CLINICAL RESEARCH

Body weight and risk of myocardial infarction and death in the adult population of eastern Finland

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Abstract

Body mass index (weight (kg) divided by height squared (m^2)) and its association with the risk of myocardial infarction and death from all causes were studied prospectively in a randomly selected population sample in eastern Finland aged 30-59 at outset in 1972. The study population consisted of 3786 men and 4120 women. The participation rate in the survey in 1972 was over 90%. All deaths and admissions to hospital in the sample were obtained from the National Death Certificate and Hospital Discharge Registers. During the seven years of follow up until 1978, 170 men and 52 women had acute myocardial infarction, and during the nine years up to 1980, 223 men and 92 women died. Independent of age, men with a body mass index of 28·5 or more had a significantly higher incidence of acute myocardial infarction. This effect was also independent of smoking but not independent of biological coronary risk factors—that is, serum cholesterol concentration and blood pressure. In the analysis stratified for smoking in men the body mass index total mortality curve was J shaped among non-smokers, whereas smoking entirely outweighed body mass index as a predictor of death. Body mass index did not contribute significantly to the risk of either acute myocardial infarction or death in women.

It is concluded that a body mass index of around 29·0-31·0 or more is not only a marker for coronary risk factors but is also a predictor of acute myocardial infarction in men.

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Introduction

Several studies have examined whether obesity is associated with increased mortality or more frequent coronary heart disease. Though the risk of death for people who are very obese has turned out to be unequivocally higher than that for non-obese people,[1-3] results have been inconsistent.[4-14] The role of obesity seems to vary in importance among different populations, at different ages, and between men and women. The detection of smoking as a powerful confounder of the relation between relative body weight and coronary heart disease and death has clarified part of this issue.[15-18]

In the Framingham study, particularly in men under 50 and in women, obesity was found to be a predictor of the incidence of and mortality from coronary heart disease independent of age, serum lipoprotein values, blood pressure, smoking, and other risk factors,[11] and in a recent report obesity was also related to mortality in non-smokers aged 65 and over.[19] These findings have not been replicated in other parts of the world. After 15 years of follow up in the seven countries study Keys *et al* could not find an excess risk for death from all causes or coronary heart disease with increasing weight in any of the regions surveyed.[14] A study in Britain found a strong modifying effect of age on the association between weight and mortality (positive in 40-49 year olds, inverse in 60-64 year olds) and could not confirm overweight as an independent risk factor.[9] In Finnish people aged 85 and over survival was positively associated with relative weight.[20]

Not surprisingly the importance of obesity in chronic conditions such as coronary heart disease has been a subject of controversy in recent reviews[21,22] and the concept of ideal body weight has been questioned.[23] Given the enigmatic role of relative weight in coronary heart disease and mortality, we decided to study this association in a population with high mortality and morbidity from cardiovascular disease in eastern Finland.[24] Prospective investigations of the study population had confirmed that smoking, serum cholesterol concentration, and blood pressure were associated with the risk of coronary heart disease.[25,26] This paper analyses the association between body mass index and the risk of myocardial infarction and death from all causes in a representative sample of middle aged men and women studied prospectively from 1972.

Subjects and methods

The study group was based on a random sample of the population of eastern Finland. The sampling frame comprised people living in two provinces, North Karelia and Kuopio, aged 25-59 in 1972 and born on the 18th or 28th of the month. The analysis reported here is restricted to men and women aged 30-59 in 1972.

The survey included a self administered questionnaire together with additional interviews and physical measurements. Details of laboratory methods, clinical examinations, and mean values for coronary risk factors have been reported.[24] Over 90% of subjects approached agreed to participate (91% of men, 94% of women). Subjects with a history of myocardial infarction, angina pectoris, and cerebrovascular stroke in the 12 months before the baseline survey were excluded from analysis. Complete data from 3786 men and 4120 women were used in the analysis.

Body weight was measured to the nearest kg in light clothing during the clinical examination and body mass index (weight (kg) divided by height squared (m^2)) calculated as an indicator of relative body weight. The data were stratified into approximate quintiles of body mass index. The same body mass index categories were used for men and women, though the variation in body mass index was greater among women. In both sexes the distribution of body mass index was slightly skewed to the left.

Information on smoking was obtained by a standard questionnaire. A previous study analysing serum thiocyanate concentrations in relation to reported smoking habits in this population had shown self reported data on smoking to be reliable.[24] Those subjects who reported that they had smoked regularly for at least a year and more than once a day on average during the month before the outset of the survey in 1972 were classified as current smokers.

