcomes, including those they had pre-specified (Hilts 1995; Lanphear 1996a; Lanphear 1999; Wasserman 2002; Brown 2006; Boreland 2009; Campbell 2011). For six studies there was insufficient information, so we rated the risk of bias for this domain as unclear (Charney 1983; Weitzman 1993; Aschengrau 1998; Farrell 1998; Rhoads 1999; Sterling 2004). One study described measuring household dust lead outcomes but did not report these data in the article. We could not obtain any information on these outcomes from the author (Jordan 2003).

Other potential sources of bias

Funding

Of the 14 included studies, two studies did not mention their funding source, so we judged them to be at unclear risk of bias (Wasserman 2002; Sterling 2004).

Other potential sources of bias in cluster-randomised trials

Two of the 14 included studies were cluster-randomised trials, a design that can be affected by additional sources of bias. Risk of recruitment bias and risk of bias due to baseline imbalance was low in Farrell 1998 and Hilts 1995, since baseline characteristics were comparable and randomisation was achieved by coin toss. Risk of bias due to unit of analysis was unclear in Farrell 1998, who used neighbourhood clusters, but it was unclear how they performed the analysis, as data were not available; and high in Hilts 1995, since Hilts 1995 randomised clusters of households but used individuals as the unit of analysis.

Effects of interventions

See: **Summary of findings for the main comparison** Education strategies versus no intervention for preventing domestic lead exposure in children; **Summary of findings 2** Environmental strategies (dust control) versus no intervention for preventing domestic lead exposure in children

We present results sequentially by intervention type, by outcome measure and by type of data (continuous and dichotomous). Cost data are presented at the end of this section for all intervention types combined.

We classified the 14 studies into 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).

Comparison 1. Education interventions versus no intervention or standard education

Primary outcomes

Cognitive and neurobehavioural outcomes in children

None of the included studies measured cognitive or neurobehavioural outcomes.

Adverse events of the intervention in children

None of the included studies measured adverse events in children in a standardised way.

Secondary outcomes

Blood lead levels in children

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.

See Summary of findings for the main comparison.

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%, Tau^2 = 0.00; 5 studies, N = 815; high quality evidence; Analysis 1.1). Exponentiation of the result produced a treatment effect of 1.0 µg/dL (95% CI 0.9 to 1.1; analysis not shown). The mean age for participants in these studies was less than two years, and baseline blood level was less than 10.0 µg/dL (0.48 µmol/L) in all studies except for Brown 2006. As the baseline blood lead level for Brown 2006 was 15.0 µg/dL to 19.0 µg/dL (0.72 µmol/L to 0.92 µmol/L), we performed a sensitivity analysis to assess the effect of clinical heterogeneity. When we excluded Brown 2006, there was still no evidence of a treatment effect (MD −0.01, 95% CI −0.13 to 0.11; I^2 = 0%; analysis not shown). Exponentiation of the result produced a treatment effect of 1.0 µg/dL (95% CI 0.9 to 1.1; analysis not shown).

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 of 10.0 µg/dL (0.48 µmol/L) or more showed no evidence of a treatment effect (RR 1.02, 95% CI 0.79 to 1.30; I^2 = 0%, Tau^2 = 0.00; N = 520; moderate quality evidence; Analysis 1.2). Meta-analysis of data reported as numbers of children with blood lead level of 15.0 µg/dL (0.72 µmol/L) or more also showed no evidence of a treatment effect (RR 0.60, 95% CI 0.33 to 1.09; I^2 = 0%, Tau^2 = 0.00; N = 520; moderate quality evidence; Analysis 1.3).

Household dust measures of lead exposure

Continuous data

Two of the five studies had log transformed summary data available on hard floor dust lead levels for this intervention (Lanphear 1996a; Lanphear 1999). The mean hard floor dust level was below the 0.431 mg/m^2 (40.0 µg/ft^2) dust lead standard established by the US Department of Housing and Urban Development (HUD) and the US Environmental Protection Agency (EPA) for the home environment. 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; I^2 = 0%, Tau^2 = 0.00; N = 318; moderate quality evidence; Analysis 1.4). Exponentiation of the result produced a treatment effect of 0.010 mg/m^2, 95% CI 0.008 to 0.014; analysis not shown (0.9 µg/ft^2, 95% CI 0.7 to 1.3). Brown 2006 reported postintervention floor dust lead levels as geometric means, but type of floor was not specified (hard floor or carpet), so we did not include the data in the meta-analysis. After one year, the dust lead level was 0.095 mg/m^2 (8.8 µg/ft^2) in the control group and 0.059 mg/m^2 (5.5 µg/ft^2) in the intervention group, and the difference was statistically significant (P < 0.05, not presented more precisely in the study). One study had data on carpet floor (Lanphear 1996a), showing 0.038 mg/m^2, 95% CI 0.017 to 0.081 (3.5 µg/ft^2, 95% CI 1.6 to 7.6) in the intervention group and 0.044 mg/m^2, 95% CI 0.014 to 0.138 (4.1 µg/ft^2, 95% CI 1.3 to 12.8) in the control group after seven months (P value = 0.72).

Three of the five studies reported outcomes on window dust lead levels. We did not pool these studies in a meta-analysis because investigators used different surfaces to collect the dust lead samples (window sill, window troughs, window wells or sills in general). Brown 2006 reported dust lead levels in "other sills", not specifying where they were. One year after the intervention the level was 0.273 mg/m^2 (25.4 µg/ft^2) in the intervention group compared to 0.563 mg/m^2 (52.3 µg/ft^2) in the control group; the difference was statistically significant (P < 0.05, not presented more precisely in the study). Lanphear 1996a measured dust lead levels in interior window sills and window wells. At the end of the study the level was 0.961 mg/m^2, 95% CI 0.260 to 3.539 (89.3 µg/ft^2, 95% CI 24.2 to 328.8) in the intervention group compared to 0.972 mg/m^2, 95% CI 0.203 to 4.648 (90.3 µg/ft^2, 95% CI 18.9 to 431.9) in the control group, and 32.421 mg/m^2, 95% CI 2.723 to 379.212 (3012.0 µg/ft^2, 95% CI 253.0 to 35,230.0) in the intervention group compared to 40.989 mg/m^2, 95% CI 4.434 to 379.212 (3808.0 µg/ft^2, 95% CI 412.0 to 35,230.0) in the control group, respectively. The difference was not statistically significant for either types of outcome. Also, Lanphear 1999 did not report any statistically significant difference between control and intervention group at the end of the study in lead in window sills (intervention: 1.153 mg/m^2 (107.1 µg/ft^2), control: 1.547 mg/m^2 (143.7 µg/ft^2); P value = 0.19) and window troughs (intervention: 23.397 mg/m^2 (2173.7 µg/ft^2), control 28.516 mg/m^2 (2649.2 µg/ft^2); P value = 0.54).

Comparison 2. Environmental interventions versus no intervention or another intervention not aiming to influence domestic lead exposure

Primary outcomes

Cognitive and neurobehavioural outcomes in children

None of the included studies measured cognitive or neurobehavioural outcomes.

Adverse events of the intervention in children

None of the included studies measured adverse events in children in a standardised way.

Secondary outcomes

Blood lead levels in children

Dust control

Continuous data

Three studies used dust control interventions (Hilts 1995; Rhoads 1999; Boreland 2009). Hilts 1995 and Boreland 2009 reported log transformed summary data while Rhoads 1999 reported arithmetic means and SDs. 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; I^2 = 90%, Tau^2 = 0.05; N = 298; low quality evidence; Analysis 2.1). An I^2 of 90% is very high, indicating high heterogeneity between studies that cannot be explained by sampling error. One possible explanation for such a high I^2 could be that the interventions were too different in these studies. While Hilts 1995 and Rhoads 1999 used high-efficiency particulate air (HEPA) vacuum cleaning as an intervention, Boreland 2009 used a more intensive intervention: house remediation work (e.g. ceiling dust removal, sealing of ceilings, paint stabilisation, replacement of floor coverings/windows, and cleaning). However, excluding Boreland 2009 from the meta-analysis increased the I^2 value. Other possible explanations include the difference in the age of the children and the limitations of using the I^2 statistic when only a few studies are included in a meta-analysis. Exponentiation of the result produced a treatment effect of 0.9 µg/dL (95% CI 0.7 to 1.1; analysis not shown).

Dichotomous data

We performed a meta-analysis of dichotomous data for two studies (Hilts 1995; Rhoads 1999). The meta-analysis for numbers of children with blood lead level of 10.0 µg/dL (0.48 µmol/L) or more showed no evidence of a treatment effect (RR 0.93, 95% CI 0.73 to 1.18; I^2 = 0%, Tau^2 = 0.00; N = 210; moderate quality evidence; Analysis 2.2), and this was also the case for children with blood lead levels at or above 15.0 µg/dL (0.72 µmol/L; RR 0.86, 95% CI 0.35 to 2.07; I^2 = 56%, Tau^2 = 0.23; N = 210; low quality evidence; Analysis 2.3).

Impact of clustering and unit of analysis errors

We calculated effective sample sizes for the cluster-randomised trial for a range of ICCs before incorporating this study into the meta-analysis (Hilts 1995). For blood lead levels of 10.0 µg/L (0.48 µmol/L) or more, there was no statistically significant treatment benefit when we adjusted the meta-analysis for clustering: ICC 0.01 (RR 0.93, 95% CI 0.73 to 1.18; I^2 = 0%, Tau^2 = 0.00; 2 studies; N = 204; Analysis 2.4); ICC of 0.1 (RR 0.95, 95% CI 0.72 to 1.24; I^2 = 0%, Tau^2 = 0.00; 2 studies; N = 173; Analysis 2.5); or ICC of 0.2 (RR 0.97, 95% CI 0.72 to 1.29; I^2 = 0%, Tau^2 0.00; 2 studies; N = 155; Analysis 2.6). For blood lead levels of 15.0 µg/dL (0.72 µmol/L) or more, there was no statistically significant treatment benefit when the meta-analysis was adjusted for clustering: ICC 0.01 (RR 0.82, 95% CI 0.37 to 1.81; I^2 = 45%, Tau^2 = 0.15; 2 studies; N = 204; Analysis 2.7); ICC 0.1 (RR 0.83, 95% CI 0.34 to 2.03; I^2 = 48%, Tau^2 = 0.20; 2 studies; N = 173; Analysis 2.8); or ICC 0.2 (RR 0.75, 95% CI 0.34 to 1.66; I^2 = 25%, Tau^2 = 0.08; 2 studies; N = 155; Analysis 2.9). Thus, correcting for unit of analysis errors did not alter the overall outcome.

Soil abatement

Two studies performed soil abatement interventions (Weitzman 1993; Farrell 1998). As no blood lead level data were available in a usable form from 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 were available for our analysis. Weitzman 1993 reported a statistically significant effect in favour of the intervention. The difference in mean change scores between the intervention group and control group A (loose interior dust abatement and paint removal) was −1.5 µg/dL (SD 4.9), and between the intervention group and control group B (loose interior paint removal only), it was −1.9 µg/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 this review.

Household dust measures of lead exposure

Dust control

One study provided household carpet lead measures for dust control interventions (Hilts 1995), reporting no clinically significant treatment effect with geometric means for post-treatment dust lead level, which was 0.360 mg/m^2 (33.5 µg/ft^2) in the intervention group and 0.230 mg/m^2 (21.4 µg/ft^2) in the control group. Rhoads 1999 provided geometric means of lead loading from floor wipes, sill wipes and vacuum after one year. Investigators reported no significant difference for floor wipes (intervention: 0.163 mg/m^2 (15.1 µg/ft^2), control: 0.207 mg/m^2 (19.2 µg/ft^2)) or vacuum (intervention: 1.618 mg/m^2 (150.3 µg/ft^2), control: 2.307 mg/m^2 (214.3 µg/ft^2)). For sill wipes, the difference was statistically significant ($P < 0.05$, not presented more precisely in the study), and lead levels were 0.263 mg/m^2 (24.4 µg/ft^2) in the intervention group and 0.522 mg/m^2 (48.5 µg/ft^2) in the control group.

Soil abatement

No studies reported household dust lead levels for this intervention.

Comparison 3. Combination interventions versus standard education

Primary outcomes

Cognitive and neurobehavioural outcomes in children

None of the included studies measured cognitive or neurobehavioural outcomes.

Adverse events of the intervention in children

None of the included studies measured adverse events in children in a standardised way.

Secondary outcomes

Blood lead levels in children

Of the four studies that used a combination of interventions, two reported continuous data (Aschengrau 1998; Campbell 2011), one arithmetic means (Aschengrau 1998), and one geometric means (Campbell 2011). One study reported dichotomous data (Sterling 2004), and the fourth was clinically very different because it was a quasi-RCT (Charney 1983), with high mean baseline blood lead

levels (> 30.0 µg/dL, or 1.44 µmol/L) and older participants (mean age 3.5 years). It was therefore not possible or appropriate to combine any of these studies in a meta-analysis.

Aschengrau 1998 reported arithmetic means for post-treatment blood lead levels as 11.5 µg/dL (SD 3.2) in the intervention group and 10.4 µg/dL (SD 3.1) in the control group. An analysis of these post-treatment scores performed in our review failed to reach statistical significance: MD 1.1 µg/dL (95% CI −1.5 to 3.7; analysis not shown). Sterling 2004 reported dichotomous data with 4/10 (40%) in intervention group 1; 6/14 (43%) in intervention group 2, and 6/15 (40%) in the control group having blood lead levels less than 10.0 µg/dL (0.48 µmol/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 at or above 10.0 µg/dL (0.48 µmol/L), showed no evidence of treatment effect (intervention group 1 – newsletters and education: RR 1.00, 95% CI 0.52 to 1.92; intervention group 2 – newsletters, education and specialised cleaning: RR 0.95, 95% CI 0.52 to 1.76; analyses not shown). Charney 1983 reported a significant effect favouring treatment with arithmetic means for post-treatment blood lead levels of 31.7 µg/dL (SD 2.6) in the intervention group and 37.8 µg/dL (SD 7.9) in the control group. Campbell 2011 reported similar geometric means for blood lead levels after 12 months for the intervention group (2.6 µg/dL) and control group (2.7 µg/dL) (P value = 0.68). Likewise, blood lead levels for the intervention group (3.5 µg/dL) and the control group (3.9 µg/dL) were not significantly different after two years (P value = 0.20).

