Compelling evidence suggests a causal relation between parental smoking during and after pregnancy and adverse effects on respiratory health in the offspring. The authors’ aim in this study was to disentangle the effects of prenatal and postnatal smoking on early childhood respiratory health. Most parents who smoke during pregnancy continue to smoke postpartum, and it is difficult to identify sufficiently large subgroups of children who were exclusively exposed in utero or after birth. This study was based on the first 22,390 children born between 2000 and 2004 in the Norwegian Mother and Child Cohort, a pregnancy cohort designed to eventually include 100,000 pregnancies. Data were collected through detailed questionnaires administered at various stages of pregnancy, starting in early pregnancy. Because of the large study population, the authors were able to disentangle the pre- and postnatal effects of parental smoking on wheeze and lower respiratory tract infection in the children’s first 18 months of life. They found maternal smoking in pregnancy to be an independent risk factor for wheeze and respiratory infection. Postnatal paternal smoking was also associated with these outcomes, independently of maternal smoking in pregnancy.

Respiratory tract symptoms and respiratory diseases are common in childhood (1, 2) and represent a major burden for children, parents, and society in terms of quality of life, use of public health resources, and lost workdays (3). Massive documentation supports a positive association between early-life exposure to parental smoking and increased susceptibility to respiratory diseases in childhood (1, 3–8). In several studies, investigators have attempted to separately assess the prenatal and postnatal effects of parental smoking on wheeze and lower respiratory tract infection (LRTI) among preschool children (9–21). However, because few parents change their smoking habits during the perinatal period, there are methodological challenges involved in separating these effects (22). Only a few cohort studies have allowed for direct comparisons between the effects of in utero and postnatal exposure to cigarette smoke on LRTI and wheeze in preschool children (9, 11–16, 18–20). Most of these studies have had weaknesses related to sample size or subject selection. Finding sufficiently large subgroups of children with contrasts in pre- and postnatal exposure has been difficult (14, 22–24).

The Norwegian Mother and Child Cohort Study (MoBa) is a large, population-based cohort study which will eventually include 100,000 pregnant women (25). Questionnaires are administered at various stages during pregnancy and after birth. For the current analysis, our study population consisted of children born in 2000–2004 to mothers included in the MoBa Study from 1999 to 2004. We used data...
from the first 22,390 children followed from fetal life to the age of 18 months. We assessed children’s exposure to different patterns of parental cigarette smoking during and after pregnancy as risk factors for LRTI and wheeze.

MATERIALS AND METHODS

Subjects

Data were collected at the Norwegian Institute of Public Health as part of the MoBa Study. Our subjects were the first 22,390 children in the study (25) for whom questionnaires were obtained up to 18 months of age. In brief, MoBa is a cohort study of pregnancy that was started in 1999, with the aim of including 100,000 pregnant women by the end of 2007. By the end of 2005, more than 60,000 pregnancies had been included. The majority of all pregnant women in Norway are invited to participate, and the response rate is approximately 44 percent. Pregnant women are recruited through a postal invitation sent out during weeks 13–17 of pregnancy, in connection with the routine ultrasound examination offered to all pregnant women in Norway at 17–18 weeks of gestation. The MoBa Study includes all geographic parts of Norway, and both rural and urban areas are represented.

Participating women receive three questionnaires during pregnancy. Questionnaires regarding general health issues are administered at inclusion (questionnaire 1) and in week 30 of pregnancy (questionnaire 3). (Questionnaire 2 is a food frequency questionnaire and was not relevant to this paper.) Additional questionnaires are administered when the child is 6 months old (questionnaire 4), 18 months old (questionnaire 5), and 3 years old (questionnaire 6). The children will be followed into adulthood. The questionnaires cover a variety of issues regarding parents and children, with detailed questions on health, socioeconomic status, nutrition, environmental exposures, and familial and psychological factors before, during, and after pregnancy. The questionnaires administered after birth also cover the child’s general health and development, environmental exposures, and a variety of symptoms and diseases.

Pregnancy and birth records from the Medical Birth Registry of Norway (26) are included in the MoBa database. These records are filled in by midwives and are mandatory. The Medical Birth Registry of Norway provides information about pregnancy outcomes, complications in pregnancy, and the neonatal period.

