Health profiles of current and former smokers and lifelong abstainers

ABSTRACT – The aim of this study was to determine the extent to which smokers and smoking quitters differ in habits and risk factors from non-smokers. Subjects comprised 8,109 patients aged 35–67 years having health checks in British primary care. We compared lifestyle and measured cardiovascular risk factors in smokers, former smokers and lifelong abstainers in cross-sectional analyses, and in prospective data in quitters. Results were adjusted for confounding factors. Considering 25 aspects of lifestyle, smokers had significantly worse habits in 20 compared to abstainers, and in 17 compared to former smokers. These included eating more white bread, full cream milk, fried food and meat, and less fruit and vegetables, wholemeal bread and bran cereals. Smokers report drinking more alcohol and taking less exercise. Smokers' mean serum levels were higher for total and low density lipoprotein cholesterol and triglycerides and lower for high density lipoprotein cholesterol. Within five years ex-smokers' data became comparable to lifelong abstainers for most factors, with apparent attenuation over up to 20 years for triglyceride, body mass index and scores for fibre and polyunsaturated fat intake. Smokers who quit after initial examinations had better health profiles even before quitting (p = 0.016) and subsequently made more beneficial health changes (p = 0.039) than continuing smokers. Smoking is associated with relatively poor health choices and risk factor levels. Stopping smoking is associated with a wide range of improved health markers beyond avoidance of tobacco toxicity.

Reports from cross-sectional studies indicate adverse lifestyle and/or cardiovascular risk factors in smokers. Compared to non-smokers they have a poor diet, exercise patterns and adverse lipid profiles[1–11]. In the 1950s Karvonen *et al* observed higher serum total cholesterol levels in smokers related to how much they smoked[1] and Brozek and Keys noted an increase in body weight following smoking cessation[2]. Subsequent studies have documented a relatively higher calorific intake in male smokers and that, for past smokers, mean levels for a variety of nutritional factors tend to be intermediate between those for current smokers and lifelong abstainers[3–10]. Smokers have, dose related, relatively high mean serum concentrations of total and low density lipoprotein (LDL) cholesterol and triglyceride and low concentrations of high density (HDL) lipoprotein cholesterol[11]. Most of the variation in lipid levels in relation to smoking status has been attributed to diet[3]. It has been suggested that 9% of the 70% excess risk of coronary artery disease attributable to cigarette smoking may be due to higher serum total cholesterol concentration[11].

Most previous studies are inadequately controlled for potential confounding effects such as social class[11], though there are considerable social class differences in both smoking habit and diet[12]. We found no data comparing never, past and current smokers with regard to these differences in both lifestyle and objectively measurable risk factors within the same cohort. There are also no prospective lifestyle data for people who spontaneously stopped smoking[3].

In this paper we aim to clarify uncertainties in relation to smokers' lifestyles and risk factors and how these differ in those who quit smoking. We present a combination of cross-sectional and prospective data from participants in the OXCHECK (OXford and Collaborators HEalth ChecK) trial of nurse-based health checks performed in primary care[13].

Patients and methods

The data were collected at health checks in five urban general practices in Bedfordshire, UK. Participants were men and women aged 35–64 years in 1989. The health checks were completed by 1993. The methods of data collection, biochemical analysis[14–16] and the trial outcome[13] have been reported. Briefly, following standardised protocols, a nurse completed the Dietary Instrument for Nutritional Education (DINE) questionnaire and recorded habits of exercise, tobacco and alcohol consumption for each participant[16]. Blood pressure, height and weight were measured and blood was taken for analysis of serum lipid levels. Biochemical estimations were made at the Luton and Dunstable Hospital laboratory which subscribes to the Wellcome quality control scheme and the United Kingdom external quality assurance scheme. Smoking cessation was confirmed by estimation of serum cotinine[13]. Smokers were offered support and follow-up by the nurses if they wished to consider quitting. Nurses were trained in explaining the adverse consequences of tobacco use, the value of setting target dates for quitting and in distraction techniques for withdrawal symptoms. They

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Table 1. Reported lifestyle; odds ratios of the percentage of current and former smokers relative to never smokers