Household dust measures in children

One study provided continuous data of hard floor and window dust lead levels for this intervention subgroup (Aschengrau 1998). We found no treatment effect, with median changes for floor dust lead level being -0.002 mg/m^2 (−0.2 µg/ft^2, SD 0.8) in the intervention group and 0.001 mg/m^2 (0.0 µg/ft^2, SD 0.2) in the control group. For window sills, the mean change in the intervention group was -0.006 mg/m^2 (−0.5 µg/ft^2, SD 1.3) in the intervention group and -0.005 mg/m^2 (−0.5 µg/ft^2, SD 1.0) in the control group; for window wells, it was -0.007 mg/m^2 (−0.7 µg/ft^2, SD 0.9) in the intervention group and 0.000 mg/m^2 (0.0 µg/ft^2, SD 1.6) in the control group. A second study provided dichotomous data with no significant difference observed in the number of households with positive dust lead levels (floor > 0.431 mg/m^2 (40.0 µg/ft^2); window > 2.691 mg/m^2 (250.0 µg/ft^2)) between the intervention (17/59) and control (11/51) groups at 12 months post-treatment (Campbell 2011) .

Cost of intervention

Six studies provided cost data for their intervention or study, reporting large variations in costs depending on the types of interventions and types of cost data collected. The calculations often omitted the costs of researchers and educators. With regard to educational interventions, Brown 2006 noted that, on average, comparison families spent USD 108.78 and intervention families spent USD 43.01 on cleaning supplies. Wasserman 2002 reported that Medicaid paid for medical check-ups, and researchers spent USD 11 per blood test. With dust control interventions, Hilts 1995 reported that the entire study cost approximately USD 200,000, but no detailed costs for the intervention were available. Boreland 2009 reported that the average cost per household was AUD 5000 (in 1994), but ranged from AUD 1000 to AUD 20,000. For soil abatement, Farrell 1998 estimated that the average cost per household was USD 1700, with the entire study costing USD 5 million. For a combination of interventions, Sterling 2004 reported an average cost per quarterly cleaning of USD 500 per household, and Campbell 2011 reported median costs of lead hazard control or remediation work over a 12-month period of USD 4656 for 42 control households and USD 5512 for 36 intervention households. No cost data were available for seven studies (Charney 1983; Weitzman 1993; Lanphear 1996a; Aschengrau 1998; Lanphear 1999; Rhoads 1999; Jordan 2003).

DISCUSSION

Summary of main results

Of 2225 identified studies, 14 met our eligibility criteria for inclusion in this review. We identified no evidence on cognitive or neurobehavioural outcomes and adverse events. The results of this systematic review suggest that educational interventions and dust control interventions are not effective in reducing children's blood lead levels. Furthermore, when we adjusted the meta-analysis for the dust control subgroup for clustering, we again found no evidence of an effect.

We could not pool the studies that used soil abatement (removal and replacement) or combination intervention groups in a meta-analysis due to substantial differences between studies. For both soil abatement and combination interventions, two of the included studies reported reductions in blood lead level for treatment groups. We could not pool these results in a meta-analysis because studies used clinically distinct intervention types (soil abatement and a combination of interventions). One study showed a treatment effect with a combined (education and dust control) intervention (Charney 1983). As this was a quasi-RCT and had participants with high baseline blood lead levels (more than 30.0 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 be more beneficial in children who have 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 µg/dL to 1.9 µg/dL (0.07 µmol/L to 0.09 µmol/L). The clinical significance of this at 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, there is currently insufficient evidence to clarify whether soil abatement or a combination of interventions reduce blood lead levels.

Overall completeness and applicability of evidence

None of the included studies 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 blood lead level reductions reported in the studies with significant treatment effect and the known correlation between blood lead level and cognition, we do not expect that we would have found any significant improvement in cognitive outcomes even if investigators had measured them.

No studies measured adverse events in children in a standardised way. Future trials need to better examine and report adverse effects and ensure that sample sizes are sufficiently large to allow this.

Some experts point out that blood lead levels are not suitable across all levels or durations of exposure. A recent review described blood lead levels as the most suitable biomarkers when assessing recent or current exposures to lead (weeks or months) at low or moderate levels (Barbosa 2005), but at high levels of exposure, a curvilinear relationship between blood lead levels and exposure makes its use as a biomarker more difficult (Bergdahl 2008). Alternative measures have not shown to be superior to blood lead level when monitoring lead exposure (Barbosa 2005; Bergdahl 2008). Lead is, moreover, a bone-seeking element, a characteristic that is especially relevant for constant lead exposure (Rust 1999). From the bone, it can be released into the blood (Rabinowitz 1991; Gulson 2003), which is especially relevant for pregnant women and children (Manton 2000; Gulson 2003). This release of lead from bone into blood adversely affects the reduction of blood lead levels (Rust 1999; Gwiazda 2005). It is therefore necessary to assess remediation efforts over prolonged periods of time (i.e. at least 6 months to 12 months).

The participants in the included studies were all children younger than six years of age. Although we looked for studies in children from birth to 18 years of age, we did not identify any study on older children or adolescents. Future studies need to focus also on preventing lead exposure in these groups. All but one of the studies were conducted in North America (Boreland 2009), and we cannot rule out that the treatment effects would be different if the interventions were transferred to other contexts.

Meta-analysis was not possible for all interventions or outcomes due to the 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. However, due to the limited number of studies within each intervention type, there were insufficient data for subgroup analyses according to baseline age.

These issues of clinical diversity, inconsistent participant compliance with household cleaning practices, variability in interventions, suboptimal recruitment numbers and losses to follow-up that reduce study power may all contribute 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 with low socioeconomic status in the USA, in rental housing and, as such, results may not be generalisable to different populations.

We lack studies of full remediation, as interventions evaluated were not able to eliminate all ongoing environmental lead sources and were limited to household interventions. Therefore, 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, like passive smoking, water, diet or sources outside the home, may have limited the possible benefit of interventions. Another reason for lack of treatment effect may be that most 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.

Quality of the evidence

For educational interventions, we assessed the quality of evidence from continuous blood lead level outcomes as high, so it is unlikely that further research will change our confidence in the effect estimate. We assessed the quality of evidence for dichotomous blood lead level outcomes as moderate because the 95% CIs around the pooled estimate included no effect and appreciable harm or benefit, and because the total number of events was less than 300. We assessed the quality of evidence for household dust measures of lead exposure to be moderate; we downgraded one level for imprecision because the total number of participants was less than 400. This means that for the latter two outcomes, further research is likely to have an important impact in our confidence in the estimate of effect and may change it.

For environmental interventions, we rated the quality of evidence from continuous and dichotomous blood lead level outcomes as moderate to low. We downgraded the evidence quality by one level because of imprecision and one level for inconsistency because I^2 was high (blood lead level, continuous: 90%; blood lead level, dichotomous ≥ 15.0 µg/dL: 56%).

Potential biases in the review process

We judge the risk of potential biases in the review process to be low. Most of the authors of this update were new. To avoid any biases caused by changing authors, the first author of the original review remained on the team, and we set a high value on intense communication and exchange of information with the original team. To minimise the risk of bias caused by a revised search strategy during the course of this update, we searched all newly added databases without time limits, and not just from the date of the last search to 2016. Publication bias and selective outcome reporting are potential limitations of any systematic review. Although we searched for grey and unpublished literature, the extent and impact of reporting biases of this body of evidence is impossible to determine.

Agreements and disagreements with other studies or reviews

A previous review limited to low-cost, lead hazard control interventions, which included only 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 of 15.0 µg/dL or more (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 of 15.0 µg/dL or more.

Another systematic review assessed the effectiveness of interventions to reduce lead exposure through consumer products and drinking water (Pfadenhauer 2016). This review reported on

blood lead levels and could also not confirm the effectiveness of educational interventions.

On the surface, these results may appear to contradict observational studies that report a reduction in dust lead loadings and, on average, a decrease in children's blood lead levels (Clark 2004). However, 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 lead 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 5.0 µg/dL or higher following abatement were high: OR 11.18 (95% CI 2.80 to 44.16) for six-month old infants; OR 3.69 (95% CI 1.68 to 8.09) for 12-month old children; OR 1.79 (95% CI 1.07 to 2.99) for 18-month old children; and OR 1.18 (95% CI 0.79 to 1.76) for 24-month old children. These results indicate that the floor clearance levels used by the HUD grantees (less than 1.076 or 2.153 mg/m^2 (100.0 or 200.0 $\mu g/ft^2$)) are insufficient to protect children. This is not surprising; there is considerable evidence that dust lead levels under 0.108 mg/m^2 (10.0 $\mu g/ft^2$) are associated with a large increase in the risk of children having a blood lead level of more than 10.0 µg/dL (Lanphear 1996b; Lanphear 1998; Lanphear 2005b; Dixon 2009). Thus, even if lead hazard controls or renovation activities can be safely implemented, if we rely on empirically derived but obsolete dust clearance standards, the measures may actually increase young children's blood lead concentrations.

AUTHORS' CONCLUSIONS

Implications for practice

Based on a review of the current research, there is evidence that educational interventions are not effective in reducing blood lead levels in children. Dust control interventions may lead to little or no difference in blood lead levels (the quality of evidence was moderate to low, meaning that future research is likely to change these results). There is currently insufficient evidence that soil abatement or combination interventions reduce blood lead levels. No study reported on cognitive/neurobehavioural outcomes and adverse events. These patient-relevant outcomes would have been of great interest to draw conclusions for practice.

Implications for research

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

Trials that look at suitable interventions in low- and middle-income countries are also urgently required, as are studies in children in more affluent areas where lead exposure is often due to renovation rather than poor maintenance and hence may be more short-term and amenable to preventive interventions.

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* Indicates the major publication for the study

CHARACTERISTICS OF STUDIES

Characteristics of included studies *[ordered by study ID]*

Aschengrau 1998

Methods	**Study design**: RCT **Study location/setting**: Boston, USA. Urban area **Recruitment**: children were screened for lead from May 1993 to April 1995 **Follow-up**: 6 months from baseline **Intention-to-treat**: available case analysis **Power calculation**: performed to determine number of participants (required number not recruited)
Participants	**Eligibility criteria** 1. Resided in the city of Boston 2. Less than 4 years of age 3. Had venous blood lead level from 11.0 to 24.0 µg/dL 4. No history of lead poisoning or chelation therapy 5. Not expected to undergo chelation treatment 6. Lived on the premises for at least 3 months with no definite plans to move within the next 3 months 7. Lived in home with lead-based paint on at least 2 window sills and/or window wells, as determined by sodium sulphide tests 8. The home had not been previously de-leaded or received lead hazard reduction activities 9. The parents spoke English, Spanish, or Cape Verdean Creole 10. No other child in the home was already a study participant **Participation rate**: 63/402 (16%) enrolled, of which 41 were randomised (22 intervention, 19 control); 22 other participants at high risk were automatically assigned to the intervention and therefore were not considered in this Cochrane review **Reason for non-participation**: 163 unreachable; 64 unable to communicate due to language barriers; 112 refused to participate (demographic characteristics similar between participants and non-participants) **Analysis**: 24/41 (59%) for blood lead levels, 22/41 (54%) for household dust **Number of dropouts/withdrawals**: 17 blood lead levels, 19 household dust lead levels **Reasons for dropout/withdrawal**: children were excluded because no 6-month follow-up blood samples were taken, their homes received non-study environmental interventions, or they received chelation therapy (no specific numbers per reason were reported for the randomised children that dropped out) I**ntervention baseline characteristics** (available for n = 11)

Aschengrau 1998 *(Continued)*

- 36.4% boys, 63.6% girls
- Mean age 28.3 months
- Blood lead level 17.6 µg/dL

Control baseline characteristics (available for n = 13)

- 53.9% boys, 46.1% girls
- 23 months
- Blood lead level 16.3 µg/dL

Interventions	**Intervention (low technology lead hazard reduction)** 1. Remove lead dust 2. Loose paint chips 3. HEPA (high efficiency particulate air) vacuum 4. Parental education: demonstrating effective housekeeping techniques and sending monthly reminders with instructions to wash hard surface floors, window sills, and wells 5. Usual outreach and educational activities (1 home visit by an outreach worker to visually assess the home for lead hazards and to educate the caregiver about the causes and prevention of lead poisoning) **Control**: usual outreach and educational activities provided to both groups (1 home visit by an outreach worker to visually assess the home for lead hazards and to educate the caregiver about the causes and prevention of lead poisoning)
Outcomes	1. Blood lead level 6 months from baseline (venous blood sample) 2. Household dust lead levels
Notes	**Funding**: this research was supported by a co-operative agreement (Grant H64/CCH108235-03) with the Centers for Disease Control and Prevention (CDC), Atlanta, GA **Conflicts of interest**: none declared **Other comments** 1. Different baseline characteristics and small sample size 2. Inconsistent parental compliance with housekeeping 3. Several participants had non-study interventions and were excluded from analysis in the report

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 from correspondence with author: "open list"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "the subjects and the investigators interacting with the subjects knew which group they were assigned to" Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: "lab analysers were blinded"

Aschengrau 1998 *(Continued)*

Incomplete outcome data (attrition bias) Blood lead level	High risk	Comment: high attrition rate (41%) and high differential attrition. Attrition rate was 18% points higher in the intervention group than in the control group
Incomplete outcome data (attrition bias) Household dust lead level	High risk	Comment: high attrition rate (46%) and high differential attrition. Attrition rate was 27% points higher in the intervention group than in the control group
Selective reporting (reporting bias)	Unclear risk	Comment: insufficient information
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Boreland 2009

