Health effects of passive smoking · 1

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Parental smoking and lower respiratory illness in infancy and early childhood

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Abstract

Background – A systematic quantitative review was conducted of evidence relating parental smoking to acute lower respiratory illness in the first three years of life.

Methods – Fifty relevant publications were identified after consideration of 692 articles selected by electronic search of the Embase and Medline databases using keywords relevant to passive smoking in children. The search, completed in April 1997, identified 24 studies ascertaining illnesses in a community setting, including five surveys of schoolchildren with retrospective ascertainment of early chest illness, and 17 studies of admissions to hospital for lower respiratory illness in early life. Thirty eight studies were included in a quantitative overview using random effects modelling to derive pooled odds ratios.

Results – The results of community and hospital studies are broadly consistent, with only one publication reporting a reduced risk among children of smokers. The pooled odds ratios were 1.57 (95% CI 1.42 to 1.74) for smoking by either parent and 1.72 (95% CI 1.55 to 1.91) for maternal smoking. There is a significantly increased risk of early chest illness associated with smoking by other household members in families where the mother does not smoke (1.29, 95% CI 1.16 to 1.44). The associations with parental smoking are robust to adjustment for confounding factors, and show evidence of a dose-response relationship in most studies in which this has been investigated.

Conclusions – The relationship between parental smoking and acute lower respiratory illness in infancy is very likely to be causal. Although it is impossible to distinguish the independent contributions of prenatal and postnatal maternal smoking, the increased risk associated with smoking by other household members suggests that exposure to environmental tobacco smoke after birth is a cause of acute chest illness in young children.

Keywords: parental smoking, tobacco smoke pollution, lower respiratory illness, infancy, childhood.

Two articles published in the Lancet in 1974 alerted readers to a possible link between parental smoking and the risk of lower respiratory illness in infancy. Although adverse effects from exposure of children to environmental tobacco smoke had been suggested previously, the association with acute chest illness was of immediate and continuing interest because of the suspected long term consequences of early episodes for lung growth, chronic respiratory morbidity in childhood, and adult chronic obstructive lung disease.

During the last two decades many epidemiological studies have reported upon the association of parental smoking and respiratory diseases throughout childhood. In this, the first of a series of systematic and quantitative reviews of health effects of passive smoking, we summarise the evidence relating specifically to acute lower respiratory illnesses in the first two or three years of life. Studies of asthma incidence, prognosis, and severity will be reviewed separately. Although there is some overlap with the studies of early wheezing illness included in this paper, the latter display certain characteristics which are distinct from asthmatic episodes of later onset. The problems of applying precise diagnostic labels to many infants with lower respiratory illness further justifies the inclusion of early wheezing illnesses in this review.

Methods

Published papers, letters, and review articles relating to passive smoke exposure in children were selected by an electronic search of the Embase and Medline databases. The Medline search strategies used were:

1. To identify all passive smoking references:
   a. MESH heading “tobacco smoke pollution”;
   b. {passive or second-hand or second hand or involuntary or parent$ or maternal or mother$ or paternal or father$ or household$} and {smok$ or tobacco$ or cigarette$} where $ = wild card;
   c. combine (a) or (b).
(2) To restrict to children:
(a) search (c) above to all relevant age
groups;
(b) search (c) above for paediatric$ or
pediatric$ or infant$ or child$ or
adolescen$;
(c) combine (a) or (b).

The Embase strategy used was based on text
word searches of titles, keywords, and abstracts
for items listed in 1(b) and 2(b) above.
This search, completed in April 1997,
yielded 3625 references of which 1593 con-
tained keywords relevant to respiratory or
allergic disease. These 1593 abstracts were re-
viewed and 692 were identified as of possible
relevance to the assessment of respiratory
health effects; 472 (68%) of these had been
published during 1990–96, the remainder dur-
ing 1972–89.
Among 75 publications which were con-
sidered in detail as of possible relevance to
illnesses in infancy, 50 were included in this
review, and 38 studies were included in quanti-
tative meta-analyses: 10 case-control studies,
21 longitudinal studies, two controlled trials,
and five cross sectional surveys of children of
school age. The latter were included because
they related parental smoking to a retrospective
history of chest illness before two years of age
(obtained by the American Thoracic Society
children’s questionnaire$). No additional re-
ditions could be derived from each study relating to the odds
to chest illness among children with and
without smokers in the family, and separately
for children exposed and unexposed to ma-
ternal smoking, whether during pregnancy or
postnatally. We also addressed specifically the
effect of smoking by other household members
(usually the father) for children whose mother
did not smoke. Not all these indices could be derived from each study. The most widely
derived measures of effect related to either
parent smoking (compared with neither) and
the effect of mother smoking (compared with
father only or neither parent smoking). Few
studies distinguished in any detail between pre-
natal and postnatal maternal smoking, but
those which did are discussed below.
The odds ratio was chosen as a measure of
association which can be derived from all types
of study (case-control, cross sectional and lon-
gitudinal). In general, odds ratios and their
95% confidence intervals were calculated from
data in published tabulations using the actual
numbers of subjects or numbers derived ap-
proximately from percentages of published col-
umn or row totals. This approach allowed flexibilit y in combining categories of household
smoke exposure for comparability across stud-
ies. Where the numbers of subjects were not
shown, the published odds ratio and its 95% con-
fidence interval were used. A few papers
quoted an incidence rate ratio rather than an
odds ratio, and these are identified in the sum-
mary tabulations. Information was also sought
on the extent to which the effects of parental
smoking were altered by adjustment for po-
tential confounding variables, and whether
there was evidence of a dose-response re-
lationship—for instance, to the amount smoked
by either parent. Only one paper from each
study (usually the most recently published) was
included in quantitative meta-analyses. How-
ever, in some studies information from other
papers contributed to the assessment of con-
firming or dose-response relationship.

