UK National Screening Committee

Screening for elevated blood lead levels in asymptomatic children aged 1 to 5 years.

External review against programme appraisal criteria for the UK National Screening Committee (UK NSC)

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The UK NSC advises Ministers and the NHS in all four UK countries about all aspects of screening policy. Its policies are reviewed on a 3 yearly cycle. Current policies can be found in the policy database at http://www.screening.nhs.uk/policies and the policy review process is described in detail at http://www.screening.nhs.uk/policyreview

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Executive Summary:

There is insufficient evidence for screening for elevated lead levels in asymptomatic children aged 1 to 5 years.

Using the NSC criteria screening is not recommended because:

- the low prevalence of raised blood levels,
- benefits from primary prevention,
- lack reliability in currently available testing strategies,
- lack of a safe blood lead level and thus the lack of a suitable cut off level for screening,
- the lack of proven treatment modalities for raised blood lead levels especially for the majority of cases (very low levels of raised blood lead levels) that would be identified by screening,
- there is no RCT evidence on a screening programme in the UK context.

1 Introduction

This paper uses evidence published up to January 2012 on screening for elevated blood lead levels in asymptomatic children aged one to five years against the UK National Screening Committee (NSC) Criteria for appraising the viability, effectiveness and appropriateness of a screening programme[1].

The present NSC policy is that screening for lead poisoning is not recommended [2].

For this update a literature review was carried out in April 2012. English language literature published 01/01/2007 to 01/01/2012.

2 The Condition

2.1 The condition should be an important health problem

2 2. The epidemiology and natural history of the condition, including development from latent to declared disease, should be adequately understood and there should be a detectable risk factor, disease marker, latent period or early symptomatic stage

Introduction

Historically, lead poisoning has been an important disease with environmental causes which can have life-long adverse health effects. Children are most susceptible, especially if living in poverty and poor housing conditions.

Exposure to lead

Lead is ubiquitous in the environment. It is usually present in small amounts from natural ecological sources in all rock, water, soil, dust and air. These sources contribute to human exposure by influencing the composition of ingested food and water and dust that is inhaled or ingested.

Humans have been mining and using lead for thousands of years, poisoning themselves in the process. Although lead poisoning is one of the oldest known work and environmental hazards, the modern understanding of the small amount of lead necessary to cause harm did not come about until the latter half of the 20th century. No safe threshold for lead exposure has been discovered—that is, there is no known amount of lead that is too small to cause the body harm.

With the Industrial Revolution in the 19th century, lead poisoning became common in the work setting. The introduction of lead paint for residential use in the 19th century increased childhood exposure to lead. An important step in the understanding of childhood lead poisoning occurred when toxicity in children from lead paint was recognized in Australia in 1897[3]. France, Belgium, and Austria banned white lead interior paints in 1909; the League of Nations followed suit in 1922. The United States did not pass laws banning lead house paint until 1971 and it was not fully banned until 1978. In the UK the concentration of white lead in paint rose to its highest levels between the years 1930 and 1955 when it was as much as half the volume in some paints. In the 1950s, alternative white pigments were introduced. In 1963 a voluntary agreement was made that resulted in labeling of paint that contained more than 1% of lead. White lead was mainly used by professional decorators for specialist use. In 1992 European Union legislation was implemented within the U.K preventing the addition of lead to all paints except those intended for use in historic buildings and as artwork.

In the 1920s, lead was added to gasoline to improve its combustion. This was finally phased out by the late 1990s. Lead from this exhaust can still persist today in soil and dust.

The main routes of exposure today are:

- occupational, primarily in adults where they are involved in producing or using products containing lead,
- exposure to lead paint, especially children from dust or soil due to contaminated paint or direct eating of paint,
- exposure to contaminated soil including eating vegetables grown in lead contaminated soil,

- water contaminated via the atmosphere, soil or plumbing,
- products containing lead such as toys, cosmetics or lead solder often made in non UK countries.

Exposure occurs primarily through inhalation and ingestion. Children are at greater risk because; their bodies take in proportionately greater amounts of environmental toxins than adults, their rapid development makes them more vulnerable to environmental interference, bone metabolism is higher creating faster shifts of lead from bone to blood and their behaviour (eating soil and picking up objective from the floor which they then put in their mouths) places them at greater risk. Lead is mainly stored in blood, soft tissue and bone. The half life for lead stored in blood is weeks, months for soft tissue and years for bone. This means that lead stored in bones can be reintroduced into the blood stream long after initial exposure. Lead is removed from the body very slowly mainly through urine.