Methods	**Study design**: RCT **Study location/setting**: Australia. Mining community **Recruitment**: October 1994 to August 1996 **Follow-up**: 12 months from baseline **Intention-to-treat**: available case analysis **Power calculation**: performed to determine number of participants (required number not recruited)
Participants	**Eligibility criteria** 1. Children from a surveillance programme, aged 12 to 60 months 2. Blood lead level ≥ 15.0 µg/dL and ≤ 30.0 µg/dL **Participation rate**: 103/365 (28%). 365 were eligible for remediation, 117 enrolled in remediation program and 103 were eligible for randomisation (all children with a blood lead level ≥ 30.0 µg/dL were offered immediate home remediation, and therefore were not part of the randomisation). Of the 103 children, 90 were matched by age and blood lead level range and were randomised (45 intervention, 45 control) **Reason for non-participation**: 13 were unable to be adequately matched **Analysis**: 88/90 (98%) blood levels analysed **Number of dropouts/withdrawals**: 2 **Reasons for withdrawal**: no blood samples for either participant **Baseline data** available for all children randomised • Overall 42% males, 58% females • Mean age 3.5 years • Mean blood lead level 19.4 µg/dL Baseline characteristics not reported separately for intervention and control group
Interventions	**Intervention (home remediation work)** 1. Performed on intervention households and varied depending on assessment of need to provide each house with a "similar level of lead safety"

Boreland 2009 *(Continued)*

	2. Work may have included: ceiling dust removal, sealing of ceilings, paint stabilisation, replacement of floor coverings/windows and cleaning 3. Visiting families at home and providing them with information about minimising lead hazards **Control**: visiting families at home and providing them with information about minimising lead hazards
Outcomes	Blood lead level six months from baseline (venous blood sample) (Internal floor dust quintile only used to examine dose response effects)
Notes	**Funding**: Australian Government Department of Health and Ageing **Conflicts of interest**: none declared **Other comments** • 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

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "children were matched in pairs and then a coin tossed to see which would be the 'case' and have their home remediated first"
Allocation concealment (selection bias)	Unclear risk	Comment: insufficient information
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Comment: no information provided on blinding. However, we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: "lab analysers were blinded"
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: attrition rate was low (2%)
Selective reporting (reporting bias)	Low risk	Comment: 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	Comment: the study appears to be free from other sources of bias

Brown 2006

Methods	**Study design**: RCT **Study location/setting**: Rhode Island, USA. Urban area **Recruitment**: all children who were identified through routine blood lead testing as having venous blood lead levels 15.0 to 19.0 µg/dL, and reported to Rhode Island Department of Health between July 1999 and June 2002, were referred to the study

Brown 2006 *(Continued)*

	Follow-up: 12 months from baseline **Intention-to-treat analysis**: available case analysis **Power calculation**: performed to determine number of participants (required number recruited)
Participants	**Eligibility criteria** • Venous blood lead level 15.0 to 19.0 µg/dL • 28 months of age or younger • Family spoke English or Spanish **Participation rate**: 175/241 (73%) consented to participate and were randomised (92 intervention, 83 control) **Reason for non-participation**: 66 refused due to work and school responsibilities (no breakdown in figures per reason reported) **Analysis**: 145/175 (83%) analysed blood lead level, 153/175 floor dust lead levels **Number of dropouts/withdrawals**: 30 blood lead level, 22 floor dust lead levels **Reasons for dropout/withdrawal**: 9 children moved away, 2 were lost to follow-up, 9 parents refused participation of their child during the study, 2 refused first and all subsequent visits. No reason for dropout/withdrawal was reported for 8 children **Intervention baseline characteristics** (available for n = 90) • % boys/girls not reported • Mean age 19.1 months • Mean blood lead level 16.5 µg/dL **Control baseline characteristics** (n = 83) • boys/girls not reported • Mean age 18.8 months • Mean blood lead level 16.3 µg/dL
Interventions	**Intervention**: parental education (with nursing care plan) via 5 home visits during 1-year period. Nurses collected interior dust and soil samples, evaluated parent-child interaction, identified occupational or recreational exposure to lead sources and other factors thought to influence lead exposure. The nursing care plan directed parent teaching and other services **Control**: children received customary care: one to two educational visits by outreach workers. These visits focused on standard health education about lead poisoning and its prevention but did not include environmental sampling, education tailored to individual circumstances, or assessment or parent-child interaction
Outcomes	1. Blood lead level 12 months from baseline (venous blood sample) 2. Household floor and window dust lead (Questionnaires on lead exposures) (Parental-infant interaction scale)
Notes	**Funding**: sponsored in part by CDC (grant TS 275 14/14) and Maternal and Child Health Bureau (grant 5T76 MC 00001; formerly MCJ201) **Conflicts of interest**: none declared

Risk of bias

Bias	Authors' judgement	Support for judgement

Brown 2006 *(Continued)*

Random sequence generation (selection bias)	Low risk	Quote from report: "[r]andom numbers table was used to assign cases to either the intervention or the comparison group, sequentially"
Allocation concealment (selection bias)	Low risk	Quote from report: "group assignments were sealed into envelopes and unknown to either study personnel or the families until after parental consent was obtained"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from report: "the nurses who provided follow up to comparison group children were blinded and nurses that provided care to intervention group were not blinded" Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from report: "venous blood samples were collected by children's pediatric health care providers"
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: the overall attrition rate (17%) was acceptable. The attrition rates in intervention (18%) and control group (15%) were similar
Incomplete outcome data (attrition bias) Household dust lead level	Low risk	Comment: the overall attrition rate was acceptable (13%). The attrition rates in intervention (12%) and control group (13%) were similar
Selective reporting (reporting bias)	Low risk	Comment: 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 the investigator
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Campbell 2011

Methods	**Study design**: RCT **Study location/setting**: Philadelphia, USA. Urban area **Recruitment**: children were recruited from urban outpatient practices located in low-income neighbourhoods of Philadelphia **Follow-up**: 24 months from baseline **Intention-to-treat analysis**: available case analysis **Power calculation**: performed to determine number of participants (required number recruited)
Participants	**Eligibility criteria** 1. Residing in Philadelphia County 2. Children speak English or Spanish 3. Home that was judged to be in a condition enabling remediation 4. No history of elevated blood lead levels 5. No former participation in the Lead Safe Babies program 6. No child of the family has ever received services from the Childhood Lead Poisoning Prevention Program of the Philadelphia Department of Public Health

Campbell 2011 *(Continued)*

	Participation rate: 314/314 (100%) newborn children enrolled and randomised (154 intervention, 160 control); 310/310 (100%) households enrolled **Reasons for non-participation**: NA **Analysis**: 279/314 (89%) blood lead levels analysed at 12 months of age; 110/306 (36%) household dust analysed at 12 months. No information on number of children for whom blood lead levels were analysed at 24 months **Number of dropout/withdrawal**: 35 (blood lead levels), 196 (household dust lead) **Reasons for dropout/withdrawal**: for 35 children no venous specimen was taken (nor reasons specified); for 196 no household dust lead level was measured because of problems finding participants who changed address or phone numbers, non-compliance with study visits, lack of approval by family members (no breakdown in specific numbers per reasons reported) **Intervention baseline characteristics** (n = 154): 53.2% boys, 46.8% girls **Control baseline characteristics** (n = 160): 51.2% boys, 48.8% girls Mean age and mean blood lead level only reported for both groups combined: 11 months and 2.7 µg/dL, respectively
Interventions	**Intervention** 1. Standard lead poisoning prevention education plus additional extensive education regarding maintaining home in lead-safe condition and home visits from outreach workers at baseline, 6 and 12 months. The additional education was compiled in a 22-page handbook. 2. Cleaning materials provided (MEG: maintenance education group) **Control**: standard lead poisoning prevention education, not described in more detail (SEG: standard education group)
Outcomes	1. Blood lead levels at 12 and 24 months (venous blood sample) 2. Housing lead dust levels at 12 months (Parental Knowledge Assessment)
Notes	**Funding**: Housing and Urban Development (HUD) Lead Technical Studies Grant **Conflicts of interest**: none declared **Other comments**: a matched comparison group was included in results of the paper, receiving community standard for prevention of elevated blood lead levels. This group was not part of the randomisation process and therefore was not integrated in this Cochrane review

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "randomized blocks using computer-generated random numbers"
Allocation concealment (selection bias)	Unclear risk	Quote from correspondence with author: "study coordinator selected next card in the random sequence to randomise that family"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "once the randomization occurred they were told of their assignment. The outreach workers who performed the randomization were made aware of the assignment category, as well."

Campbell 2011 *(Continued)*

		Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Unclear risk	Quote from correspondence with author: "the samples were sent to 2 different analytic labs during the course of the 3-year study period, and their job is to analyze BLLs. They were blinded to status". "The outreach workers collecting the dust wipe samples knew the household assignment" - this might have had an influence on household dust levels
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: attrition rate for blood lead level was acceptable (11%). No information on dropout rates in either study arm
Incomplete outcome data (attrition bias) Household dust lead level	High risk	Comment: for the outcome, household dust, attrition rate was very high (64%). No information on dropout rates in either study arm
Selective reporting (reporting bias)	Low risk	Comment: 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 the investigator
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Charney 1983

Methods	**Study design**: quasi-RCT (even/odd clinic number assignment) **Study location/setting**: Baltimore, USA. Recruited from a lead poisoning clinic **Recruitment**: children were recruited in July-October 1981 as they appeared for regular blood lead monitoring in a lead poisoning clinic **Follow-up**: 12 months from baseline **Intention-to-treat**: unclear **Power calculation**: performed to determine number of participants (unclear if required number recruited)
Participants	**Eligibility criteria** 1. Blood lead level 30.0 µg/dL to 49.0 µg/dL 2. Children from lead poisoning clinic in Baltimore 3. Between the age of 15 to 72 months 4. Lived at present address for at least 6 months **Participation rate**: 78/78 (100%) children enrolled and randomised (22 intervention, 56 control) **Reasons for non-participation**: NA **Analysis**: 49/78 (63%) analysed **Number of dropouts/withdrawals**: 29 **Reasons for dropout/withdrawal**: children moved, spent considerable time with relatives in another household, not home for visits (no specific numbers per reason reported) **Intervention baseline characteristics** (available for n = 14) • 36% boys, 64% girls

Charney 1983 *(Continued)*

- Mean age 45 months
- Mean blood lead level 38.0 µg/dL

Control baseline characteristics (available for n = 35)

- 51% boys, 49% girls
- Mean age 43 months
- Mean blood lead level 38.0 µg/dL

Interventions	**Intervention** 1. Dust control team to wet mop all rooms twice per month 2. Parental education to clean more frequently over 12-month period 3. Paint stabilisation **Control**: routine advice dust control by mopping given at clinic plus paint stabilisation
Outcomes	1. Blood lead level 12 months from baseline (venous blood sample) 2. Household floor dust (only reported in intervention group)
Notes	**Funding**: supported from Department of Housing and Urban Development (HUD) Grant **Conflicts of interest**: none declared

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	High risk	Quote from report allocation method alternate based on "even or odd clinic number"
Allocation concealment (selection bias)	High risk	Comment: not used
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "personnel was not blinded. Participants were not aware of the existence of another study group" Comment: we rated this domain as low risk, because blood lead levels won't be influenced by the participants knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Comment: blinding of outcome assessors (laboratory)
Incomplete outcome data (attrition bias) Blood lead level	Unclear risk	Comment: attrition rate was quite high (37%), but there was no difference in attrition rates between study arms (37% in both groups)
Selective reporting (reporting bias)	Unclear risk	Comment: insufficient information
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Farrell 1998

Methods	**Study design**: cluster-RCT, by neighbourhood **Study location/setting**: Baltimore, USA. Urban neighbourhoods

Farrell 1998 *(Continued)*

	Recruitment: children from 2 neighbourhoods were recruited beginning in 1988 **Follow-up**: 1 year **Intention-to-treat analysis**: no, due to exclusion of those not adhering to the study protocol (n = 226) **Power calculation**: performed to determine number of participants (required number recruited)
Participants	**Eligibility criteria for neighbourhoods** 1. Sufficient children to test hypothesis 2. Areas of exposed soil around homes 3. Pre-1950 urban housing away from major industries or highways 4. Comparable demographics 5. Moderate risk for lead exposure **Eligibility criteria for children** 1. 6 months to 6 years of age 2. Living in the same house (in selected neighbourhood) for at least 3 months and family was not planning to move **Participation rate**: NA as community recruitment; 408 children (212 intervention, 196 control) in 263 houses randomised **Reasons for non-participation**: NA **Analysis**: 182/408 (121/263 households) (45%) analysed **Number of dropouts/withdrawals**: 226 **Reasons for dropouts/withdrawals**: children did not complete the study protocol (no specific reasons reported) **Intervention baseline characteristics** (n = 212): mean blood lead level 11.0 µg/dL **Control baseline characteristics** (n = 196): mean blood lead level 10.9 µg/dL No information on sex or age of included children
Interventions	**Intervention** 1. Soil abatement consisting of removing the top 6 inches (15 cm) of soil, replacing it with lead-free soil, then sodding or seeding, all within 1 week of exterior paint stabilisation 2. External paint stabilisation as means of preventing soil recontamination **Control**: external paint stabilisation as means of preventing soil recontamination
Outcomes	1. Blood lead level at 2 years from baseline (venous blood sample) 2. (Soil lead levels)
Notes	**Funding**: US Environmental Protection Agency (EPA) **Conflicts of interest**: none declared **Other comments**: 1. Baseline soil lead levels lower than hypothesised with 54% > 1000 parts per million 2. No internal household interventions 3. Adjacent properties not abated
Risk of bias	

Farrell 1998 *(Continued)*

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "coin toss"
Allocation concealment (selection bias)	Unclear risk	Comment: insufficient information
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Comment: no information provided on blinding. We rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: "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	High risk	Comment: attrition rate was very high (55%). Attrition was similar in both groups (intervention group 53%, control group 58%)
Selective reporting (reporting bias)	Unclear risk	Comment: insufficient information
Other bias	Unclear risk	Comment: risk of bias due to cluster randomisation Used neighbourhood clusters, and it was unclear how analysis was performed as data were not available - unclear if there is unit of analysis bias. We assessed risk of recruitment bias and bias risk due to baseline imbalance as low, since baseline characteristics were comparable and randomisation was achieved by coin toss.