Where quantitative meta-analysis was con-
sidered appropriate, odds ratios were tested for
heterogeneity using the technique of Breslow
and Day.$ The heterogeneity tests were often
statistically significant, implying that a simple
‘fixed effect’ pooling of the logarithms of the
odds ratios (using weights inversely pro-
portional to their variances$) may be in-
appropriate. Odds ratios were therefore pooled
using a ‘random effects’ model which makes
allowances for heterogeneity of effect between
studies. In practice, this approach produces estimates similar to those of standard methods
but allows regression models to be fitted, if
desired, in order to explain heterogeneity be-
tween studies.$

The random effects model was implemented
by using iteratively reweighted least squares
regression, adapting a method previously de-
volved for geographical mortality studies.$
This approach has the practical advantage that
only the log odds ratios and their standard
errors are required, and not the raw data from
each individual study. The computing algo-
rithm used for this purpose is shown in the
Appendix

The log odds ratios were used as dependent
variables and their standard errors were used to
estimate the component of variance between
studies attributable to sampling variation. Any
non-sampling variation, representing hetero-
genesis of passive smoking effects between
studies, was assumed to be normally distributed
with a mean of zero. 95% confidence intervals
for the pooled odds ratio were calculated by
assuming that the estimated log odds ratio
divided by its standard error follows a $t$
distribution on $(n − 2)$ degrees of freedom where
$n$ is the number of studies pooled. In practice,
this will produce confidence intervals which
are too wide when $n$ is small and there is
little heterogeneity. For this reason, we do not
present results from random effects models
where fewer than five studies are pooled.

Results

COMMUNITY STUDIES OF LOWER RESPIRATORY ILLNESS

Twenty one studies$^{11–31}$ were identified in which
lower respiratory illnesses had been ascertained
in a community or ambulatory clinic setting and
related to parental smoking (table 1). These
comprised 14 longitudinal studies, two con-
trolled trials, two case-control studies, and
three retrospective prevalence surveys. In seven
studies$^{12–14,19,20,22,23}$ all lower respiratory diag-
noses were combined; four$^{11,15,17,21}$ contributed
information on bronchitis and pneumonia, and
two$^{16,18}$ concentrated on illnesses diagnosed
as bronchiolitis. Ten studies$^{15,17,24–31}$ focused
Specifically on illnesses associated with wheezing. Two publications contributed independent data on both bronchitis/pneumonia and wheezing illnesses. The results of these studies are summarised in Table 2 and Figs 1–3. All found an increased risk associated with parental smoking, including by the father only where this was assessed. Table 3 presents the results of meta-analyses, pooling the results of studies of early wheezing separately from those of unspecified lower respiratory illness, bronchitis, bronchiolitis or pneumonia. Although the effect of either parent smoking is similar for these two outcomes, maternal smoking appears to be relatively more important, and paternal smoking perhaps less important in studies which have ascertained wheezing illness specifically.

**STUDIES OF HOSPITAL ADMISSIONS FOR LOWER RESPIRATORY ILLNESS**

Twelve publications were identified relating to hospital admissions for lower respiratory complaints in early life. Three did not differentiate between different forms of chest illness, four related to bronchitis and/or pneumonia, and five focused on admission for wheezing illness or for bronchiolitis with or without confirmation of respiratory syncytial virus infection. One cohort study included here presented detailed findings only in relation to hospital admissions up to five years of age, but tabulations by age at admission suggest a similar strength of association between maternal smoking and admission for bronchitis or pneumonia at all ages from birth to five years. The results for all ages are therefore included in the meta-analyses.

