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## LETTERS

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## Acute otitis media after forceps delivery

While running an ambulatory paediatric clinic, the mother of a crying baby wondered whether forceps delivered babies were more prone to otitis than other babies. To our knowledge, this association has not been reported. But, on the other hand, facial nerve injury is more common among those delivered by forceps[1]; it is caused by compression of diploic bone of the mastoid process where the facial nerve is located superficially.[2] It is clear that this kind of extraction applies some pressure over the ear of the baby. The head vulnerability is well known: long term consequences have been recognised after mild head injury.[3]

To estimate the differences of proportion of acute otitis media (AOM) between children delivered by forceps and other babies we conducted an electronic medical records review of all singleton children attending a primary care paediatric clinic, born full term from 1 January 1996 to 31 December 2004. Table 1 shows the characteristics of the children. Sex was comparable, birth weight of non-operative vaginal delivery babies (NO) was lower, vacuum assisted babies (VA) were older at AOM diagnosis, and rates of neonatal admission were not comparable.

AOM was diagnosed by a history of acute onset of signs and symptoms and otoscopic examination of the eardrum.[4]

Of 1449 deliveries, there were: 754 NO, 217 F, 52 VA, and 426 CS (caesarean sections). AOM had been recorded in 234 (31.0%) of children born by NO, 87 (40.0%) of F, 15 (28.8%) of VA, and 124 (29.1%) of CS.

The analysis showed that forceps delivered babies were associated with an increase in proportion of AOM (odds ratio (OR) 1.48; 95% confidence intervals (CI) 1.08 to 2.03; $p = 0.015$) compared with NO.

There were no differences in AOM proportions between VA and NO (OR 0.85; 95% CI 0.46 to 1.58; $p>0.3$). And there were no differences in AOM proportions between CS and NO (OR 0.91; 95% CI 0.70 to 1.18; $p>0.3$).

Our finding could be confirmed or discarded by ongoing longitudinal studies. If it is confirmed, it will strengthen the resolve to pay careful attention to the comments of our patients.

It is already known that VA is at least as safe as forceps for the mother and the neonate. Long term consequences of operative vaginal delivery need to be explored: a prospective study should be undertaken to find if this association really exists.

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**Table 1** Patient characteristics

| Feature | Non-operative delivery (n = 754) | Forceps delivery (n = 217) | Vacuum delivery (n = 52) | Caesarean section (n = 426) |
|---|---|---|---|---|
| Birth weight mean (SD) | 3315.2 (400.7)* | 3429.3 (388.2) | 3496.2 (416.7) | 3383.4 (467.8) |
| Ratio male:female | 1.08 | 1.18 | 1.42 | 1.34 |
| Newborns admitted to special care (%) | 24 (3.1)* | 11 (5.0)* | 8 (15.3)* | 25 (5.8)* |
| Age, months, at AOM diagnosis mean (SD) | 18.2 (12.9) | 17.4 (10.8) | 23.7 (12.6)* | 18.9 (13.0) |

*$p<0.05$.

## Education and mortality: a role for intelligence?

In their report van Oort and others[1] clearly describe and empirically examine the potential mediating factors—broadly categorised by the authors as material, psychosocial, and behavioural—that might account for the well established inverse education-mortality gradient. We believe the role of intelligence (denoted here as IQ, and defined as a person's ability to learn, reason, and solve problems[2]) warrants mention, given its link with all cause mortality, other somatic health outcomes, and at least two (material and behavioural factors) of the aforementioned pathways.[3]

Recently reported findings from a series of cohort studies show an inverse association between IQ, assessed using psychometric tests, and later death, whether this ''exposure'' was quantified in childhood, early adulthood, middle age, or older age.[3] Although fewer data are available for cause specific outcomes, similar gradients have also been reported for childhood assessed IQ in relation to adult risk of ischaemic heart disease (but not stroke), selected cancers, and accidents.[3] IQ has also been linked with behavioural factors, including smoking patterns, such that adults with higher early life IQ scores are more likely to subsequently give up the habit than their lower performing counterparts.[4] While these findings are comparatively recent, the suggestion that early life IQ might influence later life material measures of socioeconomic position—particularly income,[2] but also car and house ownership—has a long research tradition.