The MoBa Study has been approved by the regional committee for ethics in medical research and the Data Inspectorate. This analysis was based on version 2 of the MoBa quality-assured data files, made available for research in April 2006. Our study population included all children who had reached 18 months of age and for whom all four questionnaires (two administered in pregnancy (questionnaires 1 and 3), one at 6 months (questionnaire 4), and one at 18 months (questionnaire 5)) had been processed.

Health outcomes

The main health outcomes investigated were LRTI and wheeze as reported responses on the questionnaires at 6 and 18 months of age. The questionnaires can be viewed at the MoBa website (27). LRTIs included respiratory syncytial virus, bronchiolitis, bronchitis, and pneumonia. We classified hospitalization for any of these conditions as being hospitalized for LRTI. Wheeze was defined as chest congestion or whistling/wheezing in the chest between 6 and 18 months of age. The questionnaire administered at age 6 months did not ask about episodes of wheeze occurring before age 6 months. Outcomes were treated as dichotomous. LRTIs and hospitalizations for LRTI were compared with no episodes of LRTI.

Exposure to tobacco smoke products

Smoking data were obtained from questionnaires 1, 3, and 4, covering pregnancy and the first 3 months postpartum. We used the mother’s responses regarding both her smoking and smoking by the child’s father. We classified children as exposed to maternal smoking if their mothers reported smoking more than zero cigarettes per day or reported occasional smoking. For exposure during pregnancy, children whose mothers reported smoking at any point were counted as exposed. Exposure to paternal smoking in utero was defined as having a father who smoked either daily or occasionally at any point during the mother’s pregnancy. Postnatal exposure was defined as any parental smoking during the first 3 months postpartum.

We created a variable for the number of cigarettes smoked per day by the mother during pregnancy from the mean number of cigarettes per day reported on the three questionnaires covering the pregnancy. Parents who were occasional smokers were assigned a mean value of 0.5 cigarettes per day.

Separate questions on the 6- and 18-month questionnaires asked whether the child was ever in the same room as someone who was smoking. We analyzed this measure of exposure separately.

Covariates

The following factors were considered in the analyses: gender, maternal atopy (ever having hay fever, atopic eczema, urticaria, or asthma), maternal educational level (number of years of education completed: ≤12, >12–<16, and ≥16), season of birth (classified into four seasons: spring (March–May), summer (June–August), fall (September–November), and winter (December–February)), maternal age (<25 years, 25–30 years, or ≥30 years), birth weight (<2,500 g, 2,500–4,500 g, or ≥4,500 g), breastfeeding at 6 months of age (yes or no), and parity (number of previous pregnancies of >20 weeks’ duration: 0, 1, or >1). Information regarding the child’s gender, birth weight, and month of birth were obtained from the Medical Birth Registry of Norway (26).

Statistical analyses

Our hypothesis was that in-utero exposure to maternal active smoking exerts greater effects than maternal passive smoking during pregnancy. In addition, we hypothesized that passive smoking postnatally exerts negative effects...
on the child’s respiratory health. To compare the different exposure categories directly, we first created one exposure variable with four categories: unexposed, prenatally exposed only, postnatally exposed only, and continuously exposed both in utero and during the first 3 months after birth. To be able to adjust for the respective effects in the same analyses, we used exposure to maternal prenatal smoking, exposure to maternal postnatal smoking, and exposure to paternal smoking as three separate variables. We included exposure to paternal smoking prenatally as a separate exposure and examined strata of children with only one smoking parent.

Data were analyzed using Stata 9.2 (Stata Corporation, College Station, Texas). For regression analyses, we used the binreg command with the relative risk option. This is a generalized linear model with a log-link for binary data. When we were obtaining adjusted relative risks for each outcome, the exposure variables of interest and the eight covariates were included in the analyses. We checked for interaction of maternal smoking in pregnancy with atopy and gender and for interaction of maternal smoking with paternal smoking prenatally and postnatally. The tetrachoric correlation was used for binary data.

Missing answers were not replaced by dummy variables and were not included in the analyses. For questions on smoking habits, the rates of missing data were 0.8 percent for questionnaire 1, 0.6 percent for questionnaire 3, and 4.5 percent for questionnaire 4. Rates of missing data for the health outcomes were 2.2 percent for wheeze and 6.2 percent for LRTI.