The results of these studies are summarised in Table 2 and Figs 1–3. All except one study found an increased risk associated with parental smoking. The results of meta-analyses are summarised in Table 3. The pooled odds ratios are similar in magnitude to those derived from community studies.

Two case-control studies from South Africa and the United Kingdom were excluded from the quantitative overview because they present results only for a smoky atmosphere in the home. In the South African study the principal source of exposure was wood smoke. In the British study infants admitted with suspected bronchiolitis were almost three times more likely to have a smoky atmosphere recorded by health visitors on a visit to the home at one study.
month of age (odds ratio 2.93, 95% CI 1.95 to 4.41).

STUDIES OF UPPER AND LOWER RESPIRATORY ILLNESS COMBINED

Five studies\(^4\)\(\text{a}\)\(\text{b}\) presented data relating parental smoking to all respiratory illness without distinguishing between upper and lower respiratory diagnoses (table 1). Two of these\(^4\)\(\text{a}\)\(\text{b}\)\(\text{c}\) were based in the community, and three relate to hospital admissions for respiratory illness.\(^4\)\(\text{a}\)\(\text{b}\)\(\text{c}\) One of the latter studies\(^4\)\(\text{c}\) synthesised the results of three previous papers.\(^5\)\(\text{a}\)\(\text{b}\)\(\text{c}\)\(\text{d}\)\(\text{e}\)

The findings of these studies, summarised in table 2, are broadly in line with those studies which have concentrated on lower respiratory illnesses, and their inclusion in the overall meta-analysis changes the estimates of effect only slightly (table 3).

INDEPENDENCE OF CONFOUNDERING

About half of the cohort studies, but only a quarter of the case-control or cross sectional studies, presented estimates of the effects of parental smoking, both before and after adjustment for potential confounding variables. Although a different range of confounding variables was controlled in each study, the effects of parental smoking are little altered by adjustment for measured confounders (table 4).

DOSE-RESPONSE RELATIONSHIPS

Nine of the 12 cohort studies which present evidence relating to dose-response within smoking families found a statistically significant relationship, either to the number of smokers or to the amount smoked in the household, or specifically by the mother (table 2). A formal meta-analysis of the dose-response relationship

<table>
<thead>
<tr>
<th>Reference</th>
<th>Outcome</th>
<th>Cases</th>
<th>Controls</th>
<th>Odds ratios (95% CI) for smoking by:</th>
<th>Dose-response present?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>Mother</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Bl/Pn</td>
<td>239</td>
<td>1835</td>
<td>1.96 (1.38 to 2.80)</td>
<td>—</td>
</tr>
<tr>
<td>12</td>
<td>LRI</td>
<td>31</td>
<td>—</td>
<td>1.25 (0.81 to 1.93)</td>
<td>—</td>
</tr>
<tr>
<td>13</td>
<td>LRI</td>
<td>221</td>
<td>—</td>
<td>1.27 (1.11 to 1.46)</td>
<td>—</td>
</tr>
<tr>
<td>14</td>
<td>LRI</td>
<td>820</td>
<td>7708</td>
<td>1.85 (1.56 to 2.20)</td>
<td>1.69 (1.47 to 1.96)</td>
</tr>
<tr>
<td>15</td>
<td>Bl/Pn</td>
<td>204</td>
<td>940</td>
<td>1.56 (1.15 to 2.12)</td>
<td>1.83 (1.35 to 2.49)</td>
</tr>
<tr>
<td>16</td>
<td>Bl</td>
<td>53</td>
<td>159</td>
<td>3.21 (1.42 to 7.25)</td>
<td>2.33 (1.19 to 4.57)</td>
</tr>
<tr>
<td>17</td>
<td>Bl/Pn</td>
<td>925</td>
<td>1302</td>
<td>1.25 (1.05 to 1.52)</td>
<td>—</td>
</tr>
<tr>
<td>18</td>
<td>Bl</td>
<td>20</td>
<td>60</td>
<td>3.86 (0.81 to 18.4)</td>
<td>—</td>
</tr>
<tr>
<td>Community studies: wheezing illnesses</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>LRI</td>
<td>221</td>
<td>—</td>
<td>1.27 (1.11 to 1.46)</td>
<td>—</td>
</tr>
<tr>
<td>14</td>
<td>LRI</td>
<td>820</td>
<td>7708</td>
<td>1.85 (1.56 to 2.20)</td>
<td>1.69 (1.47 to 1.96)</td>
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<tr>
<td>15</td>
<td>Bl/Pn</td>
<td>204</td>
<td>940</td>
<td>1.56 (1.15 to 2.12)</td>
<td>1.83 (1.35 to 2.49)</td>
</tr>
<tr>
<td>16</td>
<td>Bl</td>
<td>53</td>
<td>159</td>
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</tr>
<tr>
<td>17</td>
<td>Bl/Pn</td>
<td>925</td>
<td>1302</td>
<td>1.25 (1.05 to 1.52)</td>
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</tr>
<tr>
<td>18</td>
<td>Bl</td>
<td>20</td>
<td>60</td>
<td>3.86 (0.81 to 18.4)</td>
<td>—</td>
</tr>
</tbody>
</table>