Based on these findings and using the authors' own conceptual model (figure 1; page 215[1]), IQ may be regarded in at least three ways. Firstly, education may be a proxy for IQ. However, this is not to ignore potential interplay between IQ and education, such as mediation or moderation (effect modification), and the influence this might have on health. Secondly, in a related point, IQ might generate individual differences in educational attainment, in addition to being independently associated with material and behavioural factors. In studies that adjusted for education in the IQ-mortality relation, results are inconsistent with some investigators finding pronounced attenuation, while others do not.[3] Thirdly, given that education may represent a cognitive archaeological ''record'' of pre-adult insults (for example, illness, nutritional privation, poor living conditions, psychosocial stress), it is probable that IQ, given its metric properties (education is normally quantified categorically), is a more sensitive marker of such exposures.[5]

In summary, there is new and persuasive evidence to link early cognitive ability *and* education with later health outcomes. Understanding the mechanisms that may underlie these associations should include an examination of whether education may be a partial mediator of, or a surrogate for, IQ differences.

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## Authors' response

We appreciate the comments of Batty and Deary on our paper reporting on the explanations of educational inequalities in mortality. We agree with them that there is evidence linking early cognitive ability to later health outcomes and that inclusion of education and early life intelligence may be important for studies of health inequalities. However, the latter seems to be dependent on the purpose of the study. It was the aim of our study to contribute to the debate on the importance of different mediating factors in the causal pathway of educational inequalities in health, and to provide more guidance to policy recommendations to reduce these inequalities, and it is less clear if inclusion of early life intelligence would have contributed much to these aims.

We believe that the most appropriate position for early life intelligence in our conceptual model would be preceding educational achievement. In this position early life intelligence could contribute to the understanding of differences in educational achievement. In contrast, its contribution to the understanding of causal pathways from education to health would be limited.

Inclusion of intelligence in a study like ours would increase understanding of the ''core'' roots of inequalities in health and should therefore be supported. Using the same arguments as the authors did however, this can be also be said from other factors, such as birth weight, parental socioeconomic position, and neighbourhood deprivation.[1–3] These factors are also related to educational achievement, and do have mediating and interacting effects.

Finally, the connection of early life intelligence to policy recommendations is less straightforward than this is for education: recommending improvement of an individual trait as early life intelligence may be more difficult than recommending improvement of educational achievement, for example by policies that maximise the chance that children will remain at school.[4]

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## Offspring sex ratios of people exposed to electromagnetic fields

Saadat[1] wrote that there has only been one study on the association between human offspring sex ratio (proportion male) at birth and parental exposure to electromagnetic fields (EMF). He substantiated this claim with a reference to Irgens *et al*.[2] However, I[3] cited six other such studies.[4–9] These studies, although not unanimous, cumulatively suggest that both exposed men and exposed women tend to produce significant excesses of daughters. This suggestion is not much changed by the comparatively small samples adduced by Saadat.[1] The point may be illustrated by considerations of standard power analysis.[10] Suppose that you wished to test that exposure has the effect of reducing the offspring sex ratio by, say, 10% (viz from an expected value of 0.515 to 0.465). Then, to stand 8 chances in 10 of detecting a difference at the 0.05 level (one way), you would require equal sized samples (of offspring of exposed and non-exposed subjects) each numbering 1236. The number of offspring of Saadat's[1] sample of exposed subjects was 110.

In my letter I cited[3] evidence that exposure of men to EMF is associated with fatigue, headaches, dizziness, impaired memory, nausea, loss of strength in limbs, respiratory difficulties, sleep disturbances, and reduced libido. Such reports may be dismissed as subjective. However I also cited reports that exposure to EMF was associated with reduced sperm counts in men and rats: and of reduced testosterone levels in rats. And more recently, men's exposure to radiofrequency radiation has reportedly been linked to a lowered testosterone/gonadotrophin ratio.[11] And there are good grounds for suspecting this hormone profile to be causally associated with low offspring sex ratios.[12 13] More data are urgently needed on the sexes of offspring of men and women who have been exposed to EMF.

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## Author's reply

I thank Dr James for his letter. He may well be correct for the citations of the related studies and also the estimation of the sample size. However, I wish to make some comments.

Not only in my report,[1] but also in other reports[2–4] the sample size was much lower than that estimated by James. Among published data, the article of Guberan *et al*[5] was based on 1781 births (508 and 1273 births from exposed and unexposed pregnancies, respectively), which is a comparatively large sample size and it is near to the required sample size, calculated by James. Guberan *et al* found that there was no statistically significant difference between exposed and unexposed pregnancies for offspring sex ratio.[5] Irgens *et al* reported that offspring sex ratio of women in industries with electromagnetic fields was significantly reduced; while in men exposed to the fields the ratio did not show significant difference.[6] On the other hand, experimental design studies showed that when rodents (mice and rats) were exposed to electromagnetic fields, the offspring sex ratio significantly increased[7] or remained unchanged,[8 9] compared with their