Food items (amount/frequency)	Never smokers (n = 3,217)	Current smokers (n = 2,218) Odds ratio (CI)	Former smokers (n = 2,674) Odds ratio (CI)	p value for trend[a]
Food of cereal origin				
Wholemeal bread ≥ 1 piece/day	1,390	556 0.49 (0.42, 0.58)	1,032 0.90 (0.78, 1.04)*	0.0038
Brown bread > 0 pieces/day	749	425 0.80 (0.67, 0.96)	551 0.89 (0.76, 1.06)NS	0.956
White bread ≥ 3 pieces/day	605	744 1.69 (1.42, 2.01)	683 1.11 (0.94, 1.32)*	0.191
Bran cereal > 0 times/week	656	223 0.49 (0.39, 0.60)	455 0.87 (0.73, 1.04)*	0.0078
Wheat cereal ≥ 3 times/week	1,140	440 0.46 (0.39, 0.55)	882 0.88 (0.76, 1.02)*	0.0006
Sugar cereal ≥ 3 times/week	419	286 0.89 (0.72, 1.11)	372 0.97 (0.79, 1.19)NS	0.188
Rice ≥ 3 times/week	370	196 0.79 (0.62, 0.88)	310 1.15 (0.93, 1.43)**	0.031
Food rich in fat				
Fried food ≥ 3 times/week	342	478 1.89 (1.54, 2.31)	342 1.02 (0.82, 1.27)*	0.814
Full cream milk ≥ 0.5 pint/day	755	835 1.73 (1.47, 2.03)	622 0.85 (0.72, 1.00)*	0.448
Meat ≥ 3 times/week	1,479	1,224 1.38 (1.19, 1.59)	1,336 1.08 (0.94, 1.24)**	0.024
Processed meat ≥ 3 times/week	733	693 1.30 (1.10, 1.53)	645 0.89 (0.75, 1.05)*	0.555
Beefburger ≥ 1 time/week	1,191	1,102 1.41 (1.21, 1.64)	1,158 1.11 (0.96, 1.29)*	0.866
Cheese ≥ 6 times/week	335	254 1.12 (0.89, 1.41)	309 1.12 (0.90, 1.40)NS	0.070
Cakes ≥ 6 times/week	640	356 0.73 (0.60, 0.88)	463 0.75 (0.63, 0.90)NS	0.036
Biscuits ≥ 6 times/week	1,142	726 0.85 (0.73, 0.99)	895 0.89 (0.77, 1.03)NS	0.727
Foods with less fat				
Poultry ≥ 3 times/week	651	258 0.58 (0.47, 0.71)	493 1.04 (0.87, 1.24)*	0.334
Semi-skimmed milk ≥ 0.5 pint/day	1,048	572 0.70 (0.60, 0.82)	845 0.92 (0.79, 1.06)*	0.387
Fish not fried ≥ 3 times/week	419	206 0.77 (0.61, 0.98)	328 1.04 (0.84, 1.28)***	0.170
Fruit and vegetables				
Potatoes ≥ 6 times/week	1,458	1,143 1.10 (0.94, 1.27)	1,312 0.94 (0.81, 1.08)NS	0.108
Peas ≥ 3 times/week	988	780 1.10 (0.93, 1.28)	889 1.01 (0.87, 1.17)NS	0.868
Beans ≥ 3 times/week	414	303 1.04 (0.84, 1.29)	349 1.05 (0.85, 1.29)NS	0.539
Other vegetables ≥ 6 times/week	2,566	1,537 0.65 (0.54, 0.76)	2,108 1.02 (0.86, 1.21)*	0.017
Fruit ≥ 6 times/week	2,265	939 0.36 (0.31, 0.42)	1,706 0.86 (0.74, 0.99)*	0.0003
Alcohol ≥ 11 units/week	429	693 2.42 (2.00, 2.92)	761 2.10 (1.68, 2.42)***	0.0037
Exercise; top of 3 levels	980	508 0.65 (0.54, 0.76)	799 1.00 (0.86, 1.17)*	0.139

Odds ratios adjusted for age, sex and social class; 2,496 had information on years since stopping smoking.