Lead and health

Lead has no known physiologically relevant role in the body but the harmful effects are considerable. Lead and other heavy metals create reactive free radicals which damage cell structures including DNA and cell membranes. Lead also interferes with metabolism of bones and teeth, harms the developing immune system and interferes with the metabolism of calcium in cells. One of the main causes for the pathology of lead is that it interferes with the activity of an essential enzyme called delta-aminolevulinic acid dehydratase or ALAD, which is important in the biosynthesis of heme, the cofactor found in heamoglobin. Lead can also mimic other metals (calcium, iron, zinc) that take part in biological processes as a cofactor. Lead binds to, and interacts with, many of the same enzymes as these metals. Due to its different chemistry once bound it affects these enzymes ability to catalyze normally [4,5]. The net result is that lead can affect every organ system in the body but especially the nervous system. The latter is important in relation to children as it can impact on developmental processes. Clinical impacts include: kidney damage, hypertension and heart disease, infertility and miscarriage or prematurity, hearing loss, tooth decay and peripheral and central nervous system disease.

The amount of lead in the blood and tissues and the length of exposure determines toxicity[4]. Lead poisoning may be acute (from intense exposure of short duration) or chronic (from repeated low-level exposure over a prolonged period). The latter is much more common[6].This report focuses on the latter as acute poisoning results in a clinically diagnosable event whilst chronic exposure is more likely to remain undiagnosed and is pertinent to a screening programme.

Diagnosis and treatment of lead exposure are based on the amount of lead in the blood measured in micrograms of lead per deciliter of blood (µg/dL). The US Centres for Disease Control and Prevention (CDC) in 1991 and the World Health Organisation (WHO)

in 1995 stated that a blood lead level of 10 µg/dL or above was a cause for concern. However, lead may impair development and have harmful health effects at lower levels, and it is generally accepted that there is no known safe exposure level[7,8]. This has recently resulted in a level of 5 µg/dL being set by CDC as a reference level for action and concern in the CDC 2012 publication, Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention Report of the Advisory Committee on Childhood Lead Poisoning Prevention[9]**.** The UK HPA website states that a blood level of less that 10µg/dL is desirable[10]. Bellinger calls the CDC level of 10µg/dL a risk management tool rather than a threshold for intoxity[11].

Lead poisoning causes a variety of symptoms and signs which vary depending on the individual and the duration of lead exposure. Symptoms are nonspecific and may be subtle. Someone with elevated lead levels may have no symptoms. Symptoms usually develop over weeks to months as lead builds up in the body during a chronic exposure, but acute symptoms from brief, intense exposures also occur. Symptoms from exposure to organic lead, which is probably more toxic than inorganic lead due to its lipid solubility, occur rapidly. Clinical symptoms of lead poisoning begin to appear in children at around 60 µg/dL. However, the lead levels at which symptoms appear vary widely depending on the characteristics of each individual, which it is suggested include genetic polymorphism and contextual factors[11]. At blood lead levels between 25 and 60 µg/dL, neuropsychiatric effects such as delayed reaction time, irritability, and difficulty concentrating, as well as slowed motor nerve conduction and headache can occur. Anaemia may appear at blood lead levels higher than 50 µg/dL. In children, signs of encephalopathy, lack of coordination, and apathy occur at lead levels exceeding 70 µg/dL. For both adults and children, it is rare to be asymptomatic if blood lead levels exceed 100 µg/dL.

Children are more at risk of lead poisoning because they absorb lead more rapidly. One reason is because children have a greater fractional gastrointestinal absorption rate especially where there are nutritional deficiencies which are more common in children especially those more likely to be exposed to lead. The CNS impact of lead is more profound in children as this is when their CNS is developing. The impact of lead on CNS appears to be irreversible in children as compared to adults[11]. The half life of lead in blood is also, in most situations, longer in children than in adults.

Needleman in 2004[12] showed that raised blood lead levels at two years of age were associated with a neuro-cognitive performance deficit at 10 years of age. An analysis of school-aged children published in 2005 demonstrated a stronger cross-sectional inverse association of IQ with contemporary blood lead levels (mean BLL = 8 mcg/dL at age 7 years) than with baseline blood levels (mean BLL = 26 mcg/dL at 24 months old) suggesting an ongoing adverse effect of lead on cognitive performance among school-aged children[13]. Other studies suggest an association between childhood lead exposure and disorders of attention and learning, and aggressive and delinquent behavior[11,12,14,15].

