Placental and stillbirth tissue lead concentrations in occupationally exposed women

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ABSTRACT   The lead values in maternal and infant blood, in placental tissue, and in stillbirth liver, kidney, and rib- and skull-bones have been determined in samples from the Stoke-on-Trent area. The lead values in antenatal blood and placenta increase with occupational exposure; liver and kidney stillbirth lead values are lower than those of much older children and rib-bone lead values from stillbirths were on average three times as high as those from a control group comprised of cot deaths and early infant deaths from accidental causes.

The abortifactant properties of lead salts in moderate to high doses and the high rate of miscarriage in women working in a poorly controlled environment containing lead are well documented (see references within Waldron and Stöfen[1]), as are the teratogenic effects of lead alone, and with cadmium, in animals.[2-4] What is not certain, however, is whether low concentrations of lead have any effect on the human fetus. Some workers, on the basis of a few cases, have claimed that lead can be teratogenic[5][6] while others[7] suggest that the case is not proved. Our own work on Birmingham mothers showed that placental-lead concentrations were appreciably higher in cases of stillbirth or neonatal death, but our results did not distinguish between causative and consequential effects.[8] In the present investigation we have examined lead values in various tissues from women employed in the pottery industry in Stoke-on-Trent.

Tissue samples

Placentae, blood, stillbirth bone, liver, and kidney were supplied by JGD (formerly of the Health and Safety Executive) from mothers employed in the pottery industry in Stoke-on-Trent. Records were available for the occupation of the women from whom blood and placental samples had been taken. Placentae and stillbirth samples were stored at

−20°C, and the time of storage before analysis varied from 6 to 12 months. Blood samples were analysed within a few days of collection. Control samples of infant bones were supplied by Dr H A Waldron (London School of Hygiene and Tropical Medicine) and consisted of cot-deaths (age under 1 week) and accidental deaths (age under 18 weeks).

Stillbirth bone samples were wet-digested with nitric acid and diluted solutions analysed by graphite furnace atomic absorption spectrophotometry (AAS). Placental, liver, and kidney samples were solubilised with Soluene -350 and diluted solutions analysed by graphite furnace AAS using our previously reported method.[9][10] Blood samples were analysed at the medical laboratory of the Health and Safety Executive.

Placenta

In several respects the placental-lead values gave similar results to our Birmingham investigation though with lower absolute values. Thus the placental-lead values where a stillbirth had occurred (20 cases) had a high average value ($0.45 \pm 0.32\ \mu g/g$) compared with that from eight mothers who had not worked in industry during the previous two years ($0.29 \pm 0.09\ \mu g/g$). The mean value for the nine mothers who had had a malformed live-birth was not high ($0.32 \pm 0.14\ \mu g/g$) but in ten cases where record sheets gave some statement concerning fetal distress, the placental-lead values ($0.43 \pm 0.19\ \mu g/g$) were close to those where a stillbirth had resulted. The average placental-lead value for all these Stoke-on-Trent samples ($0.35 \pm 0.23\ \mu g/g$) was about one-
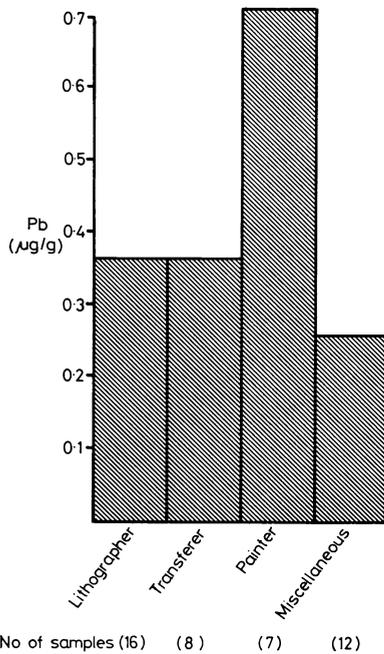
Fig 1   *Relation between placental lead and type of occupation (normal births).*

third that of our earlier reported values for 1971 Birmingham samples but appreciably higher than the value we now find for fresh Birmingham placentas (ca 0·12 μg/g). We ascribe this difference to the precipitation of placental-lead with continued storage at −20°C coupled with our method of sample
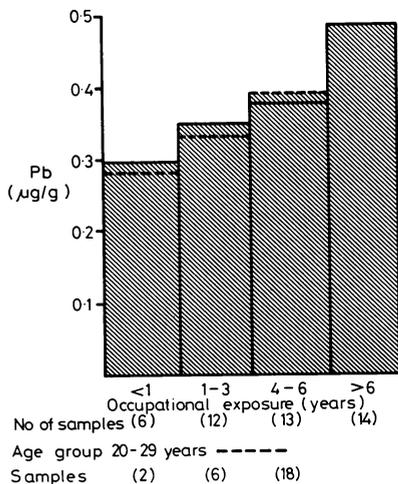
preparation which entails the prior removal of excess fluid.