Hilts 1995

Methods	**Study design**: cluster-RCT, by household (in blocks of 6 stratified by area and blood lead level) **Study location/setting**: British Columbia, Canada. Higher lead risk area (active smelter) **Recruitment**: blood screen in 1992 **Follow-up**: 10 months from baseline **Intention-to-treat** available case analysis **Power calculation**: performed to determine number of participants (required number recruited)
Participants	**Eligibility criteria** 1. Households in the study area with children under 72 months of age 2. No plans of moving 3. Living at the present address for > 1 month **Participation rate**: 122/176 eligible households (69%) enrolled and randomised (122 children; 61 intervention, 61 control) **Reasons for non-participation**: 54 households were not interested in participating **Analysis**: 111/122 (99%) analysed **Number of dropouts/withdrawals**: 11

Hilts 1995 *(Continued)*

	Reasons for dropouts/withdrawals: moved house or did not provide a final blood sample (no specific numbers per reasons reported) **Intervention baseline characteristics** (available for n = 55) • Mean age 32.9 months • Mean blood lead level 11.9 µg/dL **Control baseline characteristics** (available for n = 56) • Mean age 31.9 months • Mean blood lead level 11.3 µg/dL • No information on sex of children
Interventions	**Intervention**: 1. HEPA vacuuming (7 times in a 10-month period) 2. Routine advice regarding maintenance and general lead educational materials provided **Control**: routine advice regarding maintenance and general lead educational materials provided
Outcomes	1. Blood lead level 10 months from baseline (venous blood sample) 2. Floor dust and lead levels
Notes	**Funding**: grants to the Trail Community Lead Task Force by: BC Ministry of Health, BC, Ministry of Environment, Lands and Parks, Cominco Limited and City of Trail **Conflicts of interest**: none declared **Other comments**: potential for unit of analysis error

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "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 from correspondence with author: "done in central office"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "participants and personnel were not blinded as to treatment allocation" Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: "blood specimen collector and lab personnel did not know of group assignments", "lab personnel analysing the carpet dust samples were not aware of group assignment"
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: low attrition rate (1%)
Incomplete outcome data (attrition bias) Household dust lead level	Low risk	Comment: low attrition rate (1%)

Hilts 1995 *(Continued)*

Selective reporting (reporting bias)	Low risk	Comment: 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 the investigator
Other bias	Unclear risk	Comment: risk of bias for cluster-randomised studies Used clusters of 6 households but used individuals as unit for analysis and therefore introduced a unit of analysis error. We assessed risk of recruitment bias and bias risk due to baseline imbalance as low, since baseline characteristics were comparable and randomisation was achieved by coin toss.

Jordan 2003

Methods	**Study design**: RCT **Study location/setting**: Minneapolis, USA. Urban area **Recruitment**: recruited by door knocking and community information **Follow-up**: 3 years from baseline **Intention-to-treat**: unclear **Power calculation**: performed to determine number of participants (unclear if required number recruited)
Participants	**Eligibility criteria**: pregnant women and mothers of young infants from the Phillips Neighborhood (economically disadvantaged, ethnically diverse neighbourhood) **Participation rate**: NA as community recruitment **Reasons for non-participation**: NA **Analysis**: 607 children (299 intervention, 308 control) randomised, 378 (62%) analysed **Number of dropouts/withdrawals**: 229 **Reasons for dropouts/withdrawal**: no reasons stated No information if baseline data were available for all randomised children 1. According to the authors, there was no difference in baseline characteristics between treatment groups (no table presented, no detailed information on sex or age) 2. Mean blood lead level less than 10.0 µg/dL
Interventions	**Intervention**: 1. Intensive educational intervention: 20 bi-weekly, culturally specific educational session by peer leaders provided individually and 3-monthly boosters until child was 3 years of age 2. Routine state health brochures about lead, home assessment for lead contamination, and feedback about home inspections **Control**: routine state health brochures about lead, home assessment for lead contamination, and feedback about home inspections
Outcomes	Blood lead level (capillary until 12 months, venous > 12 months) 3 years from baseline
Notes	**Funding**: supported by Grant MCJ 270801 from the Maternal and Child Health Bureau and Grant U67/CCU510771 from the CDC **Conflicts of interest**: authors declare they have no competing financial interests

Jordan 2003 *(Continued)*

Other comments

1. Dichotomous data
2. All participants given financial incentive

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "Random number generator"
Allocation concealment (selection bias)	Low risk	Quote from correspondence with author: "Central office"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "no blinding of participants or personnel" Comment: we rated this domain as being at low risk because blood lead levels are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: outcome assessors "laboratory" blinded
Incomplete outcome data (attrition bias) Blood lead level	High risk	Comment: attrition was quite high (38%), no information on attrition rates in both groups were given, so we rated it as being at high risk of bias
Selective reporting (reporting bias)	High risk	Comment: results on household dust lead outcomes were not reported although they were measured. No information from the author on these outcomes could be obtained
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Lanphear 1996a

Methods	**Study design**: RCT **Study location/setting**: Rochester, NY, USA. Community-based trial in urban area **Recruitment**: baseline data collected between August 1993 and November 1993; follow-up samples collected between April 1994 and June 1994 **Follow-up**: 7 months from baseline **Intention-to-treat** available case analysis **Power calculation**: not performed to determine number of participants
Participants	**Eligibility criteria**: families with children who participated in the Lead-in Dust study (a cross-sectional study to assess relationship of lead-contaminated house dust and urban children's blood lead levels) **Participation rate**: 104/205 (50%) enrolled (no significant difference in those refused) and randomised (57 intervention, 47 control) **Reasons for non-participation**: 101 not interested in participating **Analysis**: blood lead level 96/104 (91%), non-carpet floor dust lead level 70/104 (67%), carpet floor dust lead level 60/104 (57%)

Lanphear 1996a *(Continued)*

Number of dropouts/withdrawals: 8 (blood lead level), 34 (non-carpet floor lead dust level), 44 (carpet floor lead dust level)

Reasons for dropout/withdrawal: 2 refused second blood tests, 3 had moved outside of the area, 2 had no time, and 1 was lost to follow-up; for others of whom floor dust lead levels were not available and no reasons were specified

Intervention baseline characteristics (n = 57)

- Mean age 19.8 months
- Mean blood lead level 6.6 µg/dL

Control baseline characteristics (n = 47)

- Mean age 20.4 months
- Mean blood lead level 6.8 µg/dL

No information on sex of participants

Interventions	**Intervention** 1. Trained interviewer emphasised the importance of dust control for reducing children's exposure to lead, provided them with cleaning supplies, gave a demonstration of how to clean and instructed families how and when to clean 2. Families were given a colouring book that described lead poisoning and its prevention 3. Families were given a brochure on lead poisoning and its prevention **Control**: families with children who participated in the Lead-in Dust study (a cross-sectional study to assess relationship of lead-contaminated house dust and urban children's blood lead levels)
Outcomes	1. Blood lead level at 7 months from baseline (venous blood sample) 2. Household floor and window dust lead
Notes	**Funding**: grant NYLPR002-94 from the US Department of Housing and Urban Development (HUD), the National Center for Lead-Safe Housing, and Institutional National Research Service Award 2T-32 PE-12002 from the Bureau of Health Professions, Health Resources and Services Administration, US Public Health Service, Department of Health and Human Services **Conflicts of interest**: none declared

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "computer random number generator"
Allocation concealment (selection bias)	Low risk	Quote from correspondence with author: "sealed opaque envelopes"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "personnel and participants were blinded"
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: "yes, blood lead specimen collectors and analysers were blinded to group allocation"

Lanphear 1996a *(Continued)*

Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: attrition rate for blood lead level was low (9%)
Incomplete outcome data (attrition bias) Household dust lead level	Low risk	Comment: attrition rate was acceptable (33%) and similar between groups (intervention group 32%, control group 36%)
Selective reporting (reporting bias)	Low risk	Comment: the study protocol is available and all of the study's pre-specified outcomes are reported in the pre-specified way
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Lanphear 1999

Methods	**Study design**: RCT (also non-study control to rule out Hawthorne effect) **Study location/setting**: Rochester, NY, USA. Urban area **Recruitment**: identified by birth data from hospitals, inner-city clinics and the Department of Social Services and Health - families were called to determine eligibility via interviews **Follow-up**: 42 months from baseline **Intention-to-treat**: available case analysis **Power calculation**: performed to determine number of participants (required number recruited)
Participants	**Eligibility criteria** 1. Living in Rochester, NY 2. No plans to relocate in the next 3 months 3. Children older than 5 months but less than 7 months of age at baseline visit **Participation rate**: 275/429 (64%) enrolled and randomised (140 intervention, 135 control) **Reasons for non-participation**: not interested in participating **Analysis**: 245/275 (89%) and 189/275 (69%) analysed at 24 and 48 months, respectively **Number of dropouts/withdrawals**: 30 at 24 months, 86 at 48 months **Reasons for dropout/withdrawal**: lost to follow-up **Intervention baseline data** (n = 140) • Mean age 6.68 months • Mean blood lead levels 2.8 µg/dL **Control baseline data** (n = 135) • Mean age 6.65 months • Mean blood lead level 2.9 µg/dL No information on sex of participants
Interventions	**Intervention** 1. Up to 8 visits by dust control advisors, cleaning equipment and supplies in 24-month period. Dust control advisors were trained to use educational model developed specifically for home visitation 2. Baseline 4 home visits by trained interviewer to collect data

Lanphear 1999 *(Continued)*

	Control 1. Baseline 4 home visits by trained interviewer to collect data 2. Families in the control group did not receive any lead exposure prevention education or intervention
Outcomes	1. Blood lead level measured at 6 months (baseline), 12-, 18-, 24-, 36- and 48-months (venous blood sample) 2. Household floor and window dust lead
Notes	**Funding**: CDC Grant (U67/CCU210773) and an Institutional National Research Service Award (#2T-32 PE-12002) from the Bureau of Health Professions, Human Resources and Services Administration, Public Health Service, Department of Health and Human Services **Conflicts of interest**: none declared

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "random number generator"
Allocation concealment (selection bias)	Low risk	Quote from correspondence with author: "sealed opaque envelopes"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "personnel and participants were blinded"
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Quote from correspondence with author: "yes, blood lead specimen collectors and analysers were blinded to group allocation", "environmental technicians and interviewers blind to group assignment"
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: attrition rate at 12 months was 11%, at 24 months it was 31% - both acceptable. Attrition rate between groups was similar
Incomplete outcome data (attrition bias) Household dust lead level	Low risk	Comment: attrition rate at 12 months was 11%, at 24 months it was 31% - both acceptable. Attrition rate between groups was similar
Selective reporting (reporting bias)	Low risk	Comment: the study protocol is available and all of the study's pre-specified outcomes are reported in the pre-specified way
Other bias	Low risk	Comment: the study appears to be free of other sources of bias

Rhoads 1999

Methods	**Study design**: RCT **Study location/setting**: Jersey City, NY, USA **Recruitment**: families that responded to posters and door hangers or were referred to the study by the municipal lead poisoning prevention programme, local health care providers, or word of mouth. **Follow-up**: 12 months from baseline

Rhoads 1999 *(Continued)*

	Intention-to-treat: unclear **Power calculation**: performed to determine number of participants (required number not recruited)
Participants	**Eligibility criteria** 1. Children aged 6 months to 3 years 2. Responsible adult had to speak English or Spanish 3. Presence of lead paint in the home 4. Home had to be in a state that could be cleaned effectively (not structural disrepaired) 5. no evidence of illicit drug use, firearms, or other major staff safety concerns 6. index child was not in regular day care **Participation rate**: 113/147(77%) enrolled and randomised (56 intervention, 57 control) **Reasons for non-participation**: 7 could not be re-contacted or refused to allow a baseline blood lead sample to be drawn, 27 were not interested in participating. **Analysis**: 99/113 (87%) analysed for blood lead levels, 95/113 (84%) analysed for floor wipes, 76/113 (67%) analysed for sill wipes, 49/113 (43%) analysed for vacuum **Number of dropouts/withdrawals**: 14 blood lead level, 18 to 64 household dust lead levels **Reasons for dropout/withdrawal**: because of frequent moves and changing circumstances of the enrolled families, it was not possible to draw final blood samples from 14 children. No explanation for missing data on household dust lead levels stated **Intervention baseline characteristics** (available for n = 46) • Mean age 1.7 years • Mean blood lead level 12.4 µg/dL **Control baseline characteristics** (available for n = 53) • Mean age 1.6 years • Mean blood lead level 11.6 µg/dL No information on sex of children
Interventions	**Intervention** 1. Bi-weekly assistance with household cleaning (HEPA vacuum and wet mopping) by community staff members for 1 year. Visits usually lasted 2 hours. 2. Offer to attend 4-5 educational sessions a year about lead prevention **Control** 1. Accident prevention group given household safety items, but no assistance with household cleaning and no special education on lead prevention during visits 2. Offer to attend 4-5 educational sessions a year about lead prevention
Outcomes	1. Blood lead level 12 months from baseline 2. Household dust lead levels 3. Maternal lead knowledge
Notes	**Funding**: work was supported by Co-operative Agreement CR820235 from the Environmental Protection Agency, by an Interagency Agreement from the National Institute for Child Health and Human Development, National Institutes of Health, to US Environmental Protection Agency, by Grant 18152 from the Robert Wood Johnson Foundation, and by Grant ES-05022 from the National Institute of Environmental Health Sciences. **Conflicts of interest**: none declared

Rhoads 1999 *(Continued)*

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "permutated blocks of varying length"
Allocation concealment (selection bias)	Low risk	Quote from correspondence with author: "sealed envelopes"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "it was not possible to blind participants or field personnel to the assignments since one group had cleaning teams come to their homes and the other group did not" Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Comment: outcome assessors were blinded
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: low attrition rate (12%)
Incomplete outcome data (attrition bias) Household dust lead level	Unclear risk	Comment: for the outcome household dust, attrition rate was higher and reasons for missing data were not available
Selective reporting (reporting bias)	Unclear risk	Comment: insufficient information
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