Lead associated effects on neurobehavioral functioning must be considered relative to other important covariates such as socioeconomic status, home and parenting, and genetic factors. The contribution of childhood lead exposure to the observed variance in cognitive ability (IQ testing) is believed to be in the range of 1-4%, while social and care giving factors may be responsible for 40% or more[11]. A meta-analysis of lead blood levels and conduct disorders published in 2010 looking at 16 studies concluded that; "overall, the relation between lead exposure and conduct problems was strikingly similar in magnitude to the relation between lead exposure and decreased IQ[16]." Importantly, blood lead levels are potentially associated with a substantial proportion of the known, modifiable variance in children's cognitive ability.

One of the few studies on lead in the UK was undertaken by Bristol University using samples taken from a subgroup of the Avon Longitudinal Study of parents and children attending a research clinic when the children were 30 months of age (n=582)[17]. Blood lead levels were measured by absorption spectrometry. Developmental, behavioural and educational outcomes were studied at 7 and 8 years. The study published in 2009 showed that after adjustment for confounders blood lead levels showed a significant association with reading, writing and spelling grades on SARs grades and antisocial behaviour. The association was apparent in levels between 5-10 µg/dL on reading and writing and on antisocial behaviour and hyperactivity over10 µg/dL. This paper proposed that the clinical threshold for concern should be reduced from the WHO level of 10 µg/dL to 5 µg/dL. This was the first time in the UK that the standard 10 µg/dL had been challenged. This paper focussed on risk and did not comment on the usefulness of population screening. Subsequently in the UK the standard level has not been reduced and remains at 10 µg/dL on the HPA website and a recent British Surveillance unit study used the same threshold. This focus on cases above 10µg/dL reflects the continuing emphasis on clinical case finding and treatment rather than reduction in population blood lead levels.

Prevalence

Blood lead levels in general population surveys around the world show a major decrease after measures were introduced to reduce lead exposure in the 1980s and beyond. These measures included reducing or banning lead in petrol, house paint, pipes and soldered cans containing food. In the USA, CDC prevalence figures for raised blood lead levels above 10µg/dL in children fell from over 7% of the surveyed population in 1997 to less than 1% in 2009[18]. In 2010 CDC started reporting blood lead levels between 5 and 9µg/dL. Six percent of the total population of children tested had blood lead levels between 5 and 9µg/dl.

The prevalence of elevated blood lead levels varies substantially among different communities and populations. Higher levels are found in: minority populations, urban residents, people of low income, people of low educational attainment, those living in older housing or with home renovation or living close to multiple demolitions, children

with pica, users of ethnic remedies or cosmetics and users of lead glazed pottery. Alcohol use and smoking are known risk factors among pregnant women. Blood lead levels can also show seasonal variation.

The highest geometric mean blood lead levels (GM blood lead levels) occur in children aged 1-5 years (GM 1.9 µg/dL) with the lowest in youths aged 6-19 years (GM 1.1 µg/dL)[19]. This picture remains despite the falling levels. Children under five are at greater risk of elevated blood lead levels because of increased hand to mouth activity, periods of crawling on the floor and around soil and increased lead absorption from the gastrointestinal tract. Geometric mean levels are significantly higher in males than in females except among children aged 1-5 years. This peak remains despite the major reduction in cases.

In the UK blood lead levels have fallen by at least 4% yearly from the early 70s to late 1980s[20]. A survey was conducted annually over the period 1984 to 1987 measuring blood lead levels in particularly exposed areas and rural areas as control[20]. Every adult and child over the age of 11 years from selected households was eligible for the survey. 81 percent of the 8483 people eligible had blood levels taken, this included 340 children. Blood levels were higher in males than females and rose with age. No children had levels over10 µg/dL and only 6 percent of adults had levels raised to that level. Only 11 adults had levels over25 µg/dL the level above which investigations are recommended by the WHO. Higher levels were associated with manual work, smoking, alcohol and living in the north. No significant difference was found with age of dwelling. There are no population surveillance figures in UK.