Data on admissions to hospital for acute myocardial infarction (ICD (8th revision) codes 410-411) up to the end of 1978 were derived from the National Hospital Discharge Register. Data on deaths up to the end of 1980 were obtained from the National Death Certificate Register. During the seven year follow up period (1972-8) 170 men and 52 women had acute myocardial infarction, and during the whole nine years of follow up (1972-80) 223 men and 92 women died.

Kaplan-Meier estimates[27] of cumulative nine year mortality among men and women aged 30-59 in relation to approximate body mass index quintiles were calculated for each calendar year and used to assess the univariate association of body mass index with the risk of death in smokers and non-smokers separately. The Cox proportional hazards model[28] was applied to the data to assess the relation between outcome of disease (incidence of acute myocardial infarction or death) and body mass index when two other determinants, age and smoking, were kept constant. The differences in disease outcome between subjects in the highest body mass index quintile and the rest of the population and between subjects in the lowest body mass index category and the rest of the population were tested for statistical significance by χ^2 test with the Mantel-Haenszel procedure.[29]

Results

Table I shows the seven year incidence of acute myocardial infarction and the nine year crude mortality from all causes by approximate quintiles of body mass index in men stratified for age and smoking. In both age groups men with a body mass index of 28·5 or more had an increased risk of acute myocardial infarction ($p<0·05$). The relation between body mass index and mortality from all causes was inverse in 30-49 year old men and U shaped in the 50-59 year olds with no significant linear trends. Closer inspection of the raw data for men showed that the excess incidence of acute myocardial infarction occurred mainly at a body mass index of 29·0-31·0 and that the excess total mortality of the lean men occurred mainly in those with a body mass index of 20·0-22·0.

The overall risks of death and acute myocardial infarction were roughly threefold for smokers compared with non-smokers. This difference in risk of death decreased with increasing body mass index, mainly due to an increase in mortality among non-smokers with increasing weight (see table III). Among both smokers and non-smokers mortality from all causes and the incidence of acute myocardial infarction were highest among the heaviest men (body mass index ≥28·5). The risk of death and acute myocardial infarction among smokers was lowest in the body mass index category 25·0-26·4, resulting in a U shaped association between body mass index and risk of death and acute myocardial infarction.

Excluding the first two years of follow up in order to control for part of the effect of unrecognised pre-existing disease at baseline did not change the results appreciably. Because age modified the relation between body mass index and mortality it was also taken into account in this analysis. The difference in mean age between smokers and non-smokers varied from only 0·2 to 3·3 years among the body mass index quintiles. The mean age of men who died during follow up was 48·6 (SD 7·3) years among smokers and 49·0 (6·8) years among non-smokers.

In women aged 50-59 there was a tendency towards a higher risk of acute myocardial infarction in the heaviest group and a higher risk of death in the leanest group, but trends were not significant (table II). Among women aged 30-49 mortality from all causes was lower in the two lowest body mass index quintiles—that is, below 25·0. Even after nine years of follow up there were too few deaths to allow a precise estimation of risk according to body mass index. Neither the risk of death nor the risk of acute myocardial infarction was consistently higher among women smokers than among non-smokers, though the risk ratios averaged 1·5 to 2·0.

The figure shows the cumulative nine year survival curves for men in relation to body mass index quintiles and smoking state. There were clearly contrasting survival patterns among the smokers and non-smokers. Smokers showed an overall steep decrease in survival with little or no difference among body mass index quintiles. Among non-smokers men with the lowest survival were in the lowest and highest body mass index quintiles. Whereas the decreasing survival of the leanest men (body mass index ≤22·9) levelled off after the sixth year of follow up, the slope in the heaviest group of men