RESULTS

The cumulative incidences of the health outcomes in relation to prenatal and postnatal exposure to parental smoking and covariates are shown in table 1. Table 2 presents the cumulative incidences of and adjusted relative risks for wheeze and LRTI among children in subgroups with different patterns of tobacco smoke exposure. Children exposed to parental smoking either prenatally, postnatally, or continuously before and after birth were at increased risk of experiencing LRTI and wheeze, with prenatal exposure to maternal smoking resulting in higher relative risks than postnatal exposure.

The correlation between exposure to maternal smoking in pregnancy and maternal smoking after birth was 0.93 \((p < 0.001)\). For exposure to paternal smoking, the correlation between exposure in pregnancy and exposure in the first 3 months after birth was 0.99 \((p < 0.001)\).

Results shown in figure 1 were based on analyses in which blockwise addition of variables was done. First, crude estimates of the effect of smoking were calculated for all outcomes; then all covariates were added to the model, and finally variables for exposure to parental smoking were added. Because of the high correlation between paternal smoking prenatally and postnatally, we used exposure to paternal postpartum smoking as the variable for paternal smoking. By adding elements into the model blockwise, we obtained the respective effects of the different smoking

### TABLE 1. Cumulative incidences of lower respiratory tract infection (LRTI) at ages 0–18 months, hospitalization for LRTI at ages 0–18 months, and wheeze at ages 6–18 months according to various characteristics in the Norwegian Mother and Child Cohort Study \((n = 22,390)\), 2000–2004*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. of subjects</th>
<th>LRTI (%)</th>
<th>Hospitalization for LRTI (%)</th>
<th>Wheeze (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>22,390</td>
<td>17.1</td>
<td>4.5</td>
<td>40.0</td>
</tr>
<tr>
<td>Exposure to maternal smoking</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>2,472</td>
<td>19.7</td>
<td>6.4</td>
<td>43.6</td>
</tr>
<tr>
<td>Postnatal</td>
<td>2,043</td>
<td>19.6</td>
<td>6.2</td>
<td>44.0</td>
</tr>
<tr>
<td>Exposure to paternal smoking</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal</td>
<td>6,522</td>
<td>18.1</td>
<td>4.9</td>
<td>41.9</td>
</tr>
<tr>
<td>Postnatal</td>
<td>4,962</td>
<td>18.5</td>
<td>5.3</td>
<td>43.2</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>11,419</td>
<td>19.4</td>
<td>5.3</td>
<td>43.9</td>
</tr>
<tr>
<td>Female</td>
<td>10,912</td>
<td>14.6</td>
<td>3.6</td>
<td>35.8</td>
</tr>
<tr>
<td>Completed maternal education (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤12</td>
<td>8,864</td>
<td>17.0</td>
<td>4.7</td>
<td>39.9</td>
</tr>
<tr>
<td>&gt;12–&lt;16</td>
<td>8,591</td>
<td>16.9</td>
<td>4.3</td>
<td>41.1</td>
</tr>
<tr>
<td>≥16</td>
<td>3,813</td>
<td>18.0</td>
<td>4.3</td>
<td>38.4</td>
</tr>
<tr>
<td>Birth weight (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;2,500</td>
<td>853</td>
<td>24.1</td>
<td>11.4</td>
<td>45.6</td>
</tr>
<tr>
<td>2,500–4,500</td>
<td>20,277</td>
<td>16.8</td>
<td>4.3</td>
<td>39.6</td>
</tr>
<tr>
<td>&gt;4,500</td>
<td>1,189</td>
<td>16.9</td>
<td>3.2</td>
<td>41.6</td>
</tr>
<tr>
<td>Maternal history of atopy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6,529</td>
<td>19.3</td>
<td>5.1</td>
<td>44.7</td>
</tr>
<tr>
<td>No</td>
<td>15,861</td>
<td>16.2</td>
<td>4.2</td>
<td>38.0</td>
</tr>
<tr>
<td>Season of birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Winter</td>
<td>5,160</td>
<td>16.6</td>
<td>5.3</td>
<td>36.3</td>
</tr>
<tr>
<td>Spring</td>
<td>6,639</td>
<td>14.1</td>
<td>3.2</td>
<td>37.9</td>
</tr>
<tr>
<td>Summer</td>
<td>5,373</td>
<td>19.0</td>
<td>4.2</td>
<td>44.2</td>
</tr>
<tr>
<td>Fall</td>
<td>5,162</td>
<td>19.5</td>
<td>5.6</td>
<td>41.8</td>
</tr>
<tr>
<td>Duration of breastfeeding (months)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;6</td>
<td>4,081</td>
<td>18.4</td>
<td>5.6</td>
<td>42.5</td>
</tr>
<tr>
<td>≥6</td>
<td>17,930</td>
<td>16.8</td>
<td>4.2</td>
<td>39.5</td>
</tr>
<tr>
<td>Maternal age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤25</td>
<td>2,803</td>
<td>16.4</td>
<td>4.0</td>
<td>38.6</td>
</tr>
<tr>
<td>25–30</td>
<td>10,296</td>
<td>17.5</td>
<td>4.5</td>
<td>41.1</td>
</tr>
<tr>
<td>&gt;30</td>
<td>9,291</td>
<td>16.9</td>
<td>4.6</td>
<td>39.2</td>
</tr>
<tr>
<td>Parity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>9,837</td>
<td>14.7</td>
<td>3.2</td>
<td>37.5</td>
</tr>
<tr>
<td>1</td>
<td>8,073</td>
<td>19.5</td>
<td>5.6</td>
<td>44.0</td>
</tr>
<tr>
<td>&gt;1</td>
<td>4,480</td>
<td>18.2</td>
<td>5.2</td>
<td>38.2</td>
</tr>
</tbody>
</table>