Sterling 2004

Methods	**Study design**: RCT **Study location**: Missouri, USA. Former lead mining areas with large amounts of lead mining waste **Recruitment**: screenings in health department lead clinics; women, infants, and children's clinics; day-care centres; door-to-door screening;and health fairs **Follow-up**: 9 months **Intention-to-treat**: only cases with all quarterly measurements were included in analysis **Power calculation**: performed to determine number of participants (unclear if required number recruited)
Participants	**Eligibility criteria** 1. Children 6-72 months of age 2. Blood lead level 10.0 to 20.0 µg/dL 3. For 1 of the 2 counties, the households were required to be below the medium income level for the area **Participation rate**: 101/134 (75%) randomised (34 intervention one, 35 intervention two, 32 control)

Sterling 2004 *(Continued)*

Reasons for non-participation: no reasons for non-participation provided
Analysis: blood lead level 39/101 (39%), household lead dust 34/101 (34%) analysed

Number of dropouts/withdrawals: 62 blood lead level, 67 household lead dust level
Reasons for dropout/withdrawal: no reasons for dropouts provided

Intervention one baseline characteristics (n = 34)

- 59% boys, 41% girls
- Mean age 2.8 years
- Mean blood lead level 12.8 µg/dL

Intervention two baseline characteristics (n = 35)

- 49% boys, 51% girls
- Mean age 3.6 years
- Mean blood lead level 12.7 µg/dL

Control baseline characteristics (n = 32)

- 47% boys, 53% girls
- Mean age 2.8 years
- Mean blood lead level 12.7 µg/dL

Interventions	**Intervention 1**: 1. A standard health education session on lead exposure reduction activities, given by a nurse educator, a letter reporting the results of the environmental lead assessment of the home, generic educational information in the form of pamphlets produced by state and federal agencies 2. Three times quarterly educational home visit by nurse (providing education on hygiene, nutrition, blood-lead screening, house cleaning and providing cleaning supplies) and 6 personalised newsletters over 9-month period **Intervention 2**: as intervention 1, plus three x quarterly professional cleans with wet mopping, HEPA (high efficiency particulate air) and carpet shampooing **Control**: standard health education session on lead exposure reduction activities, given by a nurse educator, a letter reporting the results of the environmental lead assessment of the home, generic educational information in the form of pamphlets produced by state and federal agencies
Outcomes	1. Blood lead level at 3, 6 and 9 months from baseline (not stated if venous or capillary sample) 2. Household lead dust levels until 9 months from baseline (only presented graphically
Notes	**Funding**: not stated **Conflict of interests**: none declared

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Unclear risk	Comment: method of randomisation unknown
Allocation concealment (selection bias)	Unclear risk	Comment: unknown
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "no blinding of participants or researchers occurred"

Sterling 2004 *(Continued)*

		Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Comment: outcome analysers (laboratory) blinded
Incomplete outcome data (attrition bias) Blood lead level	High risk	Comment: high attrition rate (61%). Reasons for missing data not available
Incomplete outcome data (attrition bias) Household dust lead level	High risk	Comment: high attrition rate (66%). Reasons for missing data not available
Selective reporting (reporting bias)	Unclear risk	Comment: insufficient information
Other bias	Unclear risk	Comment: no information on funding provided

Wasserman 2002

Methods	**Study design**: RCT **Study location/setting**: Florida, USA **Recruitment**: caregivers selected from clients enrolled in Broward County MediPass (Medicaid) who selected Children's Diagnostic and Treatment Center as their health care provider **Follow-up**: 4 months from baseline **Intention-to-treat** : unclear **Power calculation**: not performed to determine number of participants
Participants	**Eligibility-criteria** 1. Aged 1-3 years 2. MediPass (Medicaid) as their insurance **Participation rate**: 63/63 (100%) children randomised (32 intervention, 31 control) **Reasons for non-participation**: NA **Analysis**: 50/63 (79%) analysed **Number of dropouts/withdrawals**: 13 **Reasons for dropout/withdrawal**: reasons not specified **Intervention baseline characteristics** (available for n = 28) • Mean age 23.5 months • Mean blood lead level 4.5 µg/dL **Control baseline characteristics** (available for n = 22) • Mean age 21.5 months • Mean blood lead level 2.6 µg/dL

Wasserman 2002 *(Continued)*

Interventions	**Intervention**: education session at clinic consisting of print-based module written by the researcher and used as the basis of parental lead education, a video used to show methods parents could use in the home to prevent lead poisoning, and brochure highlighting the risks of childhood lead exposure including factors that affect the home environment, behaviours that mitigate risk, and the need for proper nutrition at first clinic **Control**: education session as described above at second clinic (wait-list control)
Outcomes	1. Blood lead level at 3-4 months from baseline (venous blood sample) 2. Parental knowledge - Chicago Lead Knowledge Test
Notes	**Funding**: not stated **Conflicts of Interest**: none declared

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "random list of numbers"
Allocation concealment (selection bias)	Low risk	Quote from report: "assigned by central office"
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "Personnel was not blinded. Participants were unaware of the existence of the comparison group" Comment: we rated this domain as low risk, because blood lead levels are unlikely to be influenced by participants' knowledge about treatment allocation
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Comment: blinding of outcome assessors (laboratory)
Incomplete outcome data (attrition bias) Blood lead level	High risk	Comment: attrition rate (21%) was acceptable. However, attrition rate was much higher in control group (30%) than in intervention group (12%). Reasons for dropouts not specified
Selective reporting (reporting bias)	Low risk	Comment: 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 the investigator
Other bias	Unclear risk	Comment: no information on funding provided

Weitzman 1993

Methods	**Study design**: RCT **Study location/setting**: Boston, USA. Urban neighbourhoods with a high incidence of childhood lead poisoning and high soil lead levels **Recruitment**: screening efforts between January and June 1989 **Follow-up**: 11 months from baseline **Intention-to-treat**: available case analysis **Power calculation**: performed to determine number of participants (required number recruited)

Weitzman 1993 *(Continued)*

Participants	**Eligibility criteria** 1. Up to 4 years of age 2. Finger-stick blood lead level of 0.48 to 0.96 µmol/L 3. Chipping or peeling paint did not exceed 30% of the total surface area on the exterior walls of the child's home or exceed 40% on the walls of abutting premises 4. Premise had a yard of at least 0.9 m^2 composed of dirt or grass, or both, that was accessible to the child and the mean or median surface soil lead level among samples taken near the house was at least 1500 parts per million 5. The child resided in a dwelling with ≤ 8 residential units and was mobile 6. Child had never been lead poisoned 7. Family resided on the premises for at least 3 months and had no plans to move within the 3 months after enrolment **Participation rate**: 152/236 (64%) children randomised (54 intervention, 51 control group 1, 47 control group 2) **Reasons for non-participation**: children who had venous blood lead levels above 1.16 µmol/L were excluded because they met the former definition of lead poisoning and were likely to undergo medical and environmental interventions that could obscure changes associated with the study interventions (not specified whether all 84 children had high blood lead levels or if other reasons account for non-participation) **Analysis**: 149/152 (98%) analysed **Number of dropouts/withdrawals**: 3 **Reasons for dropouts/withdrawals**: no specific reasons for dropouts stated **Intervention baseline characteristics** (available for n = 52) • 60% boys, 40% girls • Mean age 30.5 months • Mean blood lead level 0.6 µg/dL **Control group 1 baseline characteristics** (n = 51): • 49% boys, 51% girls • Mean age 31.4 months • Mean blood lead level 0.6 µg/dL **Control group 2 baseline characteristics** (n = 47): • 51% boys, 49% girls • Mean age 33.1 months • Mean blood lead level 0.6 µg/dL
Interventions	Phase I only **Intervention** 1. Soil abatement from yard (15 cm layer of topsoil was removed and replaced with 20 cm of clean soil) 2. Interior dust abatement (high-efficiency particulate air filter vacuuming and wiping surfaces with a wet cloth or an oil-treated rag for furniture, floors, walls, woodwork, windows, furniture surfaces were cleaned) 3. Loose interior paint removal (vacuuming the loose paint areas with HEPA, washing loose paint areas, painting window wells with primer) **Control A**

Weitzman 1993 *(Continued)*

	1. Interior dust abatement (high-efficiency particulate air filter vacuuming and wiping surfaces with a wet cloth or an oil-treated rag for furniture, floors, walls, woodwork, windows, furniture surfaces were cleaned) 2. Loose interior paint removal (vacuuming the loose paint areas with HEPA, washing loose paint areas, painting window wells with primer) **Control B** 1. Loose interior paint removal (vacuuming the loose paint areas with HEPA, washing loose paint areas, painting window wells with primer)
Outcomes	1. Blood lead levels 11 months from baseline (venous blood sample) 2. Household dust levels (only reported as percentage of households that remained at a lower dust level than at baseline after 4 to 5 weeks and after 33 weeks after the intervention)
Notes	**Funding**: grant X001822-01-06 from Environmental Protection Agency, Washington, DC **Conflicts of interest**: none declared **Other comments**: phase I and phase II of Boston Lead-In-Soil trial performed but phase II excluded as no controls

Risk of bias

Bias	Authors' judgement	Support for judgement
Random sequence generation (selection bias)	Low risk	Quote from correspondence with author: "computer-based random number generator"
Allocation concealment (selection bias)	High risk	Comment: allocation performed by 1 staff member but not actively concealed from other investigators enrolling participants
Blinding of participants and personnel (performance bias) Household dust measures	Low risk	Quote from correspondence with author: "impossible for participants or personnel to be blinded to treatment allocation" Comment: we rated this domain as low risk, because blood lead levels and household dust lead measures are unlikely to be influenced by participants' knowledge about treatment allocation.
Blinding of outcome assessment (detection bias) All outcomes	Low risk	Comment: outcome assessors (laboratory analysers) were blinded
Incomplete outcome data (attrition bias) Blood lead level	Low risk	Comment: attrition rate was low (2%)
Incomplete outcome data (attrition bias) Household dust lead level	Low risk	Comment: attrition rate was low (2%)
Selective reporting (reporting bias)	Unclear risk	Comment: insufficient information
Other bias	Low risk	Comment: the study appears to be free from other sources of bias

BLL: blood lead level; **CDC**: Centers for Disease Prevention and Control (USA); **HEPA**: high efficiency particulate air; **MEG**: maintenance education group; **NA**: not available; **SEG**: standard education group.

Characteristics of excluded studies *[ordered by study ID]*

Study	Reason for exclusion
Aschengrau 1994	No control group used for phase II
Boreland 2006	Only before-and-after intervention assessment
Butterfield 2011	Outcome was parents' self-efficacy and pre-caution adoption (not relevant for this review). Outcomes relevant for this Cochrane review not measured in this study
Dixon 2012	Observational study
Dugbatey 2005	Outcome (blood lead levels) only measured in mothers, not children. Outcomes relevant for this Cochrane review not measured in this study
EPA 1996	Retrospective data collection on 2 groups not randomly assigned
EPA 1997	Historical control group with no randomisation used
Farfel 1990	Observational study
Feit 2014	Qualitative study with semi-structured interviews
Maharaj 2007	Conference abstract arguing the link between lead poisoning and asthma
Malcoe 2004	Observational study
Marlowe 2001	Outcome measured using hair lead levels. Outcomes relevant for this Cochrane review not measured in this study
NCT00000104	Observational study
NCT00011674	Observational study
Omidpanah 1998	Control and Intervention groups from 2 different study bases
Phoenix 2013	Before-and-after design without comparison group
Pollak 2002	Historical control group with no randomisation used
Taha 1999	Retrospective control with no randomisation used
Thomas 2013	Analysis of database records and qualitative research
Untimanon 2012	Compared contamination modes, not prevention of lead exposure
Whitehead 2014	Cross-sectional study
Wilson 2015	Before-and-after design without comparison group
Yeoh 2014	Systematic review. Former version of this Cochrane review
Zimmermann 2006	No intervention of interest reported (iron fortification)

Characteristics of ongoing studies *[ordered by study ID]*

NCT00129324

Trial name or title	HOME Study (**H**ealth **O**utcomes and **M**easures of the **E**nvironment Study)
Methods	Randomised, double-blinded trial
Participants	Pregnant women and their children 1. Group 1: 174 lead hazard control 2. Group 2: 181 injury control 3. Group 3: 53 no intervention
Interventions	1. Group 1: participants received lead hazard reduction controls prior to their child's birth 2. Group 2: participants received injury hazard reduction controls prior to their child's birth 3. Group 3: no intervention
Outcomes	Blood lead concentrations and neurobehavioral outcomes
Starting date	**Started**: March 2003 **Estimated completion date**: September 2017
Contact information	**Principal investigator**: Bruce P Lanphear, MD, MPH **Address**: Children's Hospital Medical Center, Cincinnati
Notes	**Trials identifier**: NCT00129324 **More information about the study**: cincinnatichildrens.org/research/divisions/e/environmental/study/summary

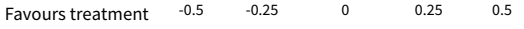
DATA AND ANALYSES

Comparison 1. Education interventions compared to no intervention or standard 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 ≥ 10.0 µg/dL (dichotomous)	4	520	Risk Ratio (IV, Random, 95% CI)	1.02 [0.79, 1.30]
3 Blood lead level ≥ 15.0 µg/dL (dichotomous)	4	520	Risk Ratio (IV, 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]

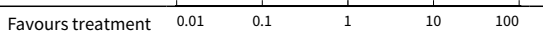
Analysis 1.1. Comparison 1 Education interventions compared to no intervention or standard education, Outcome 1 Blood lead level (continuous).

