

deaths in populations with low socioeconomic status that are linked to certain personality profiles or cognitive abilities, which in turn are related to genetic factors, early environment, social mobility, or a combination of these, are no less unfair than are premature deaths attributable to poverty, unsafe working conditions, or absence of health care. Development of interventions and policies that create equal opportunities for child development is, therefore, an urgent priority and could help to reduce health inequalities. To quote an old feminist slogan, "The personal is political", also in the health inequalities field.

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I declare that I have no conflicts of interest.

1 Chapman BP, Fiscella K, Kawachi I, Duberstein PR. Personality, socioeconomic status, and all-cause mortality in the United States. *Am J Epidemiol* 2010; **171**: 83–92.

2 Goldberg LR. The structure of phenotypic personality traits. *Am Psychol* 1993; **48**: 26–34.

- 3 Nabi H, Kivimäki M, Marmot MG, et al. Does personality explain social inequalities in mortality? The French GAZEL cohort study. *Int J Epidemiol* 2008; **37**: 591–602.
- 4 Macintyre S. The Black report and beyond: what are the issues? *Soc Sci Med* 1997; **44**: 723–45.
- 5 Singh-Manoux A, Ferrie JE, Lynch JW, Marmot M. The role of cognitive ability (intelligence) in explaining the association between socioeconomic position and health: evidence from the Whitehall II prospective cohort study. *Am J Epidemiol* 2005; **161**: 831–39.
- 6 Batty GD, Der G, Macintyre S, Deary IJ. Does IQ explain socioeconomic inequalities in health? Evidence from a population based cohort study in the west of Scotland. *BMJ* 2006; **332**: 580–84.
- 7 Batty GD, Gale CR, Tynelius P, Deary IJ, Rasmussen F. IQ in early adulthood, socioeconomic position, and unintentional injury mortality by middle age: a cohort study of more than 1 million Swedish men. *Am J Epidemiol* 2009; **169**: 606–15.
- 8 Holtzman NA. Genetics and social class. *J Epidemiol Community Health* 2002; **56**: 529–35.
- 9 Mackenbach JP. Genetics and health inequalities: hypotheses and controversies. *J Epidemiol Community Health* 2005; **59**: 268–73.
- 10 Conger RD, Donnellan MB. An interactionist perspective on the socioeconomic context of human development. *Annu Rev Psychol* 2007; **58**: 175–99.
- 11 Judge TA, Higgins CA, Thoreson CJ, Barrick MR. The big five personality traits, general mental ability, and career success across the life span. *Person Psychol* 1999; **52**: 621–52.
- 12 Hart CL, Taylor MD, Davey Smith G, et al. Childhood IQ and cardiovascular disease in adulthood: prospective observational study linking the Scottish Mental Survey 1932 and the Midspan studies. *Soc Sci Med* 2004; **59**: 2131–38.

## Even low-dose lead exposure is hazardous

Lead is the environmental pollutant with the largest toxicological database. Over 10 000 scientific publications about lead toxicity can be found on the internet. At the request of the European Commission, the European Food Safety Authority (EFSA) has now evaluated evidence and concluded that "it was not possible to exclude a risk to the developing fetus through exposure of some pregnant female consumers".<sup>1</sup> Despite the hedged language, this report is a radical diversion from classical toxicology: there is no known safe exposure to lead, EFSA says.

10 years ago, experts convened by WHO confirmed a provisional tolerable exposure limit of 25 µg lead per kg bodyweight per week.<sup>2</sup> EFSA now proposes to use a daily benchmark exposure of 0.5 µg—a decrease of over 85% when taking into account that WHO refers to weekly intake and EFSA to daily intake. However, this exposure level is not considered to be safe and should only be used to set priorities for preventive measures. All current European Union rules for lead in drinking water, food, and air, which are based on WHO limits, will therefore require downward revision. Absence of a safe exposure limit conflicts with classical toxicology. As expressed in the 16th century by Theophrastus Bombast von Hohenheim (Paracelsus), the

dose determines the poison. Thus, a toxicant is not toxic in small doses. But how small is that dose?