* Maximum number of missing subjects for each health outcome: LRTI, 1,397; wheeze, 483.
exposures and also derived the influence of characteristics other than smoking behavior. Effects of maternal smoking in pregnancy remained significant, even after adjustment for all covariates and postnatal exposure to both maternal and paternal smoking. Effects of postnatal exposure to maternal smoking did not remain significant when we adjusted for other smoking exposures. Paternal smoking significantly increased the risks for all outcomes, even after adjustment for all covariates, maternal smoking during pregnancy, and maternal smoking postpartum.

Results shown in table 3 were obtained by investigating effects in children for whom only one parent was smoking. Maternal prenatal smoking also showed significant associations for this subgroup of children with nonsmoking fathers. For children with nonsmoking mothers, we found a significant postnatal effect of paternal smoking, even after adjusting for prenatal paternal smoking. Analyses including all children, with adjustment for both prenatal paternal and maternal smoking and the other covariates, showed that prenatal paternal smoking had no effects on any of the outcomes (data not shown). For postnatal paternal smoking, we found relative risks of 1.14 (95 percent confidence interval (CI): 1.04, 1.24) for wheeze, 1.06 (95 percent CI: 0.91, 1.82) for LRTI, and 1.29 (95 percent CI: 0.91, 1.82) for hospitalization for LRTI.

In this study, 11.8 percent of the children were exposed to maternal smoking at some point in pregnancy, whereas 8.2 percent of the children had mothers who smoked daily throughout pregnancy. In the first 3 months after birth, 9.7 percent of children had mothers who smoked and 23.9 percent had fathers who smoked. We calculated the average number of cigarettes smoked per day for mothers pre- and postnatally and for fathers postnatally and explored dose-response relations. Mothers who smoked during pregnancy consumed an average of 4.2 cigarettes per day. After birth, smoking mothers smoked an average of 5.0 cigarettes per day. Smoking fathers smoked an average of 9.2 cigarettes per day after birth. We did not find significant or convincing trends for dose-response relations, either for prenatal smoking or for postnatal smoking (data not shown).

To investigate risk associated with familial atopy, we stratified the data by history of maternal atopy. Results are shown in table 4. A slightly higher incidence of disease and symptoms in the subgroup of children with a positive history of maternal atopy was found, both for children exposed to parental smoking and for children nonexposed to parental smoking. However, the results indicated no major differences in the effect of tobacco smoke exposure, since an effect of smoking was seen in both groups of children. In addition, we checked for interactions between maternal smoking during pregnancy and atopy and between postnatal exposure to maternal and paternal smoking and atopy; we did not find any significant interaction.

For the separate question regarding whether a child ever stayed in the same room as someone who was smoking, no significant results were found when we adjusted for maternal smoking in pregnancy (data not shown). Children whose mothers smoked during pregnancy were more likely to regularly be in the same room as a smoker at 6 or 18 months of age than were children of women who were nonsmokers in pregnancy (23.4 percent vs. 7.4 percent).