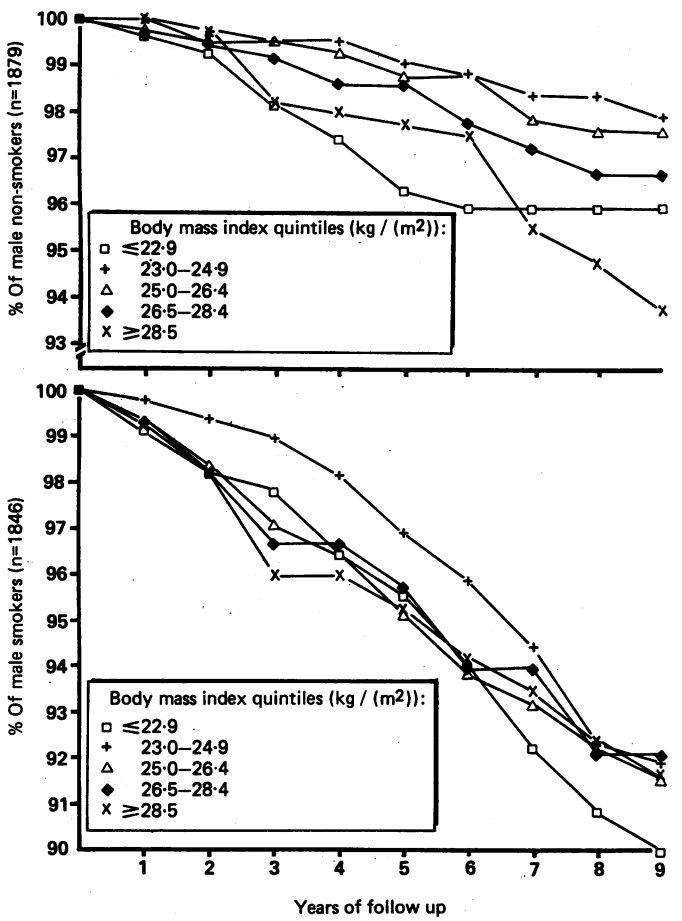
TABLE I—*Seven year incidence of acute myocardial infarction and nine year mortality from all causes per 1000 men by body mass index quintile, age, and smoking state at baseline*

	Age 30-49							Age 50-59						
	Acute myocardial infarction			Deaths				Acute myocardial infarction			Deaths			
Body mass index quintile (kg/(m^2))	No of cases	Rate/ 1000	90% Confidence interval	No	Rate/ 1000	90% Confidence interval	Subjects at risk	No of cases	Rate/ 1000	90% Confidence interval	No	Rate/ 1000	90% Confidence interval	Subjects at risk
Smokers:														
≤22·9	15	45	28 to 66	22	66	45 to 91	333	8	68	34 to 113	21	179	121 to 249	117
23·0-24·9	16	43	27 to 62	22	59	40 to 81	374	12	109	63 to 167	16	145	92 to 211	110
25·0-26·4	8	32	16 to 53	13	52	31 to 78	252	8	104	52 to 173	13	169	101 to 255	77
26·5-28·4	11	44	25 to 69	14	56	34 to 83	248	8	131	66 to 218	11	180	102 to 280	61
≥28·5	12	58	34 to 89	7	34	16 to 58	208	13	197	118 to 297	15	227	141 to 333	66
Total	62	44	35 to 54	78	55	45 to 66	1415	49	114	89 to 142	76	176	144 to 211	431
Non-smokers:														
≤22·9	0	0	0 to 40	2	13	2 to 32	159	0	0	0 to 34	1	50	2 to 166	20
23·0-24·9	3	33	9 to 72	2	22	4 to 55	91	1	59	2 to 195	0	0	0 to 40	17
25·0-26·4	2	38	7 to 95	1	19	1 to 63	52	1	50	2 to 166	1	50	2 to 166	20
26·5-28·4	0	0	0 to 11	0	0	0 to 11	59	0	0	0 to 32	1	48	2 to 158	21
≥28·5	1	22	1 to 72	3	65	18 to 141	46	0	0	0 to 29	2	87	15 to 217	23
Total	6	15	7 to 27	8	20	10 to 33	407	2	20	4 to 50	5	50	20 to 93	101
All:														
≤22·9	17	31	20 to 44	25	45	31 to 61	555	12	67	39 to 102	29	161	116 to 214	180
23·0-24·9	19	26	17 to 37	26	36	25 to 48	727	18	90	59 to 128	20	101	67 to 141	199
25·0-26·4	14	26	16 to 39	20	37	25 to 52	540	15	95	59 to 139	18	114	74 to 162	158
26·5-28·4	16	28	18 to 41	21	37	25 to 51	570	11	66	37 to 103	16	96	61 to 139	167
≥28·5	23	46	32 to 63	16	32	20 to 47	497	25	130	91 to 176	32	166	121 to 218	193
Total	89	31	26 to 37	108	37	31 to 43	2889	81	90	74 to 107	115	128	109 to 148	897

TABLE II—*Seven year incidence of acute myocardial infarction and nine year mortality from all causes per 1000 women by body mass index quintile, age, and smoking state at baseline*