Study or subgroup	Treatment N	Treatment Mean(SD)	Control N	Control Mean(SD)	Mean Difference Random, 95% CI	Weight	Mean Difference Random, 95% CI
Brown 2006	71	2.2 (0.6)	74	2.1 (0.6)		29.8%	0.07[-0.12,0.26]
Jordan 2003	142	1.7 (1.5)	154	1.6 (1.6)		8.5%	0.04[-0.31,0.39]
Lanphear 1996a	52	1.8 (0.5)	43	1.9 (0.8)		13.57%	-0.02[-0.3,0.26]
Lanphear 1999	117	1.7 (0.7)	112	1.8 (0.7)		35.11%	-0.07[-0.24,0.1]
Wasserman 2002	28	1.4 (0.6)	22	1.2 (0.4)		13.02%	0.14[-0.14,0.42]
Total ***	**410**		**405**			**100%**	**0.02[-0.09,0.12]**
Heterogeneity: Tau²=0; Chi²=2.13, df=4(P=0.71); I²=0%							
Test for overall effect: Z=0.29(P=0.77)							



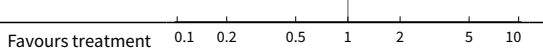
Analysis 1.2. Comparison 1 Education interventions compared to no intervention or standard education, Outcome 2 Blood lead level ≥ 10.0 µg/dL (dichotomous).

Study or subgroup	Treatment n/N	Control n/N	Risk Ratio IV, Random, 95% CI	Weight	Risk Ratio IV, Random, 95% CI
Brown 2006	39/71	37/74		65.26%	1.1[0.81,1.5]
Lanphear 1996a	11/52	12/43		12.42%	0.76[0.37,1.54]
Lanphear 1999	21/118	22/112		21.62%	0.91[0.53,1.55]
Wasserman 2002	2/28	0/22		0.7%	3.97[0.2,78.59]
Total (95% CI)	**269**	**251**		**100%**	**1.02[0.79,1.3]**
Total events: 73 (Treatment), 71 (Control)					
Heterogeneity: Tau²=0; Chi²=1.87, df=3(P=0.6); I²=0%					
Test for overall effect: Z=0.12(P=0.9)					

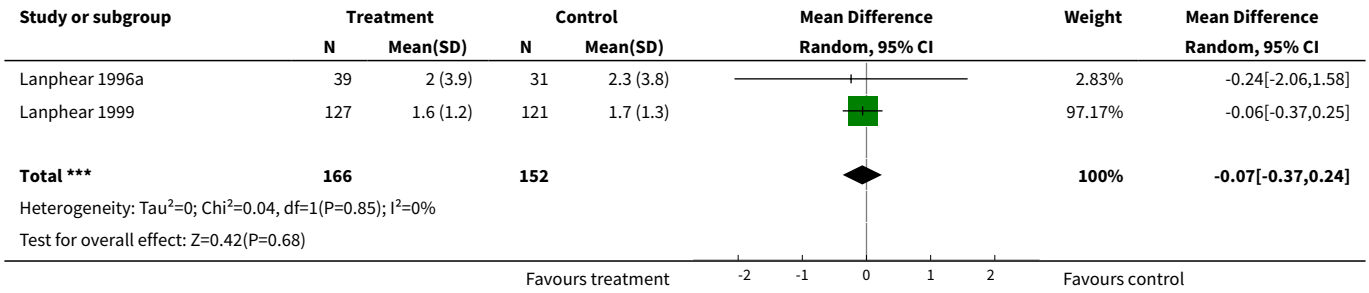


Analysis 1.3. Comparison 1 Education interventions compared to no intervention or standard education, Outcome 3 Blood lead level ≥ 15.0 µg/dL (dichotomous).

Study or subgroup	Treatment n/N	Control n/N	Risk Ratio IV, Random, 95% CI	Weight	Risk Ratio IV, Random, 95% CI
Brown 2006	8/71	11/74		48.72%	0.76[0.32,1.77]
Lanphear 1996a	3/52	6/43		20.05%	0.41[0.11,1.56]
Lanphear 1999	5/118	9/112		31.23%	0.53[0.18,1.53]
Wasserman 2002	0/28	0/22			Not estimable
Total (95% CI)	**269**	**251**		**100%**	**0.6[0.33,1.09]**
Total events: 16 (Treatment), 26 (Control)					
Heterogeneity: Tau²=0; Chi²=0.65, df=2(P=0.72); I²=0%					
Test for overall effect: Z=1.69(P=0.09)					



Analysis 1.4. Comparison 1 Education interventions compared to no intervention or standard education, Outcome 4 Floor dust - hard floor.



Comparison 2. Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure

Outcome or subgroup title	No. of studies	No. of partici-pants	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 ≥ 10.0 µg/dL (dichoto-mous)	2	210	Risk Ratio (M-H, Random, 95% CI)	0.93 [0.73, 1.18]
3 Blood lead level ≥ 15.0 µg/dL (dichoto-mous)	2	210	Risk Ratio (M-H, Random, 95% CI)	0.86 [0.35, 2.07]
4 Blood lead level ≥ 10.0 µg/dL (dichoto-mous): ICC 0.01	2	204	Risk Ratio (M-H, Random, 95% CI)	0.93 [0.73, 1.18]
5 Blood lead level ≥ 10.0 µg/dL (dichoto-mous): ICC 0.1	2	173	Risk Ratio (M-H, Random, 95% CI)	0.95 [0.72, 1.24]
6 Blood lead level ≥ 10.0 µg/dL (dichoto-mous): ICC 0.2	2	155	Risk Ratio (M-H, Random, 95% CI)	0.97 [0.72, 1.29]
7 Blood lead level ≥ 15.0 µg/dL (dichoto-mous): ICC 0.01	2	204	Risk Ratio (M-H, Random, 95% CI)	0.82 [0.37, 1.81]
8 Blood lead level ≥ 15.0 µg/dL (dichoto-mous): ICC 0.1	2	173	Risk Ratio (M-H, Random, 95% CI)	0.83 [0.34, 2.03]
9 Blood lead level ≥ 15.0 µg/dL (dichoto-mous): ICC 0.2	2	155	Risk Ratio (M-H, Random, 95% CI)	0.75 [0.34, 1.66]

Analysis 2.1. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 1 Blood lead level (continuous).

Study or subgroup	Treatment N	Treatment Mean(SD)	Control N	Control Mean(SD)	Mean Difference Random, 95% CI	Weight	Mean Difference Random, 95% CI
Boreland 2009	44	2.9 (0.3)	44	2.9 (0.3)		35.09%	-0.02[-0.14,0.1]
Hilts 1995	56	2.4 (0.3)	55	2.4 (0.4)		34.7%	0.03[-0.1,0.16]
Rhoads 1999	46	2.2 (0.5)	53	2.7 (0.6)		30.21%	-0.52[-0.74,-0.3]
Total ***	**146**		**152**			**100%**	**-0.15[-0.42,0.11]**

Heterogeneity: Tau^2=0.05; Chi^2=19.18, df=2(P<0.0001); I^2=89.57%

Test for overall effect: Z=1.12(P=0.26)

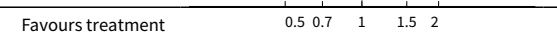
Analysis 2.2. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 2 Blood lead level ≥ 10.0 µg/dL (dichotomous).

Study or subgroup	Treatment n/N	Control n/N	Risk Ratio M-H, Random, 95% CI	Weight	Risk Ratio M-H, Random, 95% CI
Hilts 1995	33/56	35/55		64.72%	0.93[0.69,1.25]
Rhoads 1999	22/46	27/53		35.28%	0.94[0.63,1.4]
Total (95% CI)	**102**	**108**		**100%**	**0.93[0.73,1.18]**

Total events: 55 (Treatment), 62 (Control)

Heterogeneity: Tau^2=0; Chi^2=0, df=1(P=0.96); I^2=0%

Test for overall effect: Z=0.59(P=0.55)



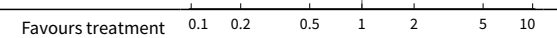
Analysis 2.3. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 3 Blood lead level ≥ 15.0 µg/dL (dichotomous).

Study or subgroup	Treatment n/N	Control n/N	Risk Ratio M-H, Random, 95% CI	Weight	Risk Ratio M-H, Random, 95% CI
Hilts 1995	12/56	9/55		52.74%	1.31[0.6,2.86]
Rhoads 1999	6/46	13/53		47.26%	0.53[0.22,1.29]
Total (95% CI)	**102**	**108**		**100%**	**0.86[0.35,2.07]**

Total events: 18 (Treatment), 22 (Control)

Heterogeneity: Tau^2=0.23; Chi^2=2.25, df=1(P=0.13); I^2=55.58%

Test for overall effect: Z=0.35(P=0.73)



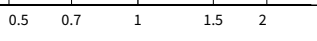
Analysis 2.4. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 4 Blood lead level ≥ 10.0 µg/dL (dichotomous): ICC 0.01.

Study or subgroup	Treatment n/N	Control n/N	Risk Ratio M-H, Random, 95% CI	Weight	Risk Ratio M-H, Random, 95% CI
Hilts 1995	31/53	33/52		63.13%	0.92[0.68,1.25]
Rhoads 1999	22/46	27/53		36.87%	0.94[0.63,1.4]
Total (95% CI)	**99**	**105**		**100%**	**0.93[0.73,1.18]**
Total events: 53 (Treatment), 60 (Control)					
Heterogeneity: Tau^2=0; Chi^2=0.01, df=1(P=0.94); I^2=0%					
Test for overall effect: Z=0.6(P=0.55)					

Favours treatment — Favours control

Analysis 2.5. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 5 Blood lead level ≥ 10.0 µg/dL (dichotomous): ICC 0.1.

Study or subgroup	Treatment n/N	Control n/N	Risk Ratio M-H, Random, 95% CI	Weight	Risk Ratio M-H, Random, 95% CI
Hilts 1995	22/37	23/37		54.56%	0.96[0.66,1.38]
Rhoads 1999	22/46	27/53		45.44%	0.94[0.63,1.4]
Total (95% CI)	**83**	**90**		**100%**	**0.95[0.72,1.24]**
Total events: 44 (Treatment), 50 (Control)					
Heterogeneity: Tau^2=0; Chi^2=0, df=1(P=0.95); I^2=0%					
Test for overall effect: Z=0.38(P=0.7)					



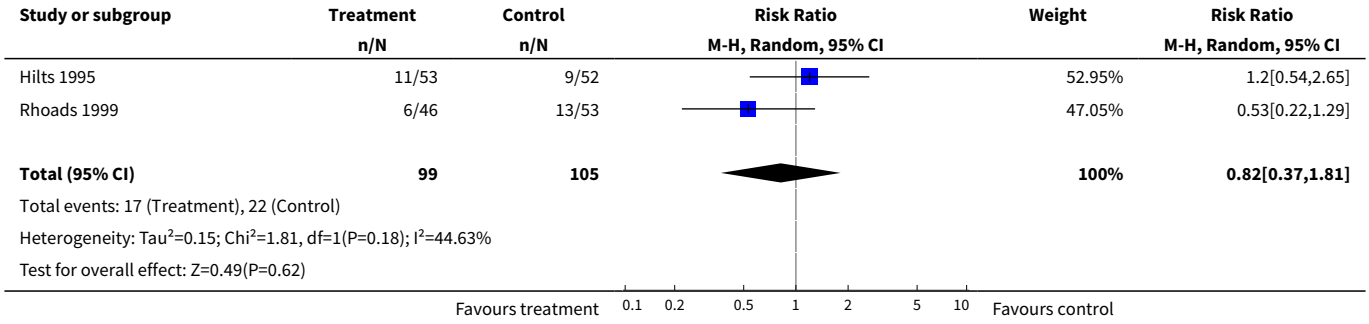
Favours treatment — Favours control

Analysis 2.6. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 6 Blood lead level ≥ 10.0 µg/dL (dichotomous): ICC 0.2.

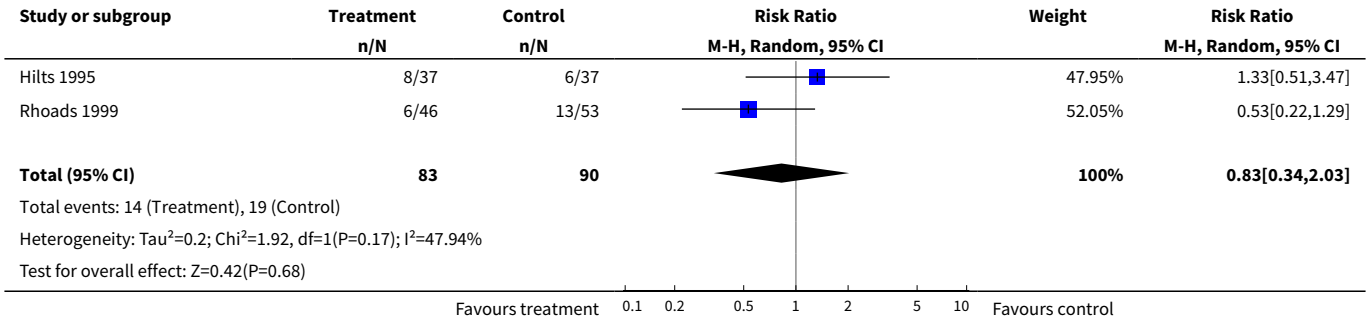
Study or subgroup	Treatment n/N	Control n/N	Risk Ratio M-H, Random, 95% CI	Weight	Risk Ratio M-H, Random, 95% CI
Hilts 1995	17/28	17/28		47.54%	1[0.66,1.52]
Rhoads 1999	22/46	27/53		52.46%	0.94[0.63,1.4]
Total (95% CI)	**74**	**81**		**100%**	**0.97[0.72,1.29]**
Total events: 39 (Treatment), 44 (Control)					
Heterogeneity: Tau^2=0; Chi^2=0.05, df=1(P=0.83); I^2=0%					
Test for overall effect: Z=0.22(P=0.82)					

Favours treatment — Favours control

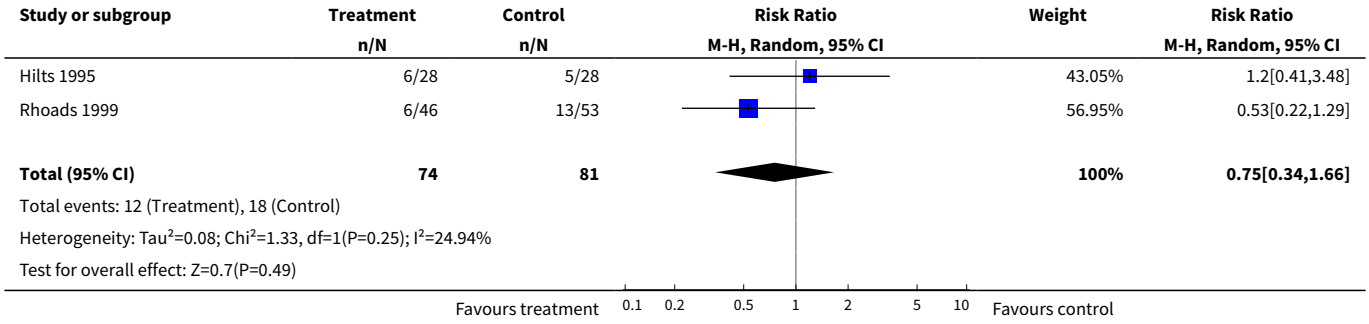
Analysis 2.7. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 7 Blood lead level ≥ 15.0 µg/dL (dichotomous): ICC 0.01.