Abbreviations as for table 1.  
\(^4\) In households where the mother did not smoke (compared with neither parent smoking).  
\(^a\) Results published as person-time incidence rates. Rate ratios, rather than odds ratios are shown.  
\(^b\) 95% confidence interval estimated as 1.00 to 3.96 for purposes of meta-analysis.  
\(^c\) Odds ratio or relative risk cited in the paper without tabulated numerical data (elsewhere, odds ratios were calculated from tabulated numbers or percentages).
is not possible. In contrast, the risk associated with both parents smoking was not substantially greater than that for either parent smoking. A comparison of both parents smoking with neither smoking was available for 11 studies and the pooled odds ratio was 1.69 (95% CI 1.37 to 2.08).

In two case-control studies, urinary cotinine levels were measured as an objective marker of tobacco smoke exposure, and in both the levels were significantly higher in the case group. These results are consistent with another small case-control study of emergency room attendances for wheezing illness which measured urinary cotinine levels but did not report in detail on parental smoking habits. None of these studies restricted their analysis to smoking families, and therefore the differences in cotinine levels may simply reflect the presence of smokers in the household, rather than evidence of a graded relationship to the amount of exposure to environmental tobacco smoke.

Figure 1 Odds ratios and 95% confidence intervals for effect of either parent smoking compared with neither smoking. The pooled odds ratios derived by fixed effects and random effects methods appear at the foot of the figure. The horizontal scale is logarithmic (base 2). Individual studies are denoted thus: circles = studies of lower respiratory illnesses; squares = studies of wheezing illnesses; diamonds = studies of upper and lower respiratory illnesses; open symbols = community studies; filled symbols = studies of hospitalised illnesses.

Figure 2 Odds ratios and 95% confidence intervals for effect of mother smoking compared with father only or neither parent smoking. Definitions of symbols as for fig 1.

EFFECT OF PARENTAL SMOKING AT DIFFERENT AGES
The early report by Colley et al suggested that the effect of parental smoking on the incidence of bronchitis and pneumonia was most marked in the first year of life (odds ratio 1.96, 95% CI 1.30 to 2.99), declining thereafter with increasing age of the child to an inverse relationship in the fifth year. Results from the Dunedin, New Zealand cohort showed a similar pattern, with a slightly greater effect in the first than the second year and little evidence of association with consultation for bronchitis or pneumonia after two years of age.

Effect on Specific Respiratory Diagnoses
Few papers have compared the effect of parental smoking on different specific clinical diagnoses, and the results are inconsistent with effects confined to tracheitis and bronchitis in one but to wheezing and pneumonia (and not bronchitis or bronchiolitis) in another. One cohort study explicitly distinguished between lower respiratory illnesses which were associated with wheezing and those which were not. The proportion of cases exposed to maternal smoking (>20 cigarettes/day) was 14% in each subgroup. This is not entirely consistent with the pooled odds ratios obtained from community studies which suggest a stronger effect of maternal smoking in studies specifically of wheezing than in those including a broader range of chest illnesses (table 3). Seven case-control studies focused specifically on bronchiolitis or illnesses associated with evidence of respiratory syncytial virus infection. These generated a somewhat stronger effect than other studies, but this may reflect positive publication bias which is discussed further below.
Table 3 Pooled odds ratios, 95% confidence intervals and heterogeneity tests from meta-analyses