The ALSPAC study of 584 children's blood levels in 1992 showed a range of 0.8 to 27.6 µg/dL with a skewed distribution, the highest at age 2 years and falling in subsequent years. These levels were lower than a comparable study 15 years previously but the distribution was similar. There was a statistically significant association with higher levels in children with younger mothers, living in rented accommodation, exposure to tobacco smoke, presence of pets and living near traffic. Breast feeding for six months was associated with a lower level as was living outside of the city[20].

In 2010 the Health Protection Agency announced a joint study with the British Paediatric Surveillance Unit (BPSU) to investigate lead poisoning levels among children in the United Kingdom and Republic of Ireland[21]. This is a study of cases over10 µg/dL with a view to identify risk factors. The study population is children, seen in secondary care by peadiatricians, with developmental problems. It is due to report in 2013. To date the data suggests there are fewer cases than anticipated but there is no process for determining the level of underreporting. Results are not yet available on the risk factors associated with the children who have raised lead levels. The statement about the study says that currently there is no monitoring of clinical cases nor knowledge of the incidence and prevalence of clinical cases in the UK. They suggest that due to this lack of data the public health response may be sub-optimal

Conclusion

Lead has a detrimental effect on health of children even at very low blood levels. The exact level of health risk at this low level is not known. Cases at higher blood levels in the UK are very much reduced meaning it is unlikely to be a major health problem. However, the lack of robust surveillance data means that in the UK it is not possible to confirm accurately the size of the problem and especially in relation to the impact of blood lead levels below 5µg/dL on very young children in their developmental years.

2.3. All the cost-effective primary prevention interventions should have been implemented as far as practicable

Primary Prevention

As discussed above there has been a considerable reduction in children's blood levels during the last three decades. It is generally considered that this is due to primary prevention in the UK which is the reduction in lead as a risk to children. Primary prevention includes: legislation to control lead in drinking soil, water, paint, fuel, toys and from industrial emissions[22].

The UK presently appears to be taking a piecemeal approach to lead poisoning prevention. The HPA strategy for children, " A Children's Environment and Health Strategy for the UK" quotes that the median blood lead levels in the 1990s (1–3 µg/dL) had declined approximately ten-fold compared with levels in the 1960s (23 µg/dL). In its strategy of 2009 the only recommendation concerning reduction of environmental lead levels relates to lead in drinking water. A new national drinking water quality standard for lead comes into force in 2013. Currently, levels of lead must be below 25 micrograms per litre (µg/l), but the new standard means that levels of lead will have to be below 10 µg/l[22]. The UK Water strategy document says that "a number of actions are currently taking place across the UK to meet this new standard including the replacement of lead pipes, the use of phosphate dosing (which reduces the amount of lead that will dissolve into drinking water from lead pipes and solder), and the testing of levels of lead in water supplies[23]." The Drinking Water Inspectorate has recommended that local authorities (in England and Wales) review how often lead levels in drinking water goes above the new standard. The Food Standards Agency launched a campaign in 2009 to stop lead poisoning on farms, caused primarily by leakage from lead batteries which subsequently get into the food chain[24]. In the Department of Health 2009 Children's Strategy for Healthy Lives, Brighter Futures lead is not mentioned as an issue[25].

The US CDC 2012 lead prevention strategy, "Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention" strongly promotes primary prevention[9]. The main message is that preventing any exposure rather than responding to a set level is the appropriate response. This argument is based on the success of regulatory policies that control or eliminate sources of lead in the environment, the lack of proven methods to reverse harm in children with an elevated BLL, and the lack of a blood lead level threshold that is safe. The strategy focuses on educating families, service providers,

advocates and public officials of the need to; ensure lead free environments in the home and other facilities, ensure all agencies to data share, develop and implement lead free housing policies, finance lead hazard remediation and inform families how to protect their children from lead hazards.

Environmental control divides into two types of intervention macro level action such as changing national policy on lead in petrol and paint and micro level interventions with families and communities. Studies on the latter such as those considering household dust and paint hazard control through cleaning, abatement and education have had mixed results. The Cochrane review published in 2012 on household interventions for preventing domestic lead exposure in children looked at randomised and quasi-randomised controlled trials of household educational or environmental interventions with at least one standardised outcome measure. Fourteen studies involving 2656 children were included. The conclusion of the meta-analysis was "that educational and dust control interventions are not effective in reducing blood lead levels of young children. There is currently insufficient evidence that soil abatement or combination interventions reduce blood lead levels and further studies need to address this. More research is needed to find out what is effective for preventing children's exposure to lead and studies should be carried out in different socioeconomic groups within developed countries as well as in developing countries[26]."