Analysis 2.8. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 8 Blood lead level ≥ 15.0 µg/dL (dichotomous): ICC 0.1.



Analysis 2.9. Comparison 2 Environmental interventions (dust control) compared to no intervention or another intervention not aimed to influence domestic lead exposure, Outcome 9 Blood lead level ≥ 15.0 µg/dL (dichotomous): ICC 0.2.



ADDITIONAL TABLES

Table 1. Mean blood lead level and age at baseline

Study ID	Mean blood lead level at baseline (µg/dL)	Age at baseline (months)
Aschengrau 1998	15.0 - 19.0	24 - 36
Boreland 2009	15.0 - 19.0	> 36
Brown 2006	15.0 - 19.0	12 - 24
Campbell 2011	2.6 - 2.7	8 - 14
Charney 1983	> 20.0	> 36
Farrell 1998	10.0 - 14.0	6 - 72
Hilts 1995	10.0 - 14.0	24 - 36
Jordan 2003	< 10.0	< 12
Lanphear 1996a	< 10.0	12 - 24
Lanphear 1999	< 10.0	< 12
Rhoads 1999	10.0 - 14.0	12 - 24
Sterling 2004	10.0 - 14.0	> 36
Wasserman 2002	< 10.0	12 - 24
Weitzman 1993	10.0 - 14.0	4 - 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	—	—	—

Table 2. Intervention type by study *(Continued)*

Lanphear 1996a	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)	Household floor dust lead	Household window dust lead	Other
Aschengrau 1998	Yes	—	Yes	Yes	—
Boreland 2009	Yes	—	—	—	—
Brown 2006	Yes	Yes	Yes	—	Parent - Child Interaction scale
Campbell 2011	Yes	—	Yes	Yes	Chicago Parents Knowledge Test
Charney 1983	Yes	Yes	—	—	—
Farrell 1998	—	—	—	—	Total effect (blood lead levels)
Hilts 1995	Yes	Yes	Yes	—	—
Jordan 2003	Yes	—	—	—	—
Lanphear 1996a	Yes	Yes	Yes	Yes	—
Lanphear 1999	Yes	Yes	Yes	Yes	—
Rhoads 1999	Yes	Yes	Yes	Yes	Maternal knowledge lead poisoning
Sterling 2004	—	Yes	—	—	—
Wasserman 2002	Yes	Yes	—	—	Chicago Parents Knowledge Test
Weitzman 1993	Yes	—	—	—	—

APPENDICES

Appendix 1. Search strategies used up to 2006

Cochrane Central Register of Controlled Trials (CENTRAL) in the Cochrane Library

#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)

Ovid MEDLINE

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$.ti,ab.
25. research design/
26. or/16-25
27. 26 not 14
28. 15 or 27
29. adolescent/ or child/ or infant.mp.
30. (child$ 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

Embase Ovid

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 (allocat$ 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 Ovid

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

CINAHL Ovid (Cumulative Index to Nursing and Allied Health Literature)

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

Sociofile Cambridge Scientific Abstracts

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

ERIC EBSCO

Lead poisoning.mp. and Lead poisoning (subject heading)

Science Citation Index ProQuest (SCI)

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

ZETOC

(zetoc.jisc.ac.uk)

"childhood lead poisoning prevention"

LILACS (Latin American and Caribbean Health Science Information database)

(lilacs.bvsalud.org/en)

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

Dissertation Abstracts ProQuest

Searched using the phrase "childhood lead poisoning prevention"

Other databases and websites

ClinicalTrials.gov (clinicaltrials.gov), Current Controlled Trials (s3-us-west-2.amazonaws.com/webcitation/6fc2d850dde348335d1ab0b66828190d475a6967), Australian New Zealand Clinical Trials Registry (ANZCTR; anzctr.org.au), and National Research Register (no longer available. For more information, see webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/en/aboutus/researchanddevelopment/atoz/dh_4002357) were searched using the terms "lead" and "children"

Appendix 2. Search strategies used from 2006 to 2016

The same strategy was used each time the search was run, unless indicated otherwise.

Cochrane Central Register of Controlled Trials (CENTRAL) in the Cochrane Library

#1MeSH descriptor: [Lead Poisoning, Nervous System, Childhood] this term only
#2MeSH descriptor: [Lead Poisoning] this term only
#3MeSH descriptor: [Lead] this term only
#4lead near poison*
#5lead near expos*
#6lead near blood*
#7lead near toxic*
#8lead near environ*
#9lead near reduc*
#10lead near hazard*
#11lead near control*
#12lead near pollut*
#13lead near contamin*
#14lead near (domestic* or home* or hous*)
#15{or #1-#14}
#16[mh infant]
#17[mh child]
#18[mh adolescent]
#19child* or baby or babies or toddler* or boy* or girl* or preschool* or pre-school* or (pre next school*) or teen* or adolescen*
#20{or #16-#19}
#21#20 and #15

Ovid MEDLINE

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 [Note: Lines 12 to 31 are the Cochrane highly sensitive search strategy for identifying randomised trials (Lefebvre 2008)]
32 14 and 20 and 31

Embase OVID

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$).tw.
19 or/16-18
20 Randomized controlled trial/
21 controlled clinical trial/
22 Single blind procedure/
23 Double blind procedure/
24 triple blind procedure/
25 Crossover procedure/
26 (crossover or cross-over).tw.
27 ((singl$ or doubl$ or tripl$ or trebl$) adj1 (blind$ or mask$)).tw.
28 Placebo/
29 placebo.tw.
30 prospective.tw.
31 factorial$.tw.
32 random$.tw.
33 assign$.ab. (2)
34 allocat$.tw.
35 volunteer$.ab.
36 or/20-35
37 15 and 19 and 36

PsycINFO OVID

1 "Lead (Metal)"/
2 Lead Poisoning/
3 Pb.tw.
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$).tw.
15 or/1-14
16 ("100" or "120" or "140" or "160" or "180" or "200").ag.
17 (child$ or baby or babies or toddler$ or boy$ or girl$ or preschool$ or pre-school$ or pre school$ or teen$ or adolescen$).tw.
18 16 or 17
19 clinical trials/
20 (randomis* or randomiz*).tw.

21 (random$ adj3 (allocat$ or assign$)).tw.
22 ((clinic$ or control$) adj trial$).tw.
23 ((singl$ or doubl$ or trebl$ or tripl$) adj3 (blind$ or mask$)).tw.
24 (crossover$ or "cross over$").tw.
25 random sampling/
26 Experiment Controls/
27 Placebo/
28 placebo$.tw.
29 exp program evaluation/
30 treatment effectiveness evaluation/
31 ((effectiveness or evaluat$) adj3 (stud$ or research$)).tw.
32 or/19-31
33 15 and 18 and 32

PsycINFO EBSCO

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*)

CINAHL EBSCO (Cumulative Index to Nursing and Allied Health Literature)

S39 S16 AND S20 AND S38
S38 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 OR S37
S37 (MH "Quantitative Studies")
S36 (MH "Treatment Outcomes")
S35 (MH "Program Evaluation")
S34 TI (evaluat* study or evaluat* research) or AB (evaluate* study or evaluat* research) or TI (effectiv* study or effectiv* research) or AB(effectiv* study or effectiv* research)
S33 TI (prospectiv* study or prospectiv* research) or AB(prospectiv* study or prospectiv* research)
S32 TI ("follow-up study" or "follow-up research") or AB ("follow-up study" or "follow-up research")
S31 AB("cross over")
S29 AB((tripl* N3 mask*) or (tripl* N3 blind*))
S28 AB((trebl* N3 mask*) or (trebl* N3 blind*))

S27 AB ((doubl* N3 mask*) or (doubl* N3 blind*))
S26 AB ((singl* N3 mask*) or(singl* N3 blind*))
S25 AB ((clinical trial*) or(control* trial*))
S24 AB((random* N3 allocat*) or(random* N3 assign*))
S23 (MH "Meta Analysis")
S22 MH random assignment
S21 (MH "Clinical Trials+")
S20 S17 OR S18 OR S19
S19 (MH "Adolescence+")
S18 (MH "Child+")
S17 (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 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 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 (home* or hous*)) or TI (lead N5 (home* or hous*))
S11 AB (lead N5 domestic*) or TI (lead N5 domestic*)
S10 AB (lead N5 control*) or TI (lead N5 control*)
S9 AB (lead N5 hazard*) or TI (lead N5 hazard*)
S8 AB (lead N5 environ*) or TI (lead N5 environ*)
S7 AB (lead N5 toxic*) or TI (lead N5 toxic*)
S6 AB (lead N5 reduc*) or TI (lead N5 reduc*)
S5 AB (lead N5 blood*) or TI (lead N5 blood*)
S4 AB (lead N5 expos*) or TI (lead N5 expos*)
S3 AB (lead N5 poison*) or TI (lead N5 poison*)
S2 (MH "Lead Poisoning")
S1 (MH "Lead")

Sociological Abstracts Cambridge Scientific Abstracts (previously searched as Sociofile)

((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 5hazard*) 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*)))

Sociological Abstracts ProQuest

(SU.EXACT("Children") OR SU.EXACT("Infants") OR SU.EXACT("Adolescents") or (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*)) AND (SU.EXACT("Lead Poisoning") or (lead near/5 poison*) or (lead near/5 expos*) or (lead near/5 blood*) or (lead near/5 reduc*) or (lead near/5 toxic*) or (lead near/5 environ*) or (lead near/5 hazard*) or (lead near/5 control*) or (lead near/5 domestic*) or (lead near/5 home*) or (lead near/5 house*) or (lead near/5 contamin*) or (lead near/5 pollut*) or "pb")

ERIC Cambridge Scientific Abstracts (Education Resources Information Center)

((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*)))

ERIC EBSCO

S23 S17 AND S22
S22 S18 OR S19 OR S20 OR S21
S21 DE "Adolescents"
S20 DE "Young Children" OR DE "Infants" OR DE "Preschool Children" OR DE "Toddlers"
S19 DE "Children" OR DE "Preadolescents" OR DE "Young Children"
S18 (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 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 OR S16
S16 (AB ("Pb") or TI ("Pb"))
S15 AB (lead N5 pollut*) or TI (lead N5 pollut*)
S14 AB (lead N5 contamin*) or TI (lead N5 contamin*)

S13 AB (lead N5 (home* or hous*)) or TI (lead N5 (home* or hous*))
S12 AB (lead N5 domestic*) or TI (lead N5 domestic*)
S11 AB (lead N5 control*) or TI (lead N5 control*)
S10 AB (lead N5 hazard*) or TI (lead N5 hazard*)
S9 AB (lead N5 environ*) or TI (lead N5 environ*)
S8 AB (lead N5 toxic*) or TI (lead N5 toxic*)
S7 AB (lead N5 reduc*) or TI (lead N5 reduc*)
S6 AB (lead N5 blood*) or TI (lead N5 blood*)
S5 AB (lead N5 expos*) or TI (lead N5 expos*)
S4 AB (lead N5 poison*) or TI (lead N5 poison*)
S3 S1 AND S2
S2 TI (lead) or AB(lead)
S1 DE "Hazardous Materials" OR DE "Poisoning" OR DE "Wastes"

ERIC ProQuest

(((SU.EXACT("Lead poisoning")) OR ((lead NEAR/5 poison*) OR (lead NEAR/5 expos*) OR (lead NEAR/5 blood*) OR (lead NEAR/5 reduc*) OR (lead NEAR/5 toxic*) OR (lead NEAR/5 environ*) OR (lead NEAR/5 hazard*) OR (lead NEAR/5 control*) OR (lead NEAR/5 domestic*) OR (lead NEAR/5 home*) OR (lead NEAR/5 hous*) OR (lead NEAR/5 contamin*) OR (lead NEAR/5 pollut) OR ("Pb"))) AND ((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*) OR (SU.EXACT("Adolescents") OR SU.EXACT("Young
Children" OR "Children" OR "Preadolescents") OR SU.EXACT("Toddlers" OR "Infants")))) AND pd(20100101-20121231)

Science Citation Index Web of Science (SCI)

6 #5 AND #4 AND #3
5 TS=(random* or RCT or intervention*)
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" near/3 (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 near/3 (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*))

ZETOC MIMAS

(zetoc.jisc.ac.uk)

ZETOC was used as a source of conference papers up to 2012, after which Conference Proceedings Citation Index was preferred because of more flexible search options.