<table>
<thead>
<tr>
<th></th>
<th>Either parent</th>
<th>Mother</th>
<th>Father only</th>
</tr>
</thead>
<tbody>
<tr>
<td>All studies</td>
<td>Number of studies</td>
<td>27</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>Heterogeneity χ²</td>
<td>55.1 (p&lt;0.001)</td>
<td>60.7 (p&lt;0.001)</td>
</tr>
<tr>
<td></td>
<td>Odds ratio (fixed)</td>
<td>1.49 (1.40 to 1.58)</td>
<td>1.64 (1.55 to 1.73)</td>
</tr>
<tr>
<td></td>
<td>and 95% CI (random)</td>
<td>1.57 (1.82 to 1.74)</td>
<td>1.72 (1.55 to 1.91)</td>
</tr>
<tr>
<td>Excluding studies which</td>
<td>Number of studies</td>
<td>24</td>
<td>23</td>
</tr>
<tr>
<td>include upper respiratory</td>
<td>Heterogeneity χ²</td>
<td>52.2 (p&lt;0.001)</td>
<td>51.8 (p&lt;0.001)</td>
</tr>
<tr>
<td>illness</td>
<td>Odds ratio (fixed)</td>
<td>1.46 (1.37 to 1.56)</td>
<td>1.61 (1.51 to 1.71)</td>
</tr>
<tr>
<td></td>
<td>and 95% CI (random)</td>
<td>1.57 (1.40 to 1.77)</td>
<td>1.69 (1.50 to 1.89)</td>
</tr>
<tr>
<td>Community studies of</td>
<td>Number of studies</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>wheezing illness</td>
<td>Heterogeneity χ²</td>
<td>25.7 (p&lt;0.002)</td>
<td>11.3 (p=0.040)</td>
</tr>
<tr>
<td></td>
<td>Odds ratio (fixed)</td>
<td>1.46 (1.35 to 1.58)</td>
<td>1.56 (1.43 to 1.71)</td>
</tr>
<tr>
<td></td>
<td>and 95% CI (random)</td>
<td>1.54 (1.31 to 1.80)</td>
<td>1.57 (1.33 to 1.86)</td>
</tr>
<tr>
<td>Community studies of</td>
<td>Number of studies</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>hospital admission for</td>
<td>Heterogeneity χ²</td>
<td>4.65 (p=0.325)</td>
<td>11.1 (p=0.049)</td>
</tr>
<tr>
<td>LRI, bronchitis, bronchiolitis or pneumonia</td>
<td>Odds ratio (fixed)</td>
<td>1.54 (1.30 to 1.81)</td>
<td>1.98 (1.71 to 2.30)</td>
</tr>
<tr>
<td></td>
<td>and 95% CI (random)</td>
<td>1.55 (1.16 to 2.08)</td>
<td>2.08 (1.59 to 2.71)</td>
</tr>
<tr>
<td>Hospital admission for</td>
<td>Number of studies</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>LRI, bronchitis, bronchiolitis or pneumonia</td>
<td>Heterogeneity χ²</td>
<td>22.2 (p&lt;0.002)</td>
<td>22.3 (p&lt;0.004)</td>
</tr>
<tr>
<td></td>
<td>Odds ratio (fixed)</td>
<td>1.45 (1.27 to 1.66)</td>
<td>1.54 (1.40 to 1.69)</td>
</tr>
<tr>
<td></td>
<td>and 95% CI (random)</td>
<td>1.71 (1.21 to 2.40)</td>
<td>1.53 (1.25 to 1.86)</td>
</tr>
</tbody>
</table>

# Number of studies too small for reliable random effects modelling. No significant heterogeneity of effects.

EFFECT ON SUSCEPTIBLE SUBGROUPS

The effect of parental smoking on early respiratory illness has been reported in two controlled trials,25 26 and one cohort study31 which recruited infants at high risk due to a parental history of allergy25 or prematurity.26 31 The odds ratios obtained from these studies were within the general range (table 2) and have therefore been included in the meta-analyses.

Only one study included here49 permitted a direct comparison between high and low risk infants. In two Chinese cohorts an adverse effect of household smoking on hospital admissions for respiratory disease was evident among both low birthweight (<2.5 kg) babies (odds ratio 6.87, 95% CI 0.89 to 53.0) and normal birthweight infants (1.36, 95% CI 0.96 to 1.93). There was no statistically significant effect modification by birthweight (test for interaction, p = 0.06).

PRENATAL VERSUS POSTNATAL EXPOSURE

The effects of smoking by other household members in homes where the mother did not smoke are summarised in tables 2 and 3. These are derived from three studies from China,40 40 which included no smoking mothers, and 11 from westernised countries where data were presented for smoking by the father only. The results are quantitatively consistent, and only two odds ratios are less than unity (fig 3). The pooled odds ratio obtained by meta-analysis is 1.29 (95% CI 1.16 to 1.44). In the Chinese studies this effect is independent of birthweight and a range of other confounding factors.40 49

Few studies have evaluated the effects of prenatal and postnatal maternal smoking in the same sample. In Western countries too few mothers change their smoking habits in the perinatal period to offer the statistical power to discriminate prenatal and postnatal effects reliably. For example, in the largest study based on a national British cohort48 half of the children were born to mothers who smoked in pregnancy. Only 8% of mothers who smoked during pregnancy subsequently gave up, and