Body mass index quintile (kg/(m²))	Age 30-49							Age 50-59						
	Acute myocardial infarction			Deaths				Acute myocardial infarction			Deaths			
	No of cases	Rate/ 1000	90% Confidence interval	No	Rate/ 1000	90% Confidence interval	Subjects at risk	No of cases	Rate/ 1000	90% Confidence interval	No	Rate/ 1000	90% Confidence interval	Subjects at risk
Smokers:														
≤22·9	2	9	1 to 23	3	14	4 to 30	212	4	68	24 to 135	8	136	69 to 226	59
23·0-24·9	3	9	3 to 19	4	12	4 to 24	346	6	71	31 to 127	4	48	17 to 95	84
25·0-26·4	6	21	9 to 37	7	25	12 to 43	284	6	78	35 to 139	5	65	26 to 121	77
26·5-28·4	5	16	6 to 30	7	22	10 to 38	316	3	30	8 to 65	3	30	8 to 65	101
≥28·5	10	36	20 to 57	7	25	12 to 43	277	12	98	57 to 150	16	130	82 to 189	123
Total	26	18	13 to 24	28	20	14 to 27	1435	31	70	51 to 92	36	81	60 to 105	444
Non-smokers:														
≤22·9	5	7	3 to 13	6	9	4 to 16	704	2	23	4 to 57	7	80	38 to 137	88
23·0-24·9	4	7	2 to 14	5	9	4 to 17	569	1	8	0 to 26	7	54	26 to 93	129
25·0-26·4	2	4	1 to 10	11	21	12 to 33	515	5	28	11 to 53	6	34	15 to 61	176
26·5-28·4	2	4	1 to 10	9	20	11 to 33	445	8	32	16 to 53	10	40	22 to 64	247
≥28·5	1	3	0 to 10	4	11	4 to 22	354	13	37	22 to 56	12	34	20 to 52	351
Total	14	5	3 to 8	35	14	10 to 18	2587	29	29	21 to 39	42	42	32 to 53	991
All:														
≤22·9	5	6	2 to 11	8	9	4 to 15	870	3	26	7 to 57	9	79	42 to 128	114
23·0-24·9	7	11	5 to 19	7	11	5 to 19	663	2	14	3 to 34	7	47	22 to 81	148
25·0-26·4	4	7	2 to 14	12	21	12 to 32	572	6	30	13 to 54	7	36	17 to 62	197
26·5-28·4	2	4	1 to 10	9	18	10 to 29	507	8	30	15 to 50	11	41	23 to 64	271
≥28·5	2	5	1 to 12	8	20	10 to 33	404	13	35	21 to 53	14	37	22 to 55	374
Total	20	7	5 to 10	44	15	12 to 19	3016	32	29	21 to 38	48	43	33 to 54	1104

(body mass index ≥28·5) was fairly linear. The best survival curves were observed in the second and the third body mass index quintiles. This survival pattern among male non-smokers suggests a J shaped association between body mass index and the risk of death. Given that the pattern of survival for the entire cohort was determined largely by the older subjects, and given the findings in table I, the result was as expected.



Nine year survival curves (Kaplan-Meier) for 30-59 year old men in relation to approximate body mass index quintiles and smoking state at baseline.

In women there was little difference in survival among the body mass index categories and between smokers and non-smokers (data not shown).

Finally, we performed a multivariate regression analysis using the Cox proportional hazards model on the incidence of acute myocardial infarction and the risk of death from all causes with body mass index, age, and smoking taken as independent variables. Table IV gives the results. Body mass index was a significant predictor of acute myocardial infarction, but not of death, among men. Smoking and age were strong predictors of acute myocardial infarction and death from all causes among men. Among women only age contributed independently to the risk of death from all causes. The Cox regression analysis also showed that the effect of age on the risk of death and acute myocardial infarction was fairly constant among smokers and non-smokers across the body mass index quintiles.