CI: 99% confidence interval.

[a] p value for trend in former smokers of the relative odds with years since quitting (years split as in Table 2). Trend is towards more healthy lifestyle in all significant cases, assuming less alcohol is more healthy.

Compared to current smokers *$p \leq 0.0001$; **$p \leq 0.001$; ***$p \leq 0.01$; NS, not significant.

were taught about, and encouraged to offer, nicotine replacement therapy.

We analysed the data from all 8,109 first health checks as participants entered the trial, stratified into the three groups; continuing smokers, former smokers and never smokers. Self-reported data on diet, exercise and alcohol are presented as odds ratios for current and former smokers in relation to never smokers (Table 1). We examined serum lipid levels, body mass index (BMI; height/weight ratio, kg/m^2), blood pressure and the summary scores for diet (Table 2). To gain an understanding of the differences over time which might accompany smoking cessation, we categorised former smokers by years since quitting.

To establish whether those who quit smoking during the trial were already healthier than persisting

Table 2. Mean levels of objectively measured risk factors and dietary scores (standard deviation) according to smoking status and years since quitting smoking

	Never smokers	Current smokers	All former smokers	Former smokers; years since quitting				*p* value for trend since quiting
				1–5	6–12	13–20	>20	
Number	3,217	2,218	2,496	442	712	770	572	
BMI	26.0 (0.08)**	25.3 (0.09)*	26.4 (0.08)	27.2 (0.20)	26.5 (0.16)	26.3 (0.15)	26.1 (0.18)	0.008
Systolic BP	128.8 (0.34)*	127.4 (0.39)NS	127.1 (0.36)	126.4 (0.86)	129.0 (0.68)	126.3 (0.66)	126.6 (0.76)	0.312
Diastolic BP	77.7 (0.21)*	75.4 (0.25)NS	76.2 (0.23)	75.3 (0.54)	77.0 (0.43)	75.9 (0.41)	76.6 (0.48)	0.312
Total cholesterol	6.11 (0.02)NS	6.28 (0.02)*	6.14 (0.02)	6.17 (0.06)	6.20 (0.04)	6.04 (0.4)	6.13 (0.05)	0.067
HDL cholesterol	1.39 (0.01)NS	1.30 (0.01)*	1.40 (0.01)	1.40 (0.02)	1.41 (0.01)	1.39 (0.01)	1.39 (0.02)	0.410
LDL cholesterol	3.88 (0.02)NS	4.02 (0.02)*	3.84 (0.02)	3.88 (0.05)	3.86 (0.04)	3.79 (0.04)	3.85 (0.04)	0.407
Triglyceride	1.85 (0.03)**	2.10 (0.03)***	1.98 (0.03)	2.01 (0.07)	2.03 (0.05)	1.89 (0.05)	1.93 (0.06)	0.014
Fibre score	37.4 (0.20)*	32.0 (0.23)*	36.1 (0.21)	34.2 (0.50)	35.8 (0.40)	36.9 (0.39)	37.6 (0.35)	0.0001
Fat score	33.5 (0.21)**	36.1 (0.24)*	32.4 (0.22)	32.1 (0.53)	32.6 (0.42)	32.6 (0.41)	32.1 (0.47)	0.801
PUFA score	8.35 (0.04)NS	7.89 (0.04)*	8.39 (0.04)	8.28 (0.09)	8.28 (0.07)	8.45 (0.07)	8.55 (0.08)	0.020

Numbers adjusted for age, social class, sex, and body mass index in all analyses.

Compared to 'all' former smokers *$p \leq 0.0001$; **$p \leq 0.001$; ***$p \leq 0.01$; NS, not signifcant.

Twenty-seven participants with no data on smoking status were excluded, 151 claiming smoking cessation within one year prior to trial entry, but unconfirmed by cotinine estimation, were excluded.

BMI, body mass index; BP, blood pressure; HDL, high density Lypoprotein; LDL, low density lipoprotein; PUFA, polyunsaturated fatty acids.

smokers, we explored the differences between these groups in the baseline data from first health checks (Table 3). This analysis was confined to entrants during the first two years of the study.