The effects of non-maternal smoking on admissions to hospital for respiratory disease in Shanghai were stronger before six months of age than in children aged 7–18 months.17 However, a significantly increased risk persisted after six months of age for children exposed to more than 10 cigarettes per day in the home (incidence ratio 1.83, 95% CI 1.03 to 3.24). In the 1970 British cohort17 the effect of maternal smoking on hospital admissions for wheezing illness, bronchitis, or pneumonia was similar at all ages up to five years.
Table 4 Effect of adjustment for potential confounders

<table>
<thead>
<tr>
<th>Reference</th>
<th>Outcome</th>
<th>Exposure</th>
<th>Unadjusted odds ratio</th>
<th>Adjusted odds ratio</th>
<th>Factors adjusted for (matching variables in parentheses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Br/Pn</td>
<td>Both v none</td>
<td>2.95</td>
<td>2.78</td>
<td>FH chest symptoms, sex, siblings, sibling illness</td>
</tr>
<tr>
<td>12</td>
<td>LRI</td>
<td>None</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>13</td>
<td>LRI</td>
<td>None</td>
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<td>None</td>
</tr>
<tr>
<td>14</td>
<td>LRI</td>
<td>None</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>15</td>
<td>Br/Pn</td>
<td>Both v none</td>
<td>2.95</td>
<td>2.78</td>
<td>FH chest symptoms, sex, siblings, sibling illness</td>
</tr>
<tr>
<td>16</td>
<td>BL</td>
<td>Mother smokes</td>
<td>2.33</td>
<td>2.68</td>
<td>(Age), SES, BF, siblings, crowding, FH asthma</td>
</tr>
<tr>
<td>17</td>
<td>Br/Pn</td>
<td>Others ≥10/day</td>
<td>1.33</td>
<td>1.31</td>
<td>Sex, BW, daycare, education, cooking fuel</td>
</tr>
<tr>
<td>18</td>
<td>BL</td>
<td>Mother ≥10/day</td>
<td>1.82</td>
<td>1.74</td>
<td>FH chest illness, season of birth, daycare, crowding</td>
</tr>
<tr>
<td>19</td>
<td>LRI</td>
<td>Either parent</td>
<td>1.52</td>
<td>1.3</td>
<td>Age, sex, area, SES, sibs, domestic crowding, heating</td>
</tr>
<tr>
<td>20</td>
<td>Br/Pn</td>
<td>Other</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>21</td>
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<td></td>
<td>None</td>
</tr>
<tr>
<td>22</td>
<td>LRI</td>
<td>Other</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>23</td>
<td>LRI</td>
<td>Other</td>
<td></td>
<td></td>
<td>None</td>
</tr>
</tbody>
</table>

Community studies: wheezing illnesses

<table>
<thead>
<tr>
<th>Reference</th>
<th>Outcome</th>
<th>Exposure</th>
<th>Unadjusted odds ratio</th>
<th>Adjusted odds ratio</th>
<th>Factors adjusted for (matching variables in parentheses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>Wheeze</td>
<td>Mother ≥20/day</td>
<td>2.85</td>
<td>2.7</td>
<td>Sex, SES</td>
</tr>
<tr>
<td>16</td>
<td>Wheeze</td>
<td>None</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>17</td>
<td>Wheeze</td>
<td>None</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>18</td>
<td>Wheeze</td>
<td>Any smoking</td>
<td>1.88</td>
<td>2.4</td>
<td>Sex, SES</td>
</tr>
<tr>
<td>19</td>
<td>Wheeze</td>
<td>Mother smokes</td>
<td>2.24</td>
<td>2.2</td>
<td>Sex, low BW, FH allergy, season of birth</td>
</tr>
<tr>
<td>20</td>
<td>Wheeze</td>
<td>None</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>21</td>
<td>Wheeze</td>
<td>None</td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>22</td>
<td>Wheeze</td>
<td>Mother smokes</td>
<td>1.98</td>
<td>1.77</td>
<td>Duration of BF</td>
</tr>
</tbody>
</table>

Community studies: upper and lower respiratory illnesses

<table>
<thead>
<tr>
<th>Reference</th>
<th>Outcome</th>
<th>Exposure</th>
<th>Unadjusted odds ratio</th>
<th>Adjusted odds ratio</th>
<th>Factors adjusted for (matching variables in parentheses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>U/LRI</td>
<td>Both v none</td>
<td>1.74</td>
<td>1.54</td>
<td>Maternal age, heating fuel, sex, sibs, FH RD, daycare, SES, stress, BF</td>
</tr>
<tr>
<td>46</td>
<td>U/LRI</td>
<td>Mother smokes</td>
<td>2.43</td>
<td>2.06</td>
<td>Maternal age, heating fuel, sex, sibs, FH RD, daycare, SES, stress, BF</td>
</tr>
</tbody>
</table>

