SHORT REPORT

Water fluoridation, stillbirths, and congenital abnormalities

R Lowry, N Steen, J Rankin

A part from an increase in dental fluorosis, recent reviews of water fluoridation found little evidence of adverse effects. Several studies looked at congenital abnormalities, two of which found a negative effect of water fluoridation, although overall the evidence was inconclusive. The reviews also raised the issue of the paucity of published data on congenital abnormalities, the possibility of publication bias, and the need for more data; and the age of existing research, the poor quality, and the failure to control for confounding factors.

METHODS AND RESULTS

Our study was based on residence within the boundaries of the former Northern health region in the north east of England, with a population of 3 million and about 35,000 deliveries per year. Artificially fluoridated and non-fluoridated areas were chosen with similar populations, socio-demographic characteristics, termination rates, and fluoride supplement regimens. Cases were identified from two population based registers, the Northern Perinatal Mortality Survey (PMS) and the Northern Congenital Abnormality Survey (NorCAS). All stillbirths occurring between 1 January 1989 and 31 December 1998 were identified from the PMS. All cases of a congenital abnormality with a final postnatal diagnosis of a trisomy (trisomy 21, 13, and 18 only, ICD-9 codes 758.0, 758.1, 758.2), a neural tube defect (as defined by the EUROCAT system of classification, ICD-9 codes 740.0, 740.1, 740.2, 741.0, 741.9, and 742.0) or facial cleft (cleft palate, cleft lip with or without cleft palate, Pierre Robin syndrome, ICD-9 codes 749.0, 749.1, 749.2, 756.03) were identified from the NorCAS. Cases resulting in a miscarriage were excluded from the analysis as it is not possible to ascertain the total number of miscarriages for the denominator.

Denominator birth data were obtained from the Office for National Statistics (ONS, formerly the Office of Population Census and Surveys) (OPCS Birth Statistics). Cases were grouped by year of delivery.

The number of defects was analysed using generalised linear models with a Poisson error structure and log link function. To take into account the different size of each geographical area, the natural logarithm of the total number of births was declared as an offset. The package MLwiN was used to model variation between areas and variation between occasions as random effects with occasions nested within areas.

The analysis was undertaken in two stages. Firstly, we hypothesised that if fluoridation influenced the risk of congenital abnormality then the largest difference would be between areas with no fluoridation (<0.3 parts fluoride per million water) and areas with full fluoridation (>0.9 parts fluoride per million water). We considered each type of abnormality separately and the difference between non-fluoridated and fully fluoridated areas was fitted as a fixed effect in the multilevel model. Results are given in the form of odds ratios—the relative odds of defects in a fluoridated area compared with those in a non-fluoridated area.

The second stage was to consider variation in congenital abnormality across all the geographical areas. To reduce the number of comparisons being made, we examined all abnormalities simultaneously—fitting type of abnormality as a fixed effect in the general linear model. Level of fluoridation was again fitted as a fixed effect (a three level categorical factor). We then determined the overall level of congenital abnormality in fully fluoridated and non-fluoridated areas compared with partially fluoridated areas (which was set up as the reference category). Results are again given in the form of odds ratios.

To take account of recognised potential confounding factors, data were also examined in relation to maternal deprivation and district of residence, and maternal age.

Our study found no significant associations at the 5% level between these outcomes and water fluoride level (table 1). When the variation in the rate of congenital abnormalities across all areas was examined, type of abnormality was highly significant. In particular the incidences of all other congenital abnormalities were much lower than the incidence of stillbirths. Overall, there was evidence that the incidence of congenital abnormalities was greater in 1994–98 than in 1989–93 (OR 1.13, 95% CI 1.06 to 1.20). The incidence was slightly greater in fully fluoridated areas compared with non-fluoridated areas (OR 1.09, 95% CI 0.98 to 1.22) but it was also greater in non-fluoridated areas than partially fluoridated areas (OR 1.12, 95% CI 1.00 to 1.25).

There was no difference in material deprivation or mean maternal age between fluoridated and non-fluoridated areas.

CONCLUSION

This analysis indicates that there is no evidence that fluoridation has had any influence on the rate of congenital abnormalities or stillbirths in the north east of England. Our study adds to the available evidence on fluoridation.

**Table 1** Relative odds of birth defects in fluoridated areas compared with non-fluoridated areas

<table>
<thead>
<tr>
<th>Condition</th>
<th>Fluoridated areas compared with non-fluoridated areas</th>
<th>Odds ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stillbirths</td>
<td></td>
<td>1.06</td>
<td>(0.91 to 1.24)</td>
</tr>
<tr>
<td>Congenital abnormality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All trisomies</td>
<td></td>
<td>1.11</td>
<td>(0.86 to 1.43)</td>
</tr>
<tr>
<td>Downs syndrome</td>
<td></td>
<td>1.05</td>
<td>(0.79 to 1.41)</td>
</tr>
<tr>
<td>Neural tube defects</td>
<td></td>
<td>0.82</td>
<td>(0.62 to 1.09)</td>
</tr>
<tr>
<td>Clefts</td>
<td></td>
<td>0.63</td>
<td>(0.46 to 0.86)</td>
</tr>
</tbody>
</table>
ACKNOWLEDGEMENTS

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REFERENCES