In addition, we prospectively examined lifestyle in smoking quitters and continuing smokers (Table 4). We determined the numbers in each group showing improvement when comparing individuals' responses at trial outcome examinations (1992–3), with those from first health checks (1989–91). We defined improvement as shown in Table 4, eg fat score decrease, fibre score increase. For two reasons this analysis was restricted to those attending final examinations: first, had non-attenders been included with an assumption of no change from baseline (intention to treat analysis) the quitters would have appeared (perhaps exaggeratedly) more different from continuing smokers. Secondly, the non-attenders to follow-up seemed generally to be at higher risk[13].

DINE provides a qualitative and semi-quantitative assessment of diet and was designed to be completed during primary care health checks. It had been previously validated and enables the categorisations found in the tables[16]. The fibre score combines data on intake of bread, cereals and fruit and vegetables; the fat score assesses intake of saturated fats in meats, fried food, pastries, confectionery and dairy products; and the polyunsaturated fatty acids (PUFA) score rises with increasing intake of vegetable, as contrasted with animal based cooking oils for frying, baking and dressing food.

Data from the DINE questionnaire were used to assess the proportion reporting a more or less healthy dietary choice. Trend tests were done only on former smokers. Data were analysed using the Statistical Analysis System, employing logistic regression and generalised linear modelling regression techniques. The data were adjusted for confounding factors as in the table footnotes. The Wilcoxon signed rank and t-tests were used where appropriate.

Results

Table 1 shows the cross-sectional data from continuing smokers, former smokers and never smokers for self-reported food choices, alcohol intake and exercise. Table 2 gives data for the same groups in relation to the measured risk factors and summary scores for dietary intake. The findings indicate that, for most measures, the lifestyle and risk factors of smoking quitters equate to those of never smokers. The gap between never and former smokers is closed to a large extent within five years of quitting, in parallel with the change in the risk of ischaemic heart disease after smoking cessation[17].

The *p* values for trend in relation to years since quitting smoking are significant for intake of a variety of foods (Table 1, final column), and for BMI, serum triglyceride levels and scores for fibre and PUFA in Table 2. The data suggest that over as many as 20 years after stopping smoking there is gradual attenuation of some differences from never smokers.

Table 3. Baseline diet and lifestyle of persistent smokers and future quitters

Lifestyle component	Persistent smokers (n = 1,144)	Future quitters (n = 95)	Odds ratio (95% CI) (quitters/smokers)
Other vegetables ≥ 6 times/week	791	70	1.20 (0.73, 1.97)
Fruit ≥ 6 times/week	466	45	1.15 (0.73, 1.80)
Alcohol ≥ 11 units/week	376	26	0.84 (0.50, 1.40)
Exercise as sport ≥ 1 times/week	166	20	1.55 (0.88, 2.72)
	Mean (standard deviation)		p value
Fat score	36.3 (12.2)	36.3 (12.4)	0.98
PUFA score	7.76 (2.06)	8.13 (1.90)	0.09
Fibre score	31.9 (10.2)	36.8 (12.9)	< 0.001

*As defined in Table 1.

Odds ratios adjusted for age, sex, social class, number of cigarettes per day and years of smoking.

Analysis includes all smokers at first health checks during years 1 and 2 of the trial.

Smokers are the group more likely to make food choices which are associated with poorer outcomes for both cancer and cardiovascular disease. They report eating less wholemeal bread, bran and wheat cereal, semi-skimmed milk, fish, poultry, fruits and vegetables apart from the staples (potatoes, peas and beans). For most comparisons with 'never smokers' the order of magnitude difference for the categories defined in Table 1 is around 50% less. Smokers report a higher consumption of similar magnitude for less healthy choices such as full cream milk, fried food, and processed meats and burgers. Smokers take significantly less exercise and drink more alcohol than former smokers and those who never smoked. In the 25 analyses in Table 1, current smokers reported statistically significantly poorer health choices in 17 of the comparisons with former smokers, and in 20 of those with lifelong abstainers. The habit showing least difference between past and persistent smokers is the relatively higher alcohol consumption than never smokers, though the trend to drink less becomes significant over time following smoking cessation.