Hospital admission for lower respiratory illness

<table>
<thead>
<tr>
<th>Reference</th>
<th>Outcome</th>
<th>Exposure</th>
<th>Unadjusted odds ratio</th>
<th>Adjusted odds ratio</th>
<th>Factors adjusted for (matching variables in parentheses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Br/Pn</td>
<td>None</td>
<td></td>
<td></td>
<td>(Age), SES</td>
</tr>
<tr>
<td>2</td>
<td>BL</td>
<td></td>
<td></td>
<td></td>
<td>(Age, sex, SES)</td>
</tr>
<tr>
<td>3</td>
<td>Br/Pn</td>
<td></td>
<td></td>
<td></td>
<td>(Age, height, school)</td>
</tr>
<tr>
<td>4</td>
<td>LRI</td>
<td></td>
<td></td>
<td></td>
<td>(Age, sex, race, season, form of health insurance)</td>
</tr>
<tr>
<td>5</td>
<td>BL</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>LRI</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>Prn/BL</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>LRI</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>BL</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>Br/Pn</td>
<td>Other ≥20/day</td>
<td>2.0</td>
<td>2.4</td>
<td>Sex, BF, BW, education, maternal age, cooking fuel</td>
</tr>
<tr>
<td>11</td>
<td>Bl</td>
<td></td>
<td></td>
<td></td>
<td>(Age)</td>
</tr>
<tr>
<td>12</td>
<td>Pn</td>
<td></td>
<td></td>
<td></td>
<td>FH asthma, BF duration</td>
</tr>
</tbody>
</table>

Hospital admission for upper or lower respiratory illness

<table>
<thead>
<tr>
<th>Reference</th>
<th>Outcome</th>
<th>Exposure</th>
<th>Unadjusted odds ratio</th>
<th>Adjusted odds ratio</th>
<th>Factors adjusted for (matching variables in parentheses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>47</td>
<td>U/LRI</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>48</td>
<td>U/LRI</td>
<td></td>
<td></td>
<td></td>
<td>None</td>
</tr>
<tr>
<td>49</td>
<td>U/LRI</td>
<td>Any smoking</td>
<td>1.49</td>
<td>1.48</td>
<td>Low BW</td>
</tr>
</tbody>
</table>

FH = family history; SES = socioeconomic status; BF = breast feeding; BW = birthweight; RD = respiratory disease; other abbreviations see table 1.

* An analysis of incidence to one year of age ref. 64 shows smoking effects are independent of BF and housing.
* No unadjusted relative risk given.
* Additional adjustment for FH asthma, pets, SES in ref. 65 (incidence to one year of age).

Parental smoking and lower respiratory illness in infancy and early childhood

6% prenatal non-smokers smoked after the child was born. The rate of hospital admissions for lower respiratory illness differed between these two groups, but not significantly so (5.9% versus 3.1%, odds ratio 1.94, 95% CI 0.96 to 3.94). The effect of postnatal smoking by mothers who did not smoke in pregnancy compared with never smoking mothers was also non-significant (odds ratio 1.36, 95% CI 0.73 to 2.54), although it is interesting to note that it was consistent with the pooled effect of father only smoking in this and other studies (table 3).

One controlled intervention study has monitored the incidence of acute lower respiratory illness after an intervention designed to modify postnatal exposure to tobacco smoke. Among 581 infants followed to six months of age there was no difference in the incidence of episodes of cough, wheeze, or rattling in the chest among the intervention group (1.6 episodes per child-year) and the control group (1.5 episodes per child-year). However, the intervention was of uncertain effectiveness in reducing tobacco smoke exposure, as mean cotinine levels did not differ between the study groups despite a reduction in reported smoke exposure of infants in the intervention group.

Discussion

The direction of the association between parental smoking and lower respiratory illness is generally consistent across different study designs, methods of case ascertainment, and diagnostic groupings (table 2). Only one study from Brazil found an inverse relation (with pneumonia), but another South American study from Chile found a highly significant doubling in risk of pneumonia in the offspring of mothers who smoked. The latter could not be
included in the meta-analysis as no confidence intervals could be derived.

Some variation between studies in the size of odds ratios would be anticipated as patterns of smoking differed between countries and over time. This is reflected in statistically significant heterogeneity in many of the pooled analyses (table 3). For this reason, the summary odds ratios derived under the fixed effects assumption should be interpreted with caution. The random effects method is more appropriate in these circumstances and suggests an odds ratio of about 1.6 as the typical effect of either parent smoking on the incidence of early chest illness, whether ascertained by parental questionnaire, primary care contacts, or hospital admissions.