**DISCUSSION**

Our results confirm earlier findings of adverse health effects in early life related to parental smoking, including associations between maternal smoking during pregnancy and increased risk of wheeze and LRTI (7, 15, 23, 28). In addition, we were able to separately assess the effects of maternal smoking in pregnancy, paternal smoking, and passive smoking postnatally on children’s respiratory health.

The availability of a large, population-based cohort, the use of a prospective design, and collection of exposure data at several stages before disease manifestation opened the possibility of addressing effects of environmental tobacco smoke exposure during critical time periods, such as gestation and early childhood. Detailed information on both prenatal and postnatal smoking for both parents and the large sample size made it possible to isolate sufficiently large subgroups of children with differences in exposure and to perform analyses that took different patterns of exposure into account.

There are, however, issues to consider when interpreting our results. Like most epidemiologic studies on the health effects of tobacco smoke exposure, this study assessed exposure through collection of questionnaire information, which involves possible inaccuracy in reporting. The prospective data collection presumably reduced dependence.

### TABLE 2. Cumulative incidences of and adjusted relative risks for lower respiratory tract infection (LRTI) at ages 0–18 months, hospitalization for LRTI at ages 0–18 months, and wheeze at ages 6–18 months according to exposure to maternal prenatal smoking and/or parental postnatal smoking in the Norwegian Mother and Child Cohort Study (n = 22,390), 2000–2004

<table>
<thead>
<tr>
<th>Smoking exposure</th>
<th>No. of subjects</th>
<th>LRTI</th>
<th>Hospitalization for LRTI</th>
<th>Wheeze</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prenatal</td>
<td>Postnatal</td>
<td>%</td>
<td>RR*, †</td>
</tr>
<tr>
<td>No</td>
<td>No</td>
<td>14,457</td>
<td>16.3</td>
<td>1.00</td>
</tr>
<tr>
<td>No</td>
<td>Yes</td>
<td>3,488</td>
<td>17.8</td>
<td>1.09</td>
</tr>
<tr>
<td>Yes</td>
<td>No</td>
<td>404</td>
<td>19.7</td>
<td>1.22</td>
</tr>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>1,962</td>
<td>19.8</td>
<td>1.21</td>
</tr>
</tbody>
</table>

* RR, relative risk; CI, confidence interval.
† Adjusted for gender, maternal age, birth weight, maternal atopy, parity, maternal educational level, season of birth, and breastfeeding at age 6 months.
between exposure and health effects (29). Furthermore, a
Norwegian validation study has shown that Norwegian preg-
nant women generally report their smoking habits correctly
(30). Assessment of smoking habits as a measure of exposure
to secondhand smoke after birth is probably less accurate than
assessment of in-utero exposure to maternal smoking. Assos-
ciations may be obscured and weakened because of under-
reporting or factors influencing the actual dose of secondhand
exposure (31). Despite these limitations, questionnaire-based
studies are a common and feasible way of assessing smoking
exposure in large epidemiologic studies (32).

For the age group investigated (0–18 months), wheeze
does not necessarily indicate chronic airway disease; tran-
sient wheeze is common. A diagnosis of asthma is difficult
to make in this age group, and we considered wheeze to be
a more reliable questionnaire-based outcome.

Smoking behavior is known to be associated with several
factors that could have confounded the estimated effects.
We reduced the possibility of confounding by controlling
for relevant characteristics in mothers and children (table 1),
including indicators of socioeconomic status. The preva-
lence of smoking in the MoBa Study is lower than that
among pregnant women in Norway in general, which is
approximately 15–20 percent (33). Because of the lower
exposure level in the study population, our results may un-
derestimate the national effects of smoking.

Our results show an association between maternal smok-
ing in pregnancy and adverse effects on the respiratory
health of offspring during the first 18 months of life. This
effect is independent of postnatal exposure and is not related
to atopy. A study by Martinez et al. (34) suggested that
wheeze, especially in males, was related to smaller air-
ways, and several studies have found a relation between
prenatal maternal smoking and deficits in children’s lung
function (35). Our findings agree with these studies and
support the view that maternal smoking in pregnancy may
influence the growth and development of fetal airways.