It is suggested that one of the problems of environmental control focussed on homes is that poor inner-city families tend to move frequently so treating a current residence may have limited benefit as the child will have moved but it is does have benefit for the present and future population of children resident in those buildings treated. Where contamination is due to lead dust from industrial facilities or soil contamination, intervention in the house alone will not be sufficient. In the developed world regulation of industrial facilities is usually sufficient and thus there is much more of a problem in the developing world where regulation is less comprehensive. Most programmes are also limited in geographical scale as they are usually locally funded, focussed in a particular area and time limited.

Conclusion

Primary preventive actions at a macro level have had considerable impact on reducing raised blood lead levels in children. Further research is required to understand the impact of more locally based interventions.

3 The Test

3.1. There should be a simple, safe, precise and validated screening test.

3.2. The distribution of test values in the target population should be known and a suitable cut-off level defined and agreed.

3.3. The test should be acceptable to the population

The screening test most commonly used is capillary testing of blood lead levels[27]. It is best for modest lead exposure but accuracy, precision, and reliability can be affected by environmental lead contamination during collection, day to day biological variability and laboratory analytic variation. Capillary testing can also be affected by lead contamination of collecting equipment and skin contamination. The USPSTF suggest that contamination may positively bias blood lead levels by up to 1.0 µg/dL on average although individual effects of skin contamination may be even greater[26]. Studies defining abnormal results as blood lead levels above 10 or 20 µg/dL have reported false positive rates of 3 to 9 % for capillary sampling compared to venous blood lead collected simultaneously[26]. Day to day biological variability and trends over time also contributes to higher false positives for capillary sampling when compared to results from venous testing done at a later date. False negative rates appear to be lower than venous blood sampling. A study in 2007 in the State of Maine, US looking at false positives in a state lead poisoning surveillance data system suggested that sample contamination, rather than delay between capillary and venous tests or laboratory error, may be a primary cause of false positive results and recommended training to overcome this. This was a unique study in that it looked at state-wide data rather than selected sample populations, and thus is much more relevant when considering applicability of the test for population screening. They found a seventy three percent level of false positives using capillary testing as confirmed by blood levels. False positives were more likely in levels between 10 and 14 µg/dL than in higher levels[28]. There are no other comparable studies to indicate if this high level of false positive was study specific.

In communities with a low prevalence screening capillary venous testing will deliver low yields of children with positive results therefore questionnaires aiming to identify children with raised lead levels are used in the US. A recent systematic review of the literature on questionnaires used to identify cases of lead poisoning was undertaken by a researcher working in Washington State Department of Health[29]. He reviewed the literature up to 2009. The conclusion was that lead questionnaires fared no better than chance at identifying cases. Questionnaires have not been used in the UK context.

Conclusion

The present screening tests have limitations especially as prevalence levels fall below 10 µg/dL.

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3.5. There should be an agreed policy on the further diagnostic investigation of individuals with a positive test result and on the choices available to those individuals

Screening test results should be confirmed with a whole blood lead concentration. As there is no screening programme in the UK further diagnostic protocols for screened cases are not available.

4. The Treatment

4.1. There should be an effective treatment or intervention for patients identified through early detection, with evidence of early treatment leading to better outcomes than late treatment

The UK Royal College of Paediatrics and Child Health guidelines on treatment of, and interventions for, symptomatic children with elevated blood lead levels are based on the level of lead. At lower levels (10 to 45) µg/dL the intervention of choice is to remove the child from the source. At higher levels over 50µg/dL, chelation is advised[30].

Early detection aims to enable intervention before the development of irreversible complications especially developmental. Thus the key is removal of the child from the source. However, there is little convincing evidence that these interventions improve health as the child is asymptomatic and long term studies show that asymptomatic children with elevated lead levels will have a reduction in the level over time in the absence of intervention due to regression, random variation and redistribution of blood to other tissues[27].