Lead and child*

Conference Proceedings Citation Index - Science Web of Science (CPCI-S)

CPCI-S was substituted for ZETOC in 2016 because of more advanced search options

6 #5 AND #4 AND #3
5 TS=(random* or RCT or intervention*)
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" near/3 (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 near/3 (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*))

Cochrane Database of Systematic Reviews (CDSR) and Database of Abstracts of Reviews of Effects (DARE) in the Cochrane Library

#1MeSH descriptor: [Lead Poisoning, Nervous System, Childhood] this term only
#2MeSH descriptor: [Lead Poisoning] this term only
#3MeSH descriptor: [Lead] this term only
#4(lead near poison*):ti,ab
#5(lead near expos*):ti,ab
#6lead near blood*:ti,ab
#7(lead near toxic*):ti,ab
#8(lead near environ*):ti,ab

#9(lead near reduc*):ti,ab
#10(lead near hazard*):ti,ab
#11(lead near control*):ti,ab
#12(lead near pollut*):ti,ab
#13(lead near contamin*):ti,ab
#14(lead near (domestic* or home* or hous*)):ti,ab
#15{or #1-#14}
#16[mh infant]
#17[mh child]
#18[mh adolescent]
#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*):ti,ab
#20{or #16-#19}
#21#15 and #20

LILACS iAH form (Latin American and Caribbean Health Science Information database)

lilacs.bvsalud.org/en/

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$ orTW 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$) AND ((Pt randomized controlled trial OR Pt controlled clinical trial OR Mh randomized controlled trials OR Mh random allocation OR Mh double-blind method OR Mh single-blind method) AND NOT (Ct animal AND NOT (Ct human and Ct animal)) OR (Pt clinical trial OR Ex E05.318.760.535$ 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]

World Health Organisation International Clinical Trial Registry Portal (WHO ICTRP)

(who.int/ictrp/en)

Advanced search. CONDITION| lead exposure OR lead poisoning AND Recruitment status| ALL [23 records]
Basic search. domestic AND lead OR house AND lead OR home AND lead OR housing AND lead OR household AND lead [48 records]

All available years searched.Two separate searches were run using different search strings, Records from both searches were imported into Excel and duplicates removed leaving 70 records

"lead" AND "children"

Clinical Trials.gov

(clinicaltrials.gov)

Search 1: Interventional studies | Lead | Child [41 records]
Search 2: Interventional Studies | Lead poisoning OR Lead,blood OR Lead toxicity OR Lead and injury reduction OR Evironmental exposures [57 records]

All available years searched. Two separate searches were run using different search strings, Records from both searches were imported into Excel and duplicates removed leaving 70 records

"lead" AND "children"

Appendix 3. Record of searches between 2006 and 2016

Record of searches from 2006 to 2016				
Database	**Search date**	**Database date range**	**Number of records**	**Limits**

(Continued)

Cochrane Central Register of Controlled Trials (CENTRAL) in the Cochrane Library	29 April 2010	2010, Issue 2	171	Year = 2010-2012
	20 January 2012	2012, Issue 1	61	Year = 2010-2012
	11 May 2015	2015, Issue 4	75	Year = 2012-2015
	3 May 2016	2016, Issue 4	19	Year = 2015-2016
Ovid MEDLINE	29 April 2010	1950 to April Week 3 2010	373	Year = 2006-current
	17 January 2012	1948 to 2012 January Week 1	192	ED = 20100427-20120112
	11 May 2015	1946 to May Week 1 2015	453	ED = 20120101-20150430
	3 May 2016	1946 to April Week 3 2016	156	ED=20150430-20160421
Embase Ovid	28 May 2010	1980 to 2010 Week 16	144	Year = 2006-current. Searched 29 April 2010; rerun with revised filter 28 May 2010
	17 January 2012	1980 to 2012 Week 2	122	EM = 201021-201201
	11 May 2015	1980 to 2015 Week 19	447	EM = 201201-201519
	3 May 2016	1980 to 2016 Week 18	167	EM =201519-201618
PsycINFO Ovid	17 January 2012	1806 to December Week 2 2011	35	Year = 2010-current
	11 May 2015	1806 to May Week 1 2015	58	UP = 20111205-20150504
	3 May 2016	1806 to April Week 4 2016	15	UP = 20150504-20160425
PsycINFO EBSCO	04 May 2010	1806 to current	271	Year = 2006-current
CINAHL EBSCO (Cumulative Index to Nursing and Allied Health Literature)	04 May 2010	1937 to current	48	Year = 2006-2010
	20 January 2012	1937 to current	21	EM 20100504 onwards
	11 May 2015	1937 to current	87	EM 20120101 onwards
	3 May 2016	1937 to current	25	EM 20150430 onwards
Sociological Abstracts Cambridge Scientific Abstracts	29 April 2010	1952 to current	45	Year = 2006-2010
Sociological Abstracts ProQuest	20 January 2012	1952 to current	137	Year = 2010-2012
	12 May 2015	1952 to current	62	Year = 2012-2015
	3 May 2016	1952 to current	18	Year = 2015-2016
ERIC Cambridge Scientific Abstracts (Education Resources Information Center)	30 April 2010	1966 to current	81	Year = 2006-2010

(Continued)

ERIC EBSCOhost	11 May 2015	1966 to current	79	EM 2012 onwards
	3 May 2016	1966 to current	30	EM 2015 onwards
ERIC ProQuest	17 January 2012	1966 to current	63	Year = 2010-2012
Science Citation Index Web of Science (SCI)	04 May 2010	1970 to current	1827	Year = 2006-2010
	20 January 2012	1970 to current	176	Year = 2010-2012
	12 May 2015	1970 to 11 May 2015	389	Year = 2012-2015
	3 May 2016	1970 to 2 May 2016	175	Year = 2015-2016
ZETOC MIMAS Conference papers only	04 May 2010	All available years	399	Year = 2006 onwards
	20 January 2012	All available years	173	Year = 2010 onwards
Conference Proceedings Citation Index - Science Web of Science (CPCI-S)	12 May 2015	1990 to 11 May 2015	71	All available years
	3 May 2016	1990 to 2 May 2016	0	Year = 2015-2016
Cochrane Database of Systematic Reviews (CDSR) in the Cochrane Library	12 May 2015	2015, Issue 5	1	All available years
	3 May 2016	2016, Issue 6	0	Year = 2015-2016
Database of Abstracts of Reviews of Effects (DARE) in the Cochrane Library)	12 May 2015	2015, Issue 2	1	All available years
	3 May 2016	NA	NA	Not searched because no new content added
LILACS iAH Search form (Latin American and Caribbean Health Science Information database) (lilacs.bvsalud.org/en)	04 May 2010	All available years	113	Year = 2006-2010
	20 January 2012	All available years	36	Year = 2010-2012
	12 May 2015	All available years	83	Year = 2012-2015
	3 May 2016	All available years	21	Year = 2015-2016
Trials registers	**Search date**	**Register date range**	**Number of records**	**Limits**
World Health Organization International Clinical Trial Registry Portal (WHO ICTRP) (apps.who.int/trialsearch)	20 January 2012	All available years	24	—
	13 May 2015	All available years	70	—
	3 May 2016	All available years	13	Registered May 2015 or later
ClinicalTrials.gov (clinicaltrials.gov)	20 January 2012	All available years	54	—
	13 May 2015	All available years	84	—
	3 May 2016	All available years	10	Received 1 May 2015 or later

Appendix 4. Unused methods

Analysis	Method not used	Explanation
Measures of treatment effect	**Dichotomous data** We will calculate numbers needed to treat, where appropriate. **Continuous data** We will compare mean changes from baseline to postintervention between groups. Also, if we find a study reporting only change from baseline, we will contact the authors to receive the post-treatment means.	**Dichotomous data** We were unable to calculate numbers needed to treat on this occasion because we could not identify evidence on patient-relevant outcomes of interest (cognitive and neurobehavioural outcomes in children or adverse events in children). **Continuous data** We did not compare mean changes from baseline to postintervention between groups, because baseline blood lead levels were similar between arms in each study included in the meta-analyses. Also, we did not find a study reporting only change from baseline.
Unit of analysis issues	**Studies with multiple treatment groups** For eligible trials that consist of multiple treatment groups, we will include data for the treatment arms and halve the data from the control arm, or collapse the data for the treatment groups into one group when this considered appropriate.	We were unable to include the one trial that included multiple treatment groups in the meta-analysis.
Dealing with missing data	For continuous outcomes, we will impute missing SDs using relevant data (e.g. SDs or correlation coefficients) from other similar studies, where possible (Follman 1992).	There were insufficient data to impute results.
Assessment of reporting biases	If 10 or more studies are included in a meta-analysis, we will draw funnel plots to investigate any relationship between effect size and study precision (closely related to sample size). Such a relationship could be due to publication or related biases or due to systematic differences between small and large studies. If a relationship is identified, clinical diversity of the studies will be further examined as a possible explanation (Egger 1997).	We were unable to draw funnel plots to assess reporting biases due to the small number of studies (2-5) included in meta-analyses.
Subgroup analyses	We will conduct subgroup analyses, providing there are clinically relevant differences between groups of participants (e.g. age, baseline blood lead levels, household setting), in either intervention, and if mode of delivery of intervention differs significantly (e.g. written information rather than delivered directly by health professional/paraprofessional).	Due to the limited number of studies within each intervention type, there were insufficient data for subgroup analyses according to baseline age, baseline blood lead level, or mode of delivery of intervention.
Sensitivity analysis	If there are studies at different risk of bias levels, we will conduct sensitivity analysis based on different risks of bias. We will conduct the sensitivity analysis to assess the impact of study quality on the results of meta-analyses. We will also conduct a sensitivity analysis on a 'best-case/worst case' basis.	We did not conduct a sensitivity analysis based on different risks of bias because the studies included in the meta-analysis were at similarly low risk of bias. Also, there were insufficient data to conduct a sensitivity analysis on a 'best-case/worst-case' basis.

WHAT'S NEW

Date	Event	Description
29 July 2015	New citation required but conclusions have not changed	Our update search identified one additional publication (Campbell 2012) of a study that has already been included (Campell 2011) in the last version of the review. This publication added information on blood lead levels at two-year follow-up. We also identified an ongoing study (HOME Study 2015) that will be finished by 2017. These findings did not change the conclusions of the review.
11 May 2015	New search has been performed	Review updated following a new search in May 2015 and a top-up search on 3 May 2016.

HISTORY

Protocol first published: Issue 2, 2006
Review first published: Issue 2, 2008

Date	Event	Description
23 April 2012	Amended	Search dates corrected
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.
9 November 2008	Amended	Converted to new review format.

CONTRIBUTIONS OF AUTHORS

BNS, BY, UG, LMP, LKB, SKL, SL, GG wrote the updated text of the review. Margaret Anderson updated and performed the search. LKB, LMP, SL, SKL, BNS and GG screened titles, abstracts and full-text articles. UG resolved conflicts regarding inclusion/exclusion of an article. SL and BNS extracted data from original studies into data extraction forms. BNS and UG assessed the blinding domain in accordance with the updated methodological criteria in the *Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011a), as 'blinding of participants and personnel' and 'blinding of outcome assessment'. BNS and GG also reassessed the domain 'attrition bias'. BNS took over first authorship and is the guarantor for the review.

DECLARATIONS OF INTEREST

WHO financially supported the update of this review as part of its guideline development process.

Barbara Nussbaumer-Streit - none known.
Berlinda Yeoh - none known.
Ursula Griebler - none known.
Lisa M Pfadenhauer and Laura K Busert - attended the Cochrane Colloquium in 2015. Their participation fees were covered by WHO and paid via Danube University Krems.
Stefan K Lhachimi - none known.
Szimonetta Lohner - none known.
Gerald Gartlehner - none known.

SOURCES OF SUPPORT

Internal sources

- None, Other.

External sources

- World Health Organization (WHO), Switzerland.

  WHO, Evidence & Policy on Environmental Health (EPE), financially supported the update of the review as part of a guideline development process with US $15,000.
- Danube University Krems, Department of Evidence-based Medicine and Clinical Epidemiology, Austria.

  Lisa M Pfadenhauer and Laura K Busert attended the Cochrane Colloquium 2015 in Vienna; their participation fee was covered by the Department for Evidence Based Medicine and Clinical Epidemiology at the Danube University Krems.

DIFFERENCES BETWEEN PROTOCOL AND REVIEW

Search methods for identification of studies

We revised the search strategies used for the original review for the 2012 update (Appendix 1) by introducing additional search strings in which 'lead' is found in proximity to other terms (rather than searching for 'lead' as a single term). We re-ran the revised strategies for this 2016 update, and searched two additional databases (Cochrane Database of Systematic Reviews (CDSR) and Database of Abstracts of Reviews of Effects (DARE). We also searched Conference Proceedings Citation Index - Science (CPCI-S) as a substitute for searching for conference papers in ZETOC, because of its more advanced search interface.

Searching other resources

We did not search the internet for unpublished studies, because we examined the reference list of relevant studies, and contacted experts to determine whether any unpublished or ongoing trials existed.

Data collection and analysis

Data extraction and management

We used the most up-to-date version of RevMan (version 5.3) for this update (RevMan 2014).

Measures of treatment effect

Binary data

We did not calculate risk differences because they strongly depend on the baseline risk and are not as stable as risk ratios (Higgins 2011b).

Continuous data

For continuous data, we compared post-treatment means between intervention and control groups, and calculated mean differences (MDs) instead of comparing mean changes (from baseline to post-treatment) between intervention and control groups, because baseline data were comparable in the included studies.

Confidence in cumulative evidence

We used the GRADE method to assess the quality of the evidence from meta-analyses per outcome.

Assessment of heterogeneity

We reported Tau^2, an estimate of the between-study variance in a random-effects meta-analysis.

Changes in author team

Berlinda Yeoh (BY), Susan Woolfenden (SW), Danielle M Wheeler (DMW), Garth Aperstein (GA), and Bruce Lanphear (BL) developed and wrote the text of the original review (Yeoh 2006). In 2012 BY, SW, BL, Greta F Ridley (GFR), and Nuala Livingstone (NL) updated the original review. In 2014 BY, SW, BL, GFR, NL, and Emile Jorgensen (EJ) updated the review again. In 2016 the authors' team changed. BNS took over first authorship, BY stayed in the authors' team, and UG, LMP, LKB, SKL, SL, GG joined the new author team.

INDEX TERMS

Medical Subject Headings (MeSH)

Dust [*prevention & control]; Environmental Exposure [*prevention & control]; Environmental Restoration and Remediation [*methods]; Lead [blood]; Lead Poisoning [*prevention & control]; Paint [toxicity]; Randomized Controlled Trials as Topic; Soil

MeSH check words

Child, Preschool; Female; Humans; Infant; Male