Smoking in pregnancy: associations with skinfold thickness, maternal weight gain, and fetal size at birth

S W D’SOUZA, PATRICIA BLACK, B RICHARDS

Abstract

Skinfold thickness is an index of subcutaneous fat, and certain maternal conditions during pregnancy affect the skinfold thicknesses of the baby. A study was performed to investigate the effect of smoking on skinfold thickness, maternal weight gain, and fetal size at birth. A total of 452 mothers with normal singleton pregnancies were grouped as: non-smokers, light-to-moderate smokers, or heavy smokers. Maternal age, height, parity, and duration of pregnancy were similar in the three groups. Heavy smokers gained significantly less weight than non-smokers, but there was no significant difference in skinfold thickness. Babies born to smokers had lower birth weights and smaller head circumferences and were shorter than those born to non-smokers, but skinfold thicknesses were similar.

The presence of a normal layer of subcutaneous fat in babies whose mothers smoked suggests that fetal growth retardation is not caused by nutritional deficiencies.

Introduction

Several studies have shown that when mothers smoke during pregnancy the baby’s birth weight is reduced, but there is uncertainty as to whether this is caused by nutritional deficiencies in the mothers or by the direct effect of the constituents of cigarette smoke. Since smoking also reduces maternal weight gain during pregnancy it has reasonably been suggested that the adverse effects of smoking are due to the mother’s nourishment.3 4 Reducing the number of cigarettes smoked daily during pregnancy apparently has some benefit; when smoking habits are altered by the end of the fourth month of pregnancy the risks of reduced birth weight are appropriate to the changed habit.

When studying the effects of several maternal conditions in pregnancy on subcutaneous fat in the newborn baby, Whitelaw5 used skinfold thickness for assessing subcutaneous fat. Skinfold thickness was measured at the triceps, biceps, subscapular, and suprailiac sites on both sides. Maternal hypertension was associated with a reduction in skinfold thickness in the baby. Maternal obesity and maternal diabetes increased the baby’s skinfold thickness, but the baby’s skinfold thickness did not appear to be affected by maternal smoking. Unfortunately, Whitelaw’s5 sample of smokers and non-smokers was not matched for parity, sex of baby, etc.

We have undertaken a study in greater detail to investigate the effect of smoking in pregnancy on skinfold thickness, maternal weight gain, and fetal size at birth.

Methods

Mothers—A total of 452 mothers who attended antenatal clinics regularly at St Mary’s Hospital, Manchester, were selected since they had a normal singleton pregnancy. These mothers were aged 19 to 35 years and had no history of stillbirths. Gestational age, as determined from the first day of the last menstrual period, was consistent with that by uterine size and by fetal biparietal diameter measured by ultrasound at the first or second visit to the antenatal clinic. Details of maternal age, height, weight, obstetric history, and social class were obtained from the case notes. The first visit to the antenatal clinic took place at eight to 10 weeks’ gestation. At each visit the mothers were asked about the number of cigarettes smoked per day, and they were grouped as follows: (a) non-smokers, (b) light-to-moderate smokers, (1-14 cigarettes/day), and (c) heavy smokers (15 or more cigarettes/day).4 Maternal weight gain in pregnancy was expressed as the mean weekly gain between the first visit to the antenatal clinic and a later visit at 36-38 weeks’ gestation. Skinfold thickness was measured using a Harpenden caliper by one person (PB) on the labour ward, with the mother standing. The triceps skinfold was measured in the left arm, halfway between the acromion and the olecranon, with the arm held

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by the side of the body and the elbow extended. The subcapsular skinfold was measured immediately below the angle of the left scapula with the fold either in a vertical line or slightly inclined, in the natural cleavage of the skin, with the arm by the side of the body.7

Babies—The following measurements were carried out within 24 hours of birth by the same person (PB). Weight was measured by a beam balance. The crown-heel length was measured using a neonate meter.8 A fibreglass measuring tape was used for measuring the occipitofrontal head circumference. Skinfold thickness was measured in the mid-triceps and subcapsular area on the left side using the Harpenden caliper.7 The caliper was held with the right hand and a skinfold was held with the left hand throughout the procedure. Pressure was maintained on the skinfold with the caliper for about 60 seconds, during which time the reading was stable.

Statistics—Student’s t test was used when comparing mean values.