There is good evidence that chelating agents benefit children with symptomatic lead poisoning, but there is little evidence available to demonstrate a clinical benefit from chelation therapy for children with lead levels <45 µg/dL or asymptomatic children. A large, multi-centre randomized controlled trial by the U.S. National Institute for Environmental Health Science (NIEHS) in 1992 to 1997 assessed the effect of oral chelation therapy with succimer on IQ in children with venous blood lead concentrations of 20 to 45 µg/dL[31]. At 36 months' follow-up, no statistically significant differences were found between treatment and control groups in mean IQ, parental rating of behaviour, or tests of learning ability. In this trial, blood lead levels decreased in both the treatment and placebo groups, and by 24 months the difference between treatment and placebo groups was not statistically significant. Re-analysis of the same data using the change in blood lead level as the independent variable demonstrated a 4.0 point improvement in cognitive scores for every 10 micro-g/dL reduction in blood lead level, but only in the placebo group, suggesting that factors other than declining blood lead contributed to cognitive improvement, or that treatment had an adverse

effect on cognitive performance[32]. Assessment of neurobehavioral outcomes at 7 years of age revealed no statistically significant differences on a battery of neurobehavioral tests, except that the succimer group had worse attention-executive function scores[33]. Treatment also appeared to have an adverse effect on mean height[34]. The Trial Group concluded that chelation therapy was not indicated for children with blood lead levels <45 micro-g/dL[32].

In the previously noted NIEHS-sponsored RCT of oral chelation in young children with venous blood lead concentrations of 20–45 µg/dL (TLC Study), which reported no effects of chelation on IQ blood lead levels fell steeply in the treatment group in the first week (mean 11 µg/dL lower) but then began to rebound. Blood lead levels also dropped in the placebo group, but more slowly. Blood lead levels were 77% of baseline in the succimer group (88% of baseline among placebo) at seven weeks after initiation of therapy. Mean blood lead levels among the treatment group were 4.5 µg/dL and 2.7 µg/dL, at six and twelve months respectively, but by 24 months the difference between treatment and placebo groups was not significant[31]. The USPSTF concluded that the data provides good evidence that chelating agents may result in short-term reductions in blood lead levels in children but that these reductions may not be sustained over longer periods due to transfer of lead from bones to the blood or continued absorption, in the absence of repeated or continuing chelation therapy or environmental interventions.

Conclusion

Chelation is not advised for blood lead levels less than 45 µg/dL. It is this level that would be mainly identified in a screening programme. For children identified at the lower level removal from the source of lead is advised and primary prevention interventions to remove the source long term.

4.2. There should be agreed evidence- based policies covering which individuals should be offered treatment and the appropriate treatment to be offered

The HPA advice is that where chelation therapy is indicated, two drugs are available; oral DMSA (succimer) 30 mg/kg/day (DMSA is not licensed in the UK) or sodium calcium edetate 40 mg/kg twice daily (or 75 mg/kg/daily) by IV infusion for 5 days. They consider that expert toxicologist advice should be sought before institution of either agent[30].

4.3. Clinical management of the condition and patient outcomes should be optimised in all healthcare providers prior to participation in a screening programme

Once diagnosed there are appropriate health services for lead poisoning. The availability of environmental interventions such as removing lead paint, cleansing contaminated areas, reducing traffic etc are dependent on the local authority and the owner of the

buildings, land or facility involved. The level of environmental health intervention is not documented but appears to be variable across the country.

5. The Screening Programme

5.1. There should be evidence from high quality Randomised Controlled Trials that the screening programme is effective in reducing mortality or morbidity.

The information that is provided about the test and its outcome must be of value and readily understood by the individual being screened

The 2006 review of the evidence for the USPSTF on screening for elevated lead levels in childhood and pregnancy prepared by the Oregon Evidence based practice centre states that "there is no available evidence to demonstrate the universal screening for blood lead results in better clinical outcomes than either screening targeted to high risk persons or individualised testing in response to clinical suspicion[35].

They considered that older studies that support intensive screening programs targeted to children in high-risk neighbourhoods and purport to reduce case fatality rates, mortality rates, and proportions of children detected with very high blood lead levels have no concurrent controls making it unclear if reported reductions are due to screening or to improvements in medical care over time. The USPSTF consider the "available evidence regarding the efficacy of screening programs is weak." The official USPSTF recommendation is that there is insufficient evidence to recommend for or against screening for asymptomatic children aged 1 to 5 years who are at increased risk and against screening for children at moderate risk.

The recent report of the CDC Advisory Committee on Childhood Lead Poisoning Prevention states that as the evidence has increased on the deleterious impact of lead levels below 10 µg/dL which is not associated with socio-economic status; screening for elevated blood levels should no longer be acceptable practice and that primary prevention should be the policy of choice. They consider that all homes should be lead free and environmental hazards reduced before children are exposed. Children should not live in older housing with lead based paints[36].