Crude analyses of postnatal exposure showed statistically
significant associations for both maternal and paternal
smoking, but after adjustment for maternal smoking during
pregnancy, the effect of maternal postnatal smoking weak-
ened substantially. Paternal postnatal smoking showed a sig-
nificant effect even after adjustment for maternal smoking.
Paternal smoking during pregnancy and paternal smoking
after birth were highly correlated, and one could argue that
the effect seen for postnatal paternal smoking could be at-
tributed to maternal passive smoking during pregnancy, not
to effects of inhaling secondhand smoke after birth. The
magnitude of the relative risks seen for paternal smoking
was quite high in comparison with relative risks associated
with maternal smoking during pregnancy. It seems biolog-
ically plausible that the fetus is more affected by active
maternal smoking than by passive maternal smoking during
pregnancy. Thus, effects seen for paternal smoking are not
likely to be generated through passive maternal smoking in
pregnancy but are more likely to be attributable to second-
hand smoking after birth.

To further elucidate the effects of paternal and maternal
smoking, we investigated the separate effects of prenatal
and postnatal exposure for children with only one smoking

parent. In the subgroup of children whose mothers were the only smoking parent, smoking during pregnancy had consistent effects, even after adjustment for postnatal maternal smoking. For children who had a smoking father and a non-smoking mother, the risks were increased for postnatal exposure but not for exposure in utero. This suggests that both smoking in pregnancy and exposure to environmental tobacco smoke postpartum are deleterious for children’s respiratory health. Some studies have found similar associations with paternal smoking (1, 36, 37), while others have not found any effects of paternal smoking (18–20, 38).

Several studies have found dose-response associations between parental smoking and respiratory disease in early childhood (14, 19, 20, 36–39), while others have not found a dose-dependent relation (7, 15). We found neither a dose-response relation for parental smoking and airway disease in childhood nor any effect of maternal postnatal smoking when controlling for prenatal exposure. This could have several explanations. A contributing factor could be that most mothers shield their children from secondhand smoke when smoking themselves. A combination of low smoking intensity among smoking mothers and increased awareness of health hazards related to secondhand smoking in Norway (40, 41) has probably resulted in lower amounts of toxic substances from secondhand smoking reaching children, making it more difficult to detect effects and to correctly determine postnatal exposure to secondhand smoke. There is also a possibility that mothers whose children exhibit

**TABLE 3.** Crude and adjusted relative risks for lower respiratory tract infection (LRTI) at ages 0–18 months, hospitalization for LRTI at ages 0–18 months, and wheeze at ages 6–18 months according to prenatal and postnatal exposure to smoking for children with only one smoking parent in the Norwegian Mother and Child Cohort Study ($n = 22,390$), 2000–2004

| Smoking exposure | LRTI | | Hospitalization for LRTI | | Wheeze | |
|------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|
|                  | Crude RR* | 95% CI* | Adjusted RR† | 95% CI | Crude RR | 95% CI | Adjusted RR† | 95% CI | Crude RR | 95% CI | Adjusted RR† | 95% CI |
| Prenatal exposure | | | | | | | | | | | | |
| Maternal smoking only ($n = 707$) | 1.25 | 1.07, 1.46 | 1.34 | 1.09, 1.66 | 1.47 | 1.07, 2.03 | 2.04 | 1.38, 3.05 | 1.10 | 1.01, 1.20 | 1.20 | 1.07, 1.35 |
| Paternal smoking only ($n = 4,075$) | 1.07 | 0.98, 1.15 | 1.00 | 0.85, 1.19 | 1.07 | 0.90, 1.27 | 0.90 | 0.61, 1.32 | 1.06 | 1.01, 1.11 | 0.93 | 0.85, 1.02 |
| Postnatal exposure | | | | | | | | | | | | |
| Maternal smoking only ($n = 532$) | 1.11 | 0.91, 1.34 | 0.86 | 0.66, 1.11 | 1.10 | 0.72, 1.66 | 0.47 | 0.26, 0.84 | 1.05 | 0.94, 1.16 | 0.92 | 0.80, 1.05 |
| Paternal smoking only ($n = 3,088$) | 1.10 | 1.01, 1.20 | 1.10 | 0.92, 1.32 | 1.16 | 0.97, 1.40 | 1.33 | 0.88, 2.00 | 1.11 | 1.06, 1.16 | 1.19 | 1.08, 1.31 |

* RR, relative risk; CI, confidence interval.
† All covariates (gender, maternal age, birth weight, maternal atopy, parity, maternal educational level, season of birth, and breastfeeding at age 6 months) and both prenatal and postnatal exposure to smoking were included in the analyses.