The self-reported dietary data are supported by the quantitative estimations of intake, the fibre and PUFA scores being lower and the fat score higher (ie all less healthy) in the continuing smoking group. In turn, these dietary data are supported by the objective measurements in smokers of higher mean serum levels of total cholesterol, triglycerides and low density lipoprotein and lower high density lipoprotein levels.

Table 3 gives the baseline data from the 95 participants who stopped smoking as confirmed by cotinine measurements. Compared with those who subsequently continued smoking, the data from those destined to quit during the trial indicated a more healthy lifestyle for six of the seven factors analysed (p = 0.016), assuming reduced alcohol consumption is an improvement. At the end of the trial those who had stopped smoking (Table 4) reported more beneficial lifestyle improvements than persistent smokers for all factors except fibre score (p = 0.039).

A further examination of the data confirmed a dose-response relationship of adverse lifestyle and risk marker levels rising with increasing tobacco consumption. This has been previously reported[3,5]; therefore data are not shown.

Discussion

Data relating smoking to lifestyle and risk factors are subject to confounding. It is incautious to rely only on self-reported, potentially biased lifestyle information, or only on measured risk factors, as these two have a dynamic interrelationship. It is essential to consider separately the three selected population groups: those whose personality and preferences led them never to take up smoking, those who have smoked at some time but have had the determination and perhaps health awareness to quit, and those who in middle age are current smokers despite all the adverse outcomes. It is also necessary, before attempting to reach conclusions, to find concordance between cross-sectional and prospective data.

We have shown, following appropriate adjustment for confounders, that there is a plausible explanation for the differences in lipid profiles between never, previous and current smokers. The last have relatively high levels of total serum cholesterol and low density lipoprotein and low levels of high density lipoprotein cholesterol. These are compatible with the relatively high intakes of a range of lipid rich foods and cooking methods which they report using. The slightly lower

Table 4. Trial end; numbers of persistent smokers and confirmed quitters with improvement in diet and lifestyle from baseline data

Lifestyle changed	Persistent smokers (n = 760)	Confirmed quitters (n = 95)	Odds ratio % (quitters/smokers) (95% CI)
Vegetable intake increased	119	19	1.41 (0.81, 2.46)
Fruit intake increased	186	31	1.55 (0.96, 2.50)
Alcohol intake reduced	281	36	1.03 (0.66, 1.62)
Exercise level increased	117	17	1.16 (0.64, 2.10)
Fat score decreased	504	72	1.73 (1.04, 2.87)
PUFA score increased	400	52	1.08 (0.69, 1.69)
Fibre score increased	417	47	0.81 (0.52, 1.26)

Odds ratios adjusted for age, sex, social class, number of cigarettes per day and years of smoking, allowing for missing values.
Analysis includes smokers at trial entry (years 1–2), who also attended final re-examinations (year 4).

blood pressure in smokers may not be attributable to lower body mass index, as adjustment was made for that in our analysis.

In our cross-sectional data we find a transition in those who quit, from the less healthy habits of continuing smokers (Table 1). The analyses indicate that the reported changes towards more healthy habits have significant trends over time for various dietary items, eg wholemeal bread, wheat cereal and rice. The comparisons for measured risk factors between the groups of former smokers (Table 2) suggest that some of the differences from those who have never smoked are no longer seen within the first five years after quitting, eg in high and low density lipoprotein. Other factors may remain less healthy in quitters than abstainers over many years, eg fibre intake and serum total cholesterol level. These data are similar to those reported over a shorter time period since quitting smoking, for estimates of dietary nutrients between never, past and current smokers by one other survey[8].

It could be that the population who had quit smoking for more than five years is in some way different from the more recent quitters, in which case our time trend data would have no meaning. We find, however, that the differences from continuing smokers, in the 'years since quitting' groups, are generally stable for both risk factors and lifestyle changes. Further, the trend analysis is compatible with continuing improvements over time (eg Table 2; total, LDL and HDL cholesterol and fat score). Cross-sectional analyses are prone to bias and confounding, but our prospective data (below) are supportive.