No evidence on screening programmes for elevated lead levels in the UK was identified in the literature search.

Conclusion

There is insufficient evidence on the benefits of screening programmes for raised blood lead levels in children aged 1 to 5 years.

5.2. There should be evidence that the complete screening programme (test, diagnostic procedures, treatment/ intervention) is clinically, socially and ethically acceptable to health professionals and the public.

A review published in 2008 looking at the issues that influenced carers on whether to take their children for lead screening or not, reviewed 34 studies of which only seven papers focused specifically on parent's attitudes to blood lead screening. They determined that the barriers to, and enablers of, screening for elevated blood lead levels appear to be similar to those identified for other screening programs and it recommended that further research is required to understand how best to encourage screening[37].

A 2005 study of physician barriers to lead screening in the US looked at physicians and paediatricians who cared for Medicaid funded children. With a response rate of 64%, 68% said that they routinely screened for lead. Of those that didn't most were aware of Medicaid's requirement for testing but did not screen because they believed that they were in a low risk area. 35% who said this were actually in a high risk area[38].

There is no evidence on acceptability of screening in the UK.

5.3. The benefit from the screening programme should outweigh the physical and psychological harm (caused by the test, diagnostic procedures and treatment).

The most common adverse effect from screening is false positive finger-stick results and the anxiety, inconvenience and economic costs of inappropriate tests.

Treatment of high levels can also be invasive and may require hospitalisation of children. Chelating agents can have side effects including transient renal, hepatic and other toxicity as well as potential adverse impacts on outcomes as discussed above.

Environmental lead reduction techniques can result in raised levels if done badly and outside of the recommended techniques.

There are no RCTs comparing benefits and harms of a lead screening programme.

5.6. The opportunity cost of the screening programme (including testing, diagnosis and treatment, administration, training and quality assurance) should be economically balanced in relation to expenditure on medical care as a whole (i.e. value for money). Assessment against these criteria should have regard to evidence from cost benefit and/or cost effectiveness analyses and have regard to the effective use of available resource.

A study undertaken in the US in 2008 estimating the benefits that might be realized if all children in the United States had a blood lead level of less than 1 µg/dL used a Markov model to project lifetime earnings, reduced crime costs, improvements in health, and reduced welfare costs. They looked at a primary preventive intervention rather than screening. Their estimation was that reducing blood lead levels to less than 1 µg/dL among all US children between birth and age 6 years would reduce crime and increase on-time high school graduation rates later in life. The net societal benefits arising from these improvements in high school graduation rates and reductions in crime would amount to $50,000 per child annually at a discount rate of 3%. This would result in overall savings of approximately $1.2 trillion and produce an additional 4.8 million QALYs (SD, 2 million QALYs) for US society as a whole[39].

There is no RCT evidence on cost benefit and opportunity costs of a lead screening programme in the UK.

5.7. All other options for managing the condition should have been considered (e.g. improving treatment, providing other services), to ensure that no more cost- effective intervention could be introduced or current interventions increased within the resources available.

There have not been any trials comparing screening in the UK to other interventions. Lower levels studies support primary prevention at a macro level.

5.8. There should be a plan for managing and monitoring the screening programme and an agreed set of quality assurance standards.

This would be available is a screening programme was implemented but is not available at present.

5.9. Adequate staffing and facilities for testing, diagnosis, treatment and programme management should be available prior to the commencement of the screening programme.

This would need to be resourced.

5. 10. Evidence-based information, explaining the consequences of testing, investigation and treatment, should be made available to potential participants to assist them in making an informed choice.

This would be developed if a programme was implemented.

6. Conclusions

There is insufficient evidence for screening for raised blood lead levels in asymptomatic children aged 1 to 5 years.

Using the NSC criteria screening is not recommended because:

- the low prevalence of raised blood levels,
- benefits from primary prevention,
- lack of sensitivity of the screening test,
- lack of a suitable cut off for screening,
- treatment modalities are not appropriate for the majority of cases (very low levels of raised blood lead levels) that would be identified by screening,
- there is no RCT evidence on a screening programme in the UK context.

6.1. Implications for policy

Consideration should be given to the introduction and implementation of a comprehensive, co-ordinated primary prevention strategy for raised blood lead levels in the UK.

6.2. Implications for research

There is a lack of evidence on interventions that reduce the inequalities in lead levels in children aged one to rive years.

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