Results

Maternal age, height, parity, and duration of pregnancy were similar in the three groups of mothers: non-smokers, light-to-moderate smokers, and heavy smokers (table I). There were significant differences in weight gain during pregnancy, with non-smokers gaining 60 g a week more than heavy smokers (p < 0.001). The mean values of maternal triceps and subcapsular skinfold thicknesses were lower in smokers than in non-smokers, but these differences were not statistically significant (triceps skinfold: heavy smokers v non-smokers: t = 1.85, p > 0.05; subcapsular skinfold: heavy smokers v non-smokers: t = 1.43, p > 0.1). Weight, length, head circumference, and skinfold thickness in newborn babies are shown in table II. In both sexes babies born to non-smokers were heavier, longer, and had larger head circumferences than those born to heavy smokers. The differences were statistically significant in all cases except for head circumference in boys. Babies born to light-to-moderate smokers occupied an intermediate position for weight, length, and head circumference in girls and for weight and length in boys. In both sexes skinfold thickness was unaffected by smoking during pregnancy.

The expected association between smoking habit and social class was not observed, but within each social class group there was a consistent pattern of lower birth weights in heavy smokers, and the skinfold thickness was not affected.

Discussion

Smoking during pregnancy appears to have caused a general retardation in intrauterine growth, resulting in babies born with lower birth weights, shorter lengths, and smaller head circumferences. Skinfold thickness, however, which is an index of subcutaneous fat, was not affected in these babies. Indeed, the presence of a normal layer of subcutaneous fat raises doubt about the possible role of nutritional deficiencies in causing fetal growth retardation when mothers smoke during pregnancy.

An analogy has been drawn between the 8% reduction in birth weight in babies born to heavy smokers compared with those born to non-smokers and the reduction in birth weights due to maternal starvation, reported in babies born during the Dutch famine of 1944-5.9 A similar reduction in birth weight also seems to occur in babies born to poorly nourished mothers in south India, where malnutrition and intrauterine growth retardation exist against a background of poverty, lack of education, and limited resources.10 Our mothers who smoked did not appear undernourished in the same way. Moreover, in babies born to poorly nourished mothers in south India, unlike those born to heavy smokers in the present study, reduction in birth weight was associated with a significant reduction by skinfold thickness. When viewed in this way the nutritional hypothesis is less convincing.

The skinfold thickness measurements of all babies in our study were between the 10th and the 90th centiles of standards of skinfold thickness for British newborn infants, by gestational age and by birthweight.11 In the human fetus the increase in body fat takes place from about 28 weeks' gestation to term, when fat is measured by chemical analysis.12 Whitelaw has shown that during the same gestational period there is an increase in skinfold thickness. Since most of body fat is subcutaneous, variations in skinfold thickness are clinically important in that they provide information about fetal and neonatal nutrition.

It is recognised that not all growth-retarded newborn babies are suffering from undernutrition. Smoke is not thin but are constitutionally small "miniatures."13 The finding of reduced skinfold thickness is useful in distinguishing the truly undernourished baby from one that is constitutionally small. Babies born to mothers who smoke during pregnancy resemble the constitutionally small group of growth-retarded babies.

It seems unlikely that smoking restricts fetal growth mainly by undernutrition. The growth-retarding effect might well be due to fetal hypoxia caused by some constituent of tobacco smoke, probably carbon monoxide. While reliable information about food intake in mothers who smoke during pregnancy is needed, until such information is available we can only be certain about the adverse effects on the fetus when mothers give up smoking.

We thank the obstetricians at St Mary's Hospital, Manchester for allowing us to study these mothers, the North-western Regional Health Authority for financial help, and Mrs J Cadman for the computer programming.

References


### Table I

<table>
<thead>
<tr>
<th>Maternal variable</th>
<th>Non-smokers (n = 281)</th>
<th>Light-to-moderate smokers (n = 86)</th>
<th>Heavy smokers (n = 85)</th>
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</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>26.07 ± 0.28</td>
<td>25.08 ± 0.51</td>
<td>25.10 ± 0.49</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>161.94 ± 0.37</td>
<td>161.23 ± 0.63</td>
<td>161.25 ± 0.75</td>
</tr>
<tr>
<td>Parity</td>
<td>1.04 ± 0.07</td>
<td>1.03 ± 0.10</td>
<td>1.06 ± 0.13</td>
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<tr>
<td>Gestation (wk)</td>
<td>40.97 ± 0.08</td>
<td>40.58 ± 0.19</td>
<td>40.73 ± 0.14</td>
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<tr>
<td>Triceps skinfold (mm)</td>
<td>15.23 ± 0.44</td>
<td>13.92 ± 0.78</td>
<td>13.83 ± 0.67</td>
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<tr>
<td>Subscapular skinfold (mm)</td>
<td>15.75 ± 0.39</td>
<td>15.96 ± 0.59</td>
<td>14.16 ± 0.73</td>
</tr>
<tr>
<td>Weight gain (g/wk)</td>
<td>425.74 ± 9.4</td>
<td>423.62 ± 16.8</td>
<td>362.44 ± 16.3***</td>
</tr>
</tbody>
</table>