The data from participants who stopped smoking during the trial indicate a trend to a healthier lifestyle prior to smoking cessation (Table 3). Compared with continuing smokers, the quitters were more likely to eat more fruit and vegetables, take more exercise, and have higher fibre and PUFA scores and lower alcohol consumption. The prospective data after stopping smoking (Table 4) suggest further improvements in the various measurements. The exception is fibre score, which showed least change over time (Table 2) and greatest baseline difference from persistent smokers (Table 3). The inference from these findings is important. It suggests (albeit from a small number of subjects) that the population of quitters may not simply be a healthier subgroup, but one that makes positive changes after smoking has been stopped. This agrees with the findings in our cross-sectional analysis.

Other cross-sectional British surveys have contrasted diets of smokers with non-smokers: the Health and Lifestyle Survey of British adults and Scottish Heart Health Study included former smokers as a separate group, and the latter also examined the temporal patterns in data from past smokers[6–8]. One survey reported on lipids, but only in relation to current smoking status[9]. The Scottish data were analysed in relation to individual nutrients and percentage of energy from differing food sources and are exceptional in being reported fully adjusted for social class[8]. The survey analysed by Margetts and Jackson included a seven-day dietary record and weighed food intake, but was not able to categorise former smokers[10]. Collectively, the data from these British reports support our own findings and those of others in relation to the diets of never and current smoking groups.

Various hypotheses have been put forward to explain the association of tobacco consumption with unhealthy lifestyle and the differences documented in health markers. Wack and Rodin, examining the relation between body weight and tobacco consumption, concluded that nicotine reduces the efficiency of calorific storage and/or increases metabolic rate[4]; a genetic difference from non-smokers, implicating a smoking prone personality has been suggested[18]. The

toxic effects of tobacco smoke on taste, smell and satiety, with consequent modification of food choices, could be a partial explanation[4]. Health attitudes and beliefs are also likely to have significant effects as evidenced by a questionnaire survey[19]. In response to 'current diet perceived harmful' and 'tried to change diet' the odds ratios (confidence interval) for smokers' responses compared to the general population were 0.65 (0.54, 0.79) and 0.73 (0.64, 0.84), respectively. In contrast in hypertensive respondents, who have a similar cardiovascular disease risk, odds ratios were 1.35 (1.08, 1.68) and 1.04 (0.87,1.24), respectively[19]. Further support for the influence of health attitude must come from our data. It is not conceivable that the smokers' relative preferences for full fat milk, butter, fried foods, processed meats and white bread, as well as less exercise and more alcohol and relative disdain for poultry and fish, could all be attributed simply to genetics or tobacco toxicity.

It seems likely that the adverse pattern of health beliefs in smokers is dynamic and the changes in attitude which provide the motive to quit smoking are also reflected in a variety of lifestyle modifications in those who stop. If this is so, the message to those involved in helping smokers to quit is to direct their efforts towards modification of general health beliefs and attitudes rather than pursue simple didactic messages.

An important implication of the unhealthy diet of smokers is that interpretation of smoking-attributable risks for development of cancer and cardiovascular disease should take account of dietary influences as well as direct carcinogenic or otherwise toxic effects of tobacco smoke. Likewise some of the reduction of risk of disease following smoking cessation may be attributed to improvements in dietary and exercise patterns. For instance, smoking cessation was associated with a considerable increase in the proportion reporting high consumption of fruit and certain categories of vegetables. Diets rich in fruit and vegetables have been independently associated with a reduction in the incidence of ischaemic heart disease and various cancers, including lung cancer[20,21]. It would be unwise, though, to overemphasise the lifestyle influences in this context. It must be remembered that the link between smoking and lung cancer and other diseases has been established in a professional group with above average access to knowledge of the adverse effects of an unhealthy diet[22]. Further, the toxicity of passive tobacco smoke[23] is not likely to be mediated by effects on the lifestyle of the recipient.

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