We did not include further variables in the regression model. As shown in table V, body mass index was associated at baseline with coronary heart disease risk factors such as serum total cholesterol concentration and blood pressure. Including these variables in the multivariate model removes the significance of the effect of body mass index on acute myocardial infarction.[26]

Discussion

This analysis among middle aged men and women in eastern Finland supports the view that high relative body weight is related to the incidence of acute myocardial infarction among men and that the mortality risk gradients associated with relative weight are at the same time modest and strongly confounded by smoking. These results thus lie between the findings of Keys *et al*[14] and other workers,[30] who could not identify significant relations, and the positive reports from the Framingham population.[11 15 21]

The body mass index at which we found an increased risk of acute myocardial infarction in men was around 29·0-31·0 or more. This differs from the limit of 27·8 recently proposed for men[2] and even more so from the findings of the Framingham study, which suggest that a body mass index of more than 26·0 (approximate Metropolitan weight 120%) represents a risk factor for coronary heart disease.[15] We could not confirm that body mass index is a better predictor of coronary heart disease among younger men and among women.[11]

Levels and changes of relative body weight are correlated with levels and changes of coronary heart disease risk factors such as blood pressure, serum cholesterol concentration, glucose tolerance, and others.[31] Such correlations, also found in our sample, enhance the probability of detecting an association between overweight and coronary heart disease but are confounded by the problem

TABLE III—*Nine year risk of mortality from all causes and seven year incidence of acute myocardial infarction per 1000 population, and rate ratios between smokers and non-smokers in men aged 30-59 by body mass index quintile*

	Body mass index quintile (kg/(m²))									
	≤22·9		23·0-24·9		25·0-26·4		26·5-28·4		≥28·5	
	Smoker	Non-smoker	Smoker	Non-smoker	Smoker	Non-smoker	Smoker	Non-smoker	Smoker	Non-smoker
Mean age of men in stratum (years)*	44·5	41·2	43·6	42·6	43·8	43·6	43·8	44·5	44·6	46·3
Mortality from all causes 1972-80	107	28	87	25	77	36	98	39	119	54
Rate ratio	3·8		3·5		2·1		2·5		2·2	
Incidence of acute myocardial infarction 1972-8	68	22	95	28	66	37	90	29	144	49
Rate ratio	3·1		3·4		1·8		3·1		2·9	

*Difference in mean age between smokers and non-smokers significant ($p<0·01$) in lowest and highest body mass index quintiles.

TABLE IV—*Cox regression analysis on acute myocardial infarction and death from all causes in men and women with body mass index, smoking, and age as independent variables*

	Acute myocardial infarction		Death from all causes	
	Regression coefficient	Regression coefficient/SE	Regression coefficient	Regression coefficient/SE
		Men		
Body mass index	0·115	2·81*	0·011	0·30
Smoking	0·028	8·18***	0·027	6·48***
Age	0·095	12·07***	0·100	14·12***
		Women		
Body mass index	0·039	0·073	−0·026	0·44
Age	0·135	0·173	0·096	7·72***

*<0·05. ***p<0·001.

TABLE V—*Linear correlation coefficients (Pearson) for correlations between body mass index and age and other cardiovascular risk factors*

	Age	Serum total cholesterol concentration	Smoking	Mean arterial pressure	Leisure time physical activity
Men	0·06*	0·14***	−0·10**	0·30**	−0·05*
Women	0·35***	0·18***	−0·07**	0·46***	−0·07**

*p<0·05. **p<0·01. ***p<0·001.

of appropriate adjustment in multivariate analysis assessing the "independent" effect of relative weight on disease outcome—that is, the incidence of coronary heart disease. Adjusting for differences in blood pressure and blood lipid values at baseline would then be adjusting for the biological pathway through which obesity exerts at least part of its effect on the incidence of coronary heart disease. We therefore chose not to adjust for these biological risk factors when analysing the association between body mass index and the risk of acute myocardial infarction. From a strictly scientific viewpoint the established association between obesity and the incidence of acute myocardial infarction may therefore not be "independent." On the other hand, our results show that obesity is a marker not only for coronary heart disease risk factors but also for the occurrence of acute myocardial infarction in men.

In women the associations between body mass index and coronary heart disease risk factors were even stronger, but we could not find significant relations of body mass index with acute myocardial infarction or death. This may have been due to the better overall survival of women, with less than half the number of deaths than in men. Thus for women a much longer period of follow up would have been needed to detect statistically significant effects of the same magnitude as in men.

In other studies it has been difficult or even impossible fully to clarify the association between weight and mortality because of the confounding effect of smoking.[16 23 32] We also met this problem in our data. In the presence of smoking body mass index had no predictive value for survival or death, which suggests that smoking is a far more important predictor of mortality than overweight among the male population. Thus overweight smokers are likely to benefit much more from giving up smoking than from weight reduction.