**TABLE 4.** Cumulative incidences of and adjusted relative risks for lower respiratory tract infection (LRTI) at ages 0–18 months, hospitalization for LRTI at ages 0–18 months, and wheeze at ages 6–18 months according to exposure to maternal prenatal smoking and/or parental postnatal smoking, by history of maternal atopy, in the Norwegian Mother and Child Cohort Study ($n = 22,390$), 2000–2004

<table>
<thead>
<tr>
<th>Maternal atopy and smoking exposure</th>
<th>No. of subjects</th>
<th>LRTI</th>
<th>Hospitalization for LRTI</th>
<th>Wheeze</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>RR†,‡</td>
<td>95% CI†</td>
</tr>
<tr>
<td>Maternal atopy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exposure</td>
<td>4,282</td>
<td>18.7</td>
<td>4.4</td>
<td>43.1</td>
</tr>
<tr>
<td>Prenatal exposure</td>
<td>685</td>
<td>22.4</td>
<td>1.15</td>
<td>0.95, 1.39</td>
</tr>
<tr>
<td>Postnatal exposure</td>
<td>1,643</td>
<td>19.9</td>
<td>0.99</td>
<td>0.85, 1.14</td>
</tr>
<tr>
<td>No maternal atopy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No exposure</td>
<td>10,175</td>
<td>15.3</td>
<td>3.8</td>
<td>36.9</td>
</tr>
<tr>
<td>Prenatal exposure</td>
<td>1,787</td>
<td>18.7</td>
<td>1.13</td>
<td>0.99, 1.29</td>
</tr>
<tr>
<td>Postnatal exposure</td>
<td>4,124</td>
<td>17.9</td>
<td>1.13</td>
<td>1.03, 1.25</td>
</tr>
</tbody>
</table>

* The reference group consisted of the unexposed in each category, and both exposure groups were included in the analyses.
‡ RR, relative risk; CI, confidence interval.
§ Adjusted for gender, maternal age, birth weight, maternal atopy, parity, maternal educational level, season of birth, and breastfeeding at age 6 months.
early signs of morbidity might stop smoking because of this. Questionnaire 4 addressed smoking 0–3 months postpartum, and women who stopped smoking early because of their child’s illness might have answered “no” to the question on smoking. All of these possible influences could have contributed to underestimation of associations.

Some studies have found the effect of smoking to differ by familial history of atopy (28,42), while others have not (18,39). We did not find convincing differences in the effects of smoking related to maternal history of atopy, either by stratification or by interaction analyses.

Despite an increased focus in Norwegian public health policy on smoking cessation in pregnancy, a relatively high number of women in Norway (10–20 percent) still smoke during pregnancy (33). The current Norwegian exposure level is probably representative of current or future exposure levels in most Western societies with increasing awareness of the adverse health effects of environmental tobacco smoke. In the United States, there was a 65 percent decline in cotinine levels among children aged 3–11 years between 1988–1991 and 2001–2002 (43). The major contributing factor is suggested to be changes in the smoking practices of adults in homes where children reside (44).

Even with the relatively low exposure levels observed in this Norwegian study, we found adverse effects of maternal smoking in pregnancy. The associations found for postnatal paternal smoking suggested an effect of passive smoking during infancy and also suggested that paternal smoking is an independent risk factor for respiratory disease. The lack of effect of maternal postnatal smoking could have been due to the low exposure levels and high awareness among smoking mothers not to expose their children to passive smoking. Future public health efforts should focus on encouraging smoking cessation before and during pregnancy, as exposure in pregnancy is shown to influence childhood respiratory health. Our results also suggest an independent effect of passive smoking after birth; thus, recommendations should include shielding children from exposure to secondhand smoke.

ACKNOWLEDGMENTS

This study was supported by grants from the Norwegian Association of Heart and Lung Patients, with the aid of EXTRA funds from the Norwegian Foundation for Health and Rehabilitation.

Conflict of interest: none declared.

REFERENCES

20. Wright AL, Holberg C, Martinez FD, et al. Relationship of parental smoking to wheezing and nonwheezing lower