When comparing the results from the different occupational groups (fig 1) it was found that lithographers (0·34 ± 0·14 μg/g) and transferers (0·34 ± 0·16 μg/g) had the same mean placental-lead value and that this was higher than that of the women described as following miscellaneous occupations (0·25 ± 0·07 μg/g). One paintress had an exceptionally high value (1·7 μg/g), and even omitting this result, the average value for this group (0·54 ± 0·09 μg/g) was significantly greater than for lithographers or transferers (p < 0·01). The placental-lead burden also increased appreciably with length of occupational exposure (fig 2). Although placental lead also increases with maternal age from an average value of 0·30 ± 0·16 μg/g for age < 20, 0·35 ± 0·14 μg/g for age 20-29 to 0·51 ± 0·44 μg/g for age ⩾ 30, within a group of mothers aged 20-29 (26 samples) the placental-lead figures showed an increase with increasing job exposure (fig 2).

Blood

In common with several other reports we find a highly significant positive correlation (r = +0·40, p = <0·001) between lead values in maternal and infant blood (65 samples) but an insignificant correlation between maternal blood lead and placental lead (r = +0·16). There was little variation between the lead values in maternal blood samples taken immediately before birth (12 ± 5 μg/100 ml) irrespective of occupation or length of employment, but when antenatal samples (mean value 17 ± 7 μg/ 100 ml) are grouped, an occupational variation is apparent (table).

Antenatal blood-lead concentrations in different occupational groups

	Transferer	Lithographer	Paintress
Pb (μg/100 ml)	12	19 ± 9	24 ± 9
No of samples	2	27	11

Kidney and liver

Lead values in 11 stillbirth kidney samples varied from 0·30 to 0·69 μg/g with a mean value of 0·48 ± 0·13 μg/g (wet wt). The same stillbirths had liver-lead values of 0·02-1·12 with a mean of 0·42 ± 0·30 μg/g. Previous workers have quoted liver and kidney values for stillbirths of 53 and 38 μg/g ash wt and for children aged 0-19 years of 74 and 62 μg/g ash wt respectively.[11] No samples were available for comparison using our present analytical techniques.

Fig 2   *Relation between placental lead and duration of occupational exposure (normal births).*

Bones

Lead values in 10-rib samples from stillbirths ranged from 0·73 to 3·75 μg/g with a mean value of 2·01 ± 0·97 μg/g (wet wt basis). The lead values in skull-bone samples from seven of the same births ranged from 0·36 to 2·53 with a mean of 1·28 ± 0·66 μg/g. The ratios of wet:dry weight (1·85) and wet weight: ash (3·23) were in the expected range. A series of cot-deaths (age less than 1 week) and two infant deaths (age less than 18 weeks) yielded rib-bone lead values of 0·72 ± 0·46 μg/g, which was significantly lower than the stillbirth ribs ($p < 0·001$).

Conclusion

Women working in the Stoke-on-Trent area in jobs exposed to lead have higher antenatal blood lead concentrations and higher than average placental-lead concentrations. In addition, concentrations of lead in stillbirth tissues appear to be high and, in the case of rib-bone lead where we were able to make direct comparisons, there is an accumulation of lead. It is not possible to say, however, whether the occupational lead exposure caused any of the stillbirths that were examined or whether the high lead values were consequential to the fetal death. Nevertheless, even in the absence of any direct causal evidence of the subclinical effects of lead, we would regard it as only prudent to ensure that wherever possible lead is removed from the environment of the pregnant worker. The ethical implications with respect to the continuation of employment of women in such an environment have been discussed, particularly in American reports.[12][13] A consultative document has been issued by the Health and Safety Commission[14] in Britain in which proposals are made that, "In order to safeguard the developing fetus from lead (1) pregnant women should be suspended from lead work and (2) a woman of child-bearing capacity should not be employed in lead work when her blood lead concentration exceeds 1·9 μmol/l."

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