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ENVIRONMENTAL LEAD EXPOSURE AND CHILDHOOD DEVELOPMENT

The Port Pirie Cohort Study

SHILU TONG

Abstract

‘Environmental health’ is as broad as it is diverse in scope. As seen from this book, Tony McMichael contributed and advanced many aspects of the discipline, and indeed the field more broadly. In particular, his influence on the direction of the discipline and the policy responses to so many of the key issues that he addressed are recognised throughout this book as being seminal. Lead is one of the topics to which Tony’s research contributed. I was fortunate at the time to have been an eager student and the lead project was one in which I participated.

Background

In the late 1970s and early 1980s, another important but unanswered question was whether environmental lead exposure might affect young children’s health and development. Lead, from old house paint flakes, car exhaust and some industrial activity was a widespread pollutant. Many cross-sectional studies examined whether exposure to environmental lead at levels previously believed to be innocuous affected neuropsychological development and most of them found an inverse association between exposure measures and neuropsychological performance (Needleman et al., 1979; Smith et al., 1983; Winneke et al., 1983; Harvey et al., 1984; Schroeder et al., 1985; Lansdown et al., 1986; Fulton et al., 1987; Pocock et al., 1987; Hatzakis et al., 1989).

Cross-sectional studies cannot address the temporal relationship between exposure to environmental lead and neuropsychological development because they measure the exposure and outcome(s) at the same time. To test this hypothesis and address the limitations of cross-sectional studies, cohort studies have subsequently been conducted in several countries.

The Port Pirie Lead Smelter Study

Port Pirie, South Australia, has the largest lead smelter in the southern hemisphere. The Port Pirie cohort study (PPCS) commenced in 1979 (McMichael et al., 1988). This was the world's first and largest long-term follow-up study of the influence of early childhood exposure to environmental lead on the neurological and cognitive development of children. The research team had recruited a cohort of 723 children by 1981. These represented an estimated 90 per cent of all pregnancies in and around Port Pirie over a three-year period.

Blood samples were taken from each child at specified ages, and blood lead concentration was measured. Children's 'first teeth' – after they were shed – were collected to provide a further measure of accumulated lead exposure. Lead exposures varied greatly within the community, depending both on the proximity of residence to the smelter and on the levels of lead contamination in house dust and soil. At children's ages 2, 4, 7 and 12, a structured interview was conducted with one of the parents (usually the mother) and neuropsychological tests were administered by a well-trained research psychologist.

I came from China to join Tony and his team in early 1990, to work on the ongoing PPCS. Tony was then Professor of Environmental and Occupational Medicine in the Department of Community Medicine at the University of Adelaide. Shortly after I arrived in Adelaide, Tony wanted me to learn a little about Australian culture and environment and took me to Victor Harbour, which was about 70 km away from the town. Tony and I enjoyed the natural beauty there. By the time I joined the PPCS, the children were around 10 years old. For my PhD research (my supervisors: Tony, Drs Peter Baghurst and Michael Sawyer), I coordinated the follow-up and neuropsychological assessments in the later stage of childhood, at ages 11–13.

Results and Discussion

Of the 723 singleton live births recruited into the study, 601 were assessed at age 2, 548 at age 4, 494 at age 7 and 375 at 11–13 years of age. The geometric mean blood lead concentration of the children in this cohort increased from 8.3

$\mu\text{g}/\text{dl}$ ($0.40 \mu\text{mol}/\text{l}$) at birth to $21.2 \mu\text{g}/\text{dl}$ ($1.02 \mu\text{mol}/\text{l}$) at age 2, and decreased to $7.9 \mu\text{g}/\text{dl}$ ($0.38 \mu\text{mol}/\text{l}$) by the age of 11–13. The PPCS collected information on critical features of lead exposure, such as the timing and extent, together with many other socio-environmental factors that might confound the relationship between lead exposure and neuropsychological development. In this study, we found that exposure to lead in this cohort of children was associated inversely with cognitive performance at ages 2, 4, 7 and 11–13, and that this association was still apparent after adjustment for a wide range of confounding factors.

Similar to the PPCS, most other cohort studies (Needleman et al., 1990; Dietrich et al., 1991; Baghurst et al., 1992; Bellinger et al., 1992; Wasserman et al., 1992; Fergusson and Horwood, 1993; McMichael et al., 1994; Lanphear et al., 2005), but not all (Cooney et al., 1989; Ernhart et al., 1989), also found a significant inverse relationship between early exposure to environmental lead and cognitive functioning in childhood after adjustment for confounding factors. However, an important question was raised about whether the apparent deleterious effect of early-life exposure to lead could be reversed when, later in childhood, exposure was reduced. Such exposure reduction could occur either by environmental management or as a consequence of the decreased absorption that appears to accompany growth.

