the timing of the data collection, our information could not
be biased by the women's knowledge about the
outcome of pregnancy. Potential misclassification is
likely to be non-differential, and our results may thus
underestimate the true association between coffee
drinking and stillbirth. Due to a higher intake of coffe
and a faster metabolism among smokers\textsuperscript{11} we
hypothesised that the fetotoxic effect of caffeine could
depend on smoking habits during pregnancy. However,
the risk of stillbirth associated with coffee was
similar in smokers and non-smokers.

There did not seem to be one single cause that
could explain the increased risk of stillbirth among
women with a high intake of coffee (see bmj.com).

Information on coffee intake during pregnancy
was missing in a quarter of the population. Women
with missing information had a different risk profile
than women with valid information. However, we have
no reason to believe that the association between coffee
and stillbirth among women with non-valid information
would be different from the one we found.

We thank Morten Frydengberg, associate professor, for statistical
advice.
Distribution of asthma and lung function in participants aged 42 according to severity of asthma at age 7 or 10

<table>
<thead>
<tr>
<th>Symptoms age 7</th>
<th>No (%) at age 42</th>
<th>Lung function at age 42</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No recent asthma (n=199)</td>
<td>Intermittent asthma (n=54)</td>
</tr>
<tr>
<td>Mild wheezy bronchitis</td>
<td>40 (66)</td>
<td>12 (20)</td>
</tr>
<tr>
<td>Wheezy bronchitis</td>
<td>50 (77)</td>
<td>13 (15)</td>
</tr>
<tr>
<td>Asthma</td>
<td>28 (28)</td>
<td>19 (19)</td>
</tr>
<tr>
<td>Severe asthma</td>
<td>8 (11)</td>
<td>9 (13)</td>
</tr>
<tr>
<td>Control</td>
<td>73 (85)</td>
<td>5 (6)</td>
</tr>
</tbody>
</table>

FEV1 = forced expiratory volume in 1 second; FVC = forced vital capacity.

*P<0.001 compared with controls.

Fifteen of the original cohort had died at follow up, one from asthma. Of the remaining 464, 403 participated in the current review, giving a continuing participation rate of 87%. In all, 267 participants attended the laboratory for measurement of lung function. We calculated mean values of lung function using standard two sample t tests and confidence intervals of the mean by standard methods.

The table shows the clinical expression of asthma at age 42 according to severity of disease at recruitment. The distribution of severity at age 42 has not changed with time. The proportion of cases with no recent asthma has increased steadily from 20% at age 14 years to 40% (126/317 = 39.9%) at age 42.

Lung function was similar to that of controls in children who had had wheezy bronchitis in childhood (table). Participants who had had asthma at age 7 had reduced lung function at age 42.

**Comment**

Our study shows that the pattern of asthma during childhood predicts outcome. Most children with persistent asthma had continuing symptoms into adult life and reduced lung function. However, children who had intermittent symptoms associated with respiratory tract infections generally had completed resolution of symptoms in adult life. The small number of participants who still had mild, intermittent symptoms at age 42 had normal lung function. This good outcome was achieved despite the fact that anti-inflammatory treatments were not available for most of their childhood.

Contributors: CFR, AO, and JW initiated the project and, together with EH, AL, MR and LW, developed the protocol. EH, AL, MR, and LW were responsible for recruitment, data collection, and data analysis. JBC was the statistician. The manuscript was jointly written and reviewed by all of the authors. CFR is the guarantor.

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**Spontaneous loss of early pregnancy and risk of ischaemic heart disease in later life: retrospective cohort study**

Gordon C S Smith, Jill P Pell, David Walsh

We recently showed that complications in late pregnancy are associated with an increased risk of maternal ischaemic heart disease (IHD) in later life.1 We hypothesised that this may reflect common determinants, such as thrombophilic genetic defects and anticardiolipin antibodies. Spontaneous losses of pregnancy are also associated with inherited and acquired thrombophilias in the mother.2 We examined whether spontaneous losses of early pregnancy are associated with maternal risk of IHD.

**Participants, methods, and results**

We used routine national maternity data (SMR2) to identify all 129 290 eligible women who delivered their first liveborn infant in Scotland during 1981-5. The exclusion and inclusion criteria, definitions, and demographic characteristics were as previously described.3 We used national death (GRO) and discharge (SMR1) data to determine the risk of death or hospital admission due to IHD during 1981-99. The cumulative probabilities of survival free from IHD events were assessed with Cox’s proportional hazards models with age as the time scale (Stata version 7.0, StataCorp, College Station, TX, USA).

A history of any spontaneous loss of early pregnancy before the first live birth was associated with an increased risk of IHD (table). The association was independent of maternal age at the time of first birth, height, socioeconomic deprivation, essential hypertension, and complications during the first pregnancy. The magnitude of the risk increased with the number of previous losses. By contrast, there was no association between therapeutic abortion and subsequent risk of IHD (adjusted hazard ratio 0.93, 95% confidence interval 0.59 to 1.46). Only 0.1% (162) of women had had a hernia repair, and there was no significant association...