*p < 0.001

### Table II

<table>
<thead>
<tr>
<th>Infant measurements</th>
<th>Maternal smoking habit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-smokers</td>
</tr>
<tr>
<td>Number</td>
<td>147</td>
</tr>
<tr>
<td>Birthweight (g)</td>
<td>3552.08 ± 33.97</td>
</tr>
<tr>
<td>Length (cm)</td>
<td>49.91 ± 0.26</td>
</tr>
<tr>
<td>Head circumference (cm)</td>
<td>35.24 ± 0.09</td>
</tr>
<tr>
<td>Triceps skinfold (mm)</td>
<td>4.52 ± 0.08</td>
</tr>
<tr>
<td>Subscapular skinfold (mm)</td>
<td>4.31 ± 0.08</td>
</tr>
</tbody>
</table>

**p < 0.001

Non-smokers v smokers ***p < 0.001

Non-smokers v smokers **p < 0.01

Non-smokers v smokers *p < 0.05

Non-smokers v smokers *p < 0.05; **p < 0.01; ***p < 0.001
**SHORT REPORTS**

**Arteritis of the arms**

Arteritis affecting the arm vessels occurs in giant-cell arteritis, Takayasu’s arteritis, syphilis, and Buerger’s disease. The lesions are seldom confined to the arms, however, and each condition has a characteristic clinical presentation. We report on a patient with symmetrical arteritis of the arms associated with circulating immune complexes.

**Case report**

A 68-year-old man was admitted in February 1979 with a two-month history of severe left-sided headaches associated with two episodes of blurred vision in the left eye. He did not smoke. Physical examination was unremarkable. Results of investigations were white cell count 10.2 x 10^9/l with a normal film, and erythrocyte sedimentation rate 116 mm in first hour. A biochemical screen was normal. Biopsy of the left temporal artery showed only intimal fibrosis.

Treatment with prednisolone 80 mg daily led to improvement in the headache, but shortly afterwards he developed painful dysesthesia in the hands. This increased in severity, and three months later he was readmitted with severe pain in all fingers in the left hand and in the fourth and fifth fingers in the right hand. The fingers were cold and bluish, and all pulses in the arms below the axillary arteries were reduced in volume though still palpable. Intra-arterial thymoxamine, intravenous dextran 40, and oral phenoxbenzamine were given in addition to the prednisolone. The radial and ulnar pulses later became impalpable, though the symptoms in his arms remained unchanged. Arteriography showed severe stenosis of both brachial arteries, suggesting an arteritic lesion (figure). Investigations showed C3 12 mg/100 ml serum (normal range 60-135), C4 normal; high concentrations of immune complexes as judged by Clq binding; and raised concentrations of C-reactive protein. Serological tests for syphilis were negative.

Maintenance treatment with oral azathioprine and prednisolone was given. Initially his symptoms of ischaemia improved, but three weeks later the circulation in his hands deteriorated acutely and plasma exchange was started. Biopsy specimens of the saphenous vein, right posterior tibial artery (obtained at insertion of Scribner shunts for plasma exchange), and right gastrocnemius muscle showed no evidence of vasculitis. After five plasma exchanges improvement was only marginal and cyclophosphamide was substituted for azathioprine. The circulation to his arms improved steadily, so that one year later he had only mild ischaemic pain in the forearms on exercise and the skin colour and temperature were normal. The erythrocyte sedimentation rate fell to 27 mm in first hour, the complement profile returned to normal, and no immune complexes could be detected in the blood.

**Comment**

The initial clinical diagnosis in this man was giant-cell arteritis. Although giant-cell arteritis of the subclavian vessels has been reported, the detection of circulating immune complexes and depressed complement concentrations made this diagnosis unlikely. Buerger’s disease may present in the arms but is almost confined to young men who smoke and is usually diagnosed only in patients in whom the lesion affects vessels distal to the brachial arteries. Takayasu’s arteritis classically affects young women aged 10-30, the sex ratio being 7:1, and may be excluded in our patient because of the high concentration of immune complexes and low serum C3 concentration.

Our patient had arteritis localised to the brachial arteries associated with circulating immune complexes. He had no other evidence of immune complex deposition such as glomerulonephritis or arthritis. While immunologically mediated arteritis is rarely localised to a single vascular territory, the circulation to the limbs improved at the same time as the immune complexes disappeared. Whether this improvement was due to the spontaneous development of a collateral circulation or to reversal of the arteritis process is uncertain, as is whether the progression of the arterial disease was arrested by cyclophosphamide. Interestingly, however, it was not until cyclophosphamide was given that the circulation to the arms improved, and this improvement was associated with the disappearance of the circulating immune complexes.

The figure is reproduced by courtesy of Dr C Woodham.


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