The children's lifetime average blood lead level covered a wide range, from a low level of around $3\text{--}5 \mu\text{g}/\text{dl}$ up to around $30 \mu\text{g}/\text{dl}$ (Tong et al., 1996). Figure 7.1 shows that the children are grouped into three exposure categories: low, medium and high, by thirds of lifetime average blood lead concentration up to age 2 (the age when the children's developmental status was assessed for the first time in this cohort study). Their development score, on the vertical axis, changed in a fairly linear way across those three categories, and that was similar at each of the four assessment ages – shown by the four coloured bars. There was an approximately 5 per cent decline in IQ across a whole range of blood lead levels, after potential confounders had been taken into account.

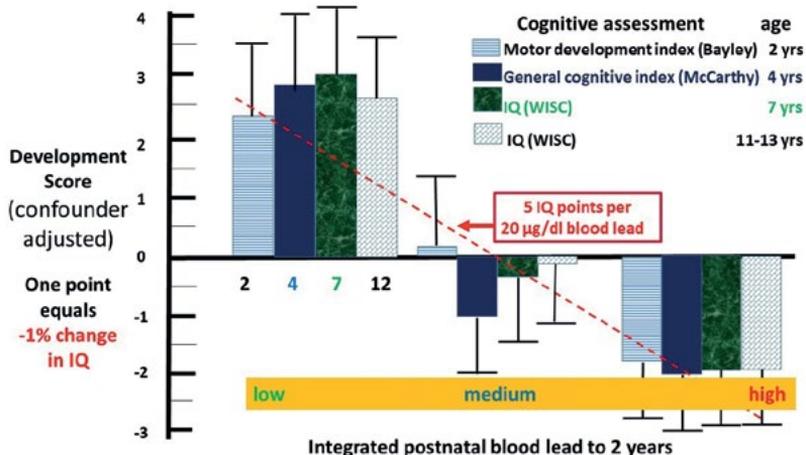


Figure 7.1 Developmental status at different ages by thirds of lifetime average blood lead concentration at age 2.

Source: Author's work.

Additionally, we assessed the reversibility of the apparent effects of lead on cognitive abilities in early childhood by testing whether reductions in blood lead concentrations beyond the age of 2 were associated with improvements in cognition (Tong et al., 1998). The key finding was that the cognitive deficits associated with exposure to environmental lead in early childhood appeared, at most, to be only partially reversed by a subsequent decline in blood lead (Table 7.1). The results from the PPCS indicate that the putative effects of lead on the neuropsychological development of children are genuinely irreversible, and therefore efforts to reduce the exposure of children to environmental lead from a very early age become increasingly important.

Table 7.1 Changes in blood lead concentration and changes in cognitive functioning between 2, 4 and 7 and 11–13 years.

| Decline in blood lead (µg/dl) | N | ΔZ score ^a | | |
|-------------------------------|-----|-----------------------|---------------|----------------------|
| | | Mean (SD) | 95% CI | P-value ^b |
| From 2 to 11–13 years | | | | |
| <10.2 | 109 | 0.03 (0.94) | -0.15 to 0.21 | |
| 10.2~16.2 | 108 | 0.04 (1.01) | -0.15 to 0.23 | 0.74 |
| >16.2 | 109 | -0.01 (1.02) | -0.20 to 0.18 | |
| From 4 to 11–13 years | | | | |
| <6.0 | 109 | 0.05 (0.90) | -0.12 to 0.23 | |
| 6.0–10.3 | 108 | 0.01 (0.91) | -0.17 to 0.18 | 0.42 |
| >10.3 | 109 | 0.01 (0.96) | -0.17 to 0.19 | |

| Decline in blood | | ΔZ score ^a | | |
|----------------------------------|----------|-------------------------------|--------------|------------------------------|
| lead ($\mu\text{g}/\text{dl}$) | <i>N</i> | Mean (SD) | 95% CI | <i>P</i> -value ^b |
| From 7 to 11–13 years | | | | |
| <2.3 | 109 | -5.4 (8.3) | -3.8 to -7.0 | |
| 2.3–4.9 | 108 | -5.0 (9.3) | -3.2 to -6.8 | 0.45 |
| >4.9 | 109 | -3.8 (7.9) | -2.3 to -5.3 | |

Notes:

^a ΔZ score = $(Z_{11-13} - Z_2)$ or ΔZ score = $(Z_{11-13} - Z_4)$ or ΔZ score = $(Z_{11-13} - Z_7)$

^b analysis of variance test.

Source: Tong et al., 1998.

Conclusion

This study provided strong evidence of the detrimental and dose-related impact of lead exposure on early child development, and showed the irreversible nature of lead effects. It demonstrated the importance of prevention and the early treatment of children with lead poisoning. However, lead contamination in a community can last for a long time and is difficult to eliminate. For example, there has not been much reduction in lead smelter emissions, and lead contamination is still widespread in Port Pirie (Taylor et al., 2013).

These results contributed to Australia's move to mandatory lead-free petrol in the 1990s, and were important in the World Health Organization (WHO) and Organisation for Economic Co-operation and Development (OECD) revisions of lead exposure standards soon after. Tony, in conjunction with Drs Peter Baghurst, Graham Vimpani and Neil Wigg, led this seminal study, which made a significant contribution to protecting children from lead effects, both nationally and internationally (Burns et al., 1999; Tong et al., 2000).

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