Blood concentrations of lead in the European Union average about 20 µg/L (0.1 µmol/L), but as late as 1970, the US Centers for Disease Control and Prevention (CDC) considered that, for children, blood concentrations of lead up to 30-fold higher than 20 µg/L were acceptable. The CDC's limit has been successively decreased, but remains at 100 µg/L.<sup>3</sup> Lead compounds added to automobile fuel for octane-boosting were the most serious source of environmental lead pollution worldwide, with daily lead emissions of up to 1000 tons. Since the 1970s, when the phase-out of leaded petrol began, lead exposure and blood concentrations of lead have declined substantially.<sup>1</sup> However, lead additives are still produced by one company, Innospec, in Ellesmere Port, UK, for use in a few countries in Asia, North Africa, and on the Balkan peninsula. Innospec admitted to bribing Indonesian officials to delay a ban of leaded petrol in Indonesia for several years.<sup>4</sup> Other sources of lead exposure include paints, cosmetics, traditional medicines, improperly fired ceramic tableware, water pipes, tobacco, and industrial emissions.<sup>1</sup> Control of most of these additional sources would now need to



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be tightened in the European Union, but even more so in developing countries.

Because lead pollution is global, epidemiological research has only recently addressed the possible adverse effects of the lower exposure levels that now prevail. If one assumes that lead absorption in the gut is 10% and that lead is evenly distributed throughout the body, the current average intake in European Union citizens of about 1 µg per kg bodyweight per day would correspond to about 100 lead atoms for each cell every day. Would Paracelsus consider that a small dose?

From early on, the lead industry was willing to control lead pollution, but any decisions would have to be made on the basis of facts rather than on opinions.<sup>5</sup> Although that might sound reasonable, there was a substantial delay in the emergence of convincing evidence. We now know that lead exposure increases the risk of diminished intelligence, attention deficit hyperactivity disorder, school failure, and criminal behaviour.<sup>1</sup> Worse, decrements in intellectual function per unit increase in blood concentrations of lead

are greater at low exposure levels—ie, below limits that were previously considered safe.<sup>6</sup> EFSA also emphasises that lead is associated with ailments that are common in elderly people, such as hypertension, renal dysfunction, and neurocognitive decline, possibly at exposure levels only slightly above those that affect brain development in children.

Regulatory strategies need to be revised in view of new scientific knowledge, but the insights gained should also be applied to a wider perspective beyond lead. Before the EFSA report, absence of evidence was often taken to be evidence of an absence of adverse effects. So, a chemical hazard was innocent until proven otherwise. Although we now know better, a generation of children paid the price for us to obtain insights into lead pollution. Future risk assessments should not ignore risks of low-level toxicity in susceptible populations because convincing evidence is not available.

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I was a member of the EFSA working group that drafted the opinion, but did not participate in the revision and final adoption.

- 1 European Food Safety Authority. Scientific Opinion on Lead in Food. EFSA panel on contaminants in the food chain (CONTAM). *EFSA J* 2010; **8**: 1570.
- 2 Joint FAO/WHO Expert Committee on Food Additives. Evaluation of certain food additives and contaminants. 2000. [http://whqlibdoc.who.int/trs/WHO\\_TRS\\_896.pdf](http://whqlibdoc.who.int/trs/WHO_TRS_896.pdf) (accessed Aug 18, 2010).
- 3 Centers for Disease Control and Prevention. Preventing lead poisoning in young children. August, 2005. <http://www.cdc.gov/nceh/lead/publications/prevleadpoisoning.pdf> (accessed Aug 18, 2010).
- 4 Serious Fraud Office. Innospec Limited prosecuted for corruption by the SFO. March 18, 2010. <http://www.sfo.gov.uk/press-room/latest-press-releases/press-releases-2010/innospec-limited-prosecuted-for-corruption-by-the-sfo.aspx> (accessed Aug 18, 2010).
- 5 Markowitz GE, Rosner D. Deceit and denial: the deadly politics of industrial pollution. Berkeley, CA: University of California Press, 2002: 35.
- 6 Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect* 2005; **113**: 894–99.

## Ending inequities in access to effective pain relief?

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The undertreatment of pain caused by cancer and other conditions is a global health tragedy. WHO estimates that 5 billion people live in countries with low or no access to opioid analgesics.<sup>1</sup> Each year, tens of millions of patients suffer without adequate treatment, including 5.5 million patients with terminal cancer.<sup>1</sup> The fact that this appalling situation needs to be remedied was recognised at the annual meeting of the UN Commission on Narcotic Drugs, in March, 2010.

There is a striking global inequity in access to opioid analgesics. In 2008, the 13% of the world's population living in Australia, Canada, New Zealand, the USA, and the member states of the European Union consumed more than 90% of the morphine consumed globally.<sup>2</sup>

Inequities in access to health services reflect social and economic causes that are widely recognised.<sup>3</sup> Access to opioids for analgesia or the treatment of drug dependence is also constrained internationally and