J shaped and U shaped weight mortality curves with increased mortality among lean subjects have been explained as a "lean smoker effect"—that is, a smoking related higher mortality among smokers, who are known to be leaner on average.[15 16] In our sample, however, the J or U shape of the mortality curve was more clearly seen among non-smokers, especially in the age group 50-59. This higher mortality among the lean, also evident when controlling for smoking, has been reported[9 13] but is difficult to explain biologically. A U shaped pattern of the weight mortality curve may have resulted from weight loss before baseline due to diagnosed or undetected serious disease and early subsequent death during follow up. We could not control for this in our analysis but we excluded people with a history of recent cardiovascular events at outset.

Another way to counter the probable bias of undetected serious disease at baseline is to have a sufficient length of follow up (nine years in our study) and exclude events during the first one to two years. Workers in the Framingham study found that the full impact of obesity on cardiovascular diseases may become apparent only after a long "incubation period" of, say, two decades.[33] The survival curve among male non-smokers in our study tends to support the view that an appropriate length of follow up is essential to "wash out" baseline selection.

Many different methods have been used to determine obesity and despite the fact that body mass index (weight (kg) over height squared (m²)) may not be equivalent to measures of total body fat ($r=0·7$ between body mass index and percentage of hydrostatic fat[34]) it has been recommended and widely accepted as the measure of obesity in epidemiological surveys.[2] Though our study population was generally leaner and the variation in body mass index less than, for instance, in the United States population of the same age, we cannot explain our unequivocal results by the way in which obesity was measured.

On the one hand, obesity is probably strongly determined genetically[35] but, on the other hand, the lifestyle characteristics influencing obesity most—diet and physical activity—are both themselves related to mortality, certainly indirectly.[36 37] Those complex interrelations, in addition to the non-linear character of the association, may make it virtually impossible to isolate the "true" independent impact of relative weight on morbidity and mortality.[23 38]

In conclusion, our prospective study of a representative population sample of middle aged people in eastern Finland shows that a high body mass index is a significant predictor of acute myocardial infarction among men but not among women, that among male non-smokers the weight mortality curve is J shaped, and that smoking entirely outweighs relative weight as a predictor of risk of death.

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Passive smoking in utero: its effects on neonatal appearance

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Abstract

Smoking causes changes in the appearance of adults and has profound effects on the fetus, but little is known about its effects on the appearance of newborn infants. Two colour photographs (face and whole body) of 15 newborn infants (seven born to mothers who had smoked during pregnancy and eight born to mothers who had not) were shown to 100 medical and nursing staff, who in a double blind trial were asked to identify which babies had been born to smokers. The mean number correctly identified was 9·1, which was significant compared with the number expected by random selection (7·5).

No specific features were identified that distinguished the two groups of infants; selection was intuitive. Nevertheless, the fact that differences can be detected in some way may be useful for antismoking health education.

Introduction

Smoking in adults causes changes in facial appearance: smoker's wrinkles and facies.[1,2] Smoking during pregnancy has long been recognised to have profound effects on the fetus, but little mention has been made of its effects on the appearance of newborn infants. We report that infants born to mothers who smoke can be distinguished from those born to non-smokers by their appearance alone.

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Methods and results

Fifteen healthy white babies (11 girls and four boys), who were consecutive admissions to the routine care postnatal ward, were photographed under identical conditions on the first day of life. Two colour photographs were taken of each infant in daylight (face and whole body) when the infant was judged to be asleep or quietly awake by a modified Brazelton assessment.[3] All infants were born at term by normal vaginal delivery to healthy mothers after an uncomplicated pregnancy. None of the mothers abused alcohol or drugs. Seven of the infants were born to mothers who smoked (≥10 cigarettes a day). The range of birth weights was 2600-4120 g, the mean birth weight for infants at this hospital being 3440 g.

The photographs were shown to 100 medical and nursing staff (21 paediatricians at registrar level or above; 28 general practitioners or non-paediatric hospital medical staff; and 51 nurses, all of whom had had some paediatric nursing experience); the staff were not given any other information about the infants. Of the 100 interviewed, 38 were parents and 25 were smokers. Questioning was double blind, and each person interviewed had to decide for each infant whether his or her mother had smoked during her pregnancy.

The infants born to non-smoking mothers were heavier than those born to mothers who smoked (3425 g compared with 3170 g), but this difference was not significant, nor was there a significant difference in length or occipitofrontal circumference (Student's t test). Social class was equally distributed between the two groups.

The total number of infants identified correctly was 910 out of 1500, the mean number of infants identified correctly by each person interviewed thus being 9·1 out of the 15 (SD 1·84). The number of infants identified correctly