The papers cited were selected by mention of keywords relevant to passive smoking and children in the title or abstract. When cross-checked against previous reviews of passive smoking in children, no major omissions were identified, whereas our systematic search included relevant references not cited elsewhere. There is a possibility that our selection was biased towards studies reporting a positive association, since it is more likely that statistically significant findings would be mentioned in the abstract. Three of the higher odds ratios were derived from small case-control studies in which passive smoking was not the focus of the original research and where bibliographical bias may have operated. The slightly higher pooled odds ratios obtained by this random effects method than by the fixed effects method (table 3) reflects greater weight assigned by the random effects approach to these small studies with relatively large odds ratios. On the other hand, inclusion of the large Chinese studies in the meta-analysis of the effect of either parent smoking will have had a conservative effect due to the absence of maternal smoking in these communities.

The nature of the common lower respiratory tract illnesses of infancy remains a subject of uncertainty and debate. Although many appear to be triggered by viral infections, there is evidence of premorbid susceptibility related to lung function abnormalities detectable from birth. Many early wheezing episodes, including bronchiolitis, probably form part of this spectrum of viral illnesses, although others may be the first evidence of more persistent childhood asthma with associated atopic manifestations. Respiratory viruses are isolated with equal frequency from infants in smoking and non-smoking households. The effect of parental smoking on the incidence of wheezing and non-wheezing illnesses appears similar, suggesting a general increase in susceptibility to clinical illness on exposure to respiratory infections, rather than influences on mechanisms more specifically related to asthma. Two such characteristics — allergic sensitisation and bronchial hyperresponsiveness — will be considered in detail in future reviews in this series.

The pooled results from families where the mother does not smoke suggest that this effect of parental smoking is at least partly due to postnatal (environmental) exposure to tobacco smoke in the home. The somewhat stronger effect of smoking by the mother than by other household members may be related to a higher degree of postnatal exposure from the mother as principal care giver, although there is insufficient evidence to exclude a specific adverse effect of maternal smoking during pregnancy, perhaps through its effect on intrauterine lung development.

The effect of parental smoking is largely independent of confounding variables where these have been measured, suggesting that residual confounding by other factors is unlikely to be important. Thus it seems to be the smoking, rather than the family in which people smoke, which is the influential factor. It is therefore reasonable to conclude, as have recent overviews, that there is a causal relationship between parental smoking and acute lower respiratory illness, at least in the first two years of life.

Appendix
Algorithm for random effects meta-analysis and meta-regression using GLIM

\$units 27 ! Set to number of studies included in ! meta-analysis
\$var OR LCL UCL $read OR LCL UCL
\$cal LNOR=%log(OR):
\$cal SE=%log(UCL) - %log (LCL))/2
\$cal TAU2=SE**2 ! Variance of each log odds
\$cal W = 1/weight W
\$var LNOR
\$fit $display e! $ Fits ordinary least squares estimate
\$giving equal weight to each study

\$mac IN ! Calculates initial value for between ! study variance SIGSQ
\$cal %S=%CU((%YV - %FV)**2 - TAU2)/SL
\$print 'INITIAL SIGSQ = ' %S
\$endmac

\$mac SIG ! Carries out one iteration to recalculate ! SIGSQ
\$cal %R=%S
\$cal %G=%CU((%YV - %FV)**2)/(1 + TAU2/ \%R)**2)%CU(1/(1+TAU2/%R))
\$cal %E= (%S - %R) * 100/%S: %A=%A - 1
\$endmac

\$mac REFIT ! Refits model with weights combining ! SIGSQ and TAU2
\$cal %G=%F
\$cal W=1/(%S + TAU2) $scale 1 $fit
\$display e
\$print 'NEW CHI-SQUARE, D.F.' %X2 %DF
\$endmac

\$mac ITER ! Calls SIG repeatedly to produce new ! SIGSQ (20 calls usually sufficient ! for convergence) and then refits ! model by calling REFIT
\$cal %A=20 $while %A SIG
\$use REFIT $print 'n = ' %SL 'NEW SIGSQ = ' %S
\$endmac

\$use IN
\$use ITER ! Call repeatedly until convergence ! (five calls usually sufficient).
Parental smoking and lower respiratory illness in infancy and early childhood

This review was commissioned by the UK Department of Health. The views expressed are those of the authors and are not necessarily those of the Department of Health. We are indebted to Jenny Taylor and Claire Chazot for their diligent work assembling the relevant literature, and to lain Carey for assistance with production of figures.


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D P Strachan and D G Cook

Thorax 1997 52: 905-914
doi: 10.1136/thx.52.10.905

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