International Journal of Innovative Research and Review ISSN: 2347 – 4424 (Online)
An Online International Journal Available at http://www.cibtech.org/jirr.htm
2015 Vol. 3 (1) January-March, pp.11-20/Ghouse
Research Article

SMOKING CESSATION EFFECTS ON BODY WEIGHT, HAEMOGLOBIN, TOTAL AND HDL CHOLESTEROL AND BLOOD PRESSURE

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ABSTRACT
Tobacco use including both the smoking and the nonsmoking forms of tobacco is common in India. The World Health Organization reported that tobacco smoking killed 100 million people worldwide in the 20th century and warned that it could kill one billion people around the world in the 21st century. The increase in body weight may mediate between smoking cessation on the one hand and total cholesterol and blood pressure change on the other hand. Hemoglobin concentration increases in smokers because the inhaled carbon monoxide results in increased carboxyhemoglobin, which has no oxygen-carrying capacity Smoking intensity also has been associated with small, statistically significant increases in low-density lipoprotein cholesterol (LDL-C) and decreases in HDL-C. Some have described small dense LDL particles among current smokers and improvements in lipids after smoking cessation; however, these findings have been less consistent. This emphasizes that some, at least, of the adverse effects of smoking appear to be rapidly reversible on quitting, strengthening the argument for encouraging smokers to quit.

Keywords: *Smoking, Quitting, Haemoglobin, Body Weight, HDL Chlolesterol*

INTRODUCTION
Tobacco use including both the smoking and the nonsmoking forms of tobacco is common in India (Jindal *et al.,* 2006). The number of smokers in the population of the Third World will increase from 4.5 billion to 7.1 billion by 2025. The World Health Organization reported that tobacco smoking killed 100 million people worldwide in the 20th century and warned that it could kill one billion people around the world in the 21st century (WHO Report, 2008). Each year, smoking contributes to more than 443,000 smoking related deaths in the United States and nearly 20% of all coronary heart disease deaths can be attributed to smoking (American Heart Association, 2009; Center for Disease Control and Prevention, 2009). Tobacco smoking rates have decreased in industrialized countries since 1975, but there has been a corresponding 50% increase in smoking rates in low- income countries (Yu *et al.,* 1989). Although the strong relationship between smoking and cardiovascular disease (CVD) has been well-documented. Several means by which smoking increases the risk of cardiovascular disease have been postulated. First the formation of carboxyhemoglobin in the blood results in anoxemia in the myocardium, thereby weakening it. Second, smoking is known to increase vasoconstriction, platelet aggregation and adhesion, in effect, increasing the blood's clotting ability. Third, nicotine may chemically induce various cardiac arrhythmias. This effect may result directly from the elimination of the harmful effects of cigarette smoke, such as increased heart rate and myocardial contractility, decreased oxygen transport of the blood and increased blood coagulation. In addition, smoking cessation may also have an effect on other risk factors for CVD, such as serum total and HDL cholesterol level, blood pressure and body weight.
The blood haemoglobin estimation is one of the most frequently used laboratory parameters in clinical settings. Hemoglobin values, however, vary with age, sex, and stage of pregnancy, and they are also affected by ethnicity, altitude, and smoking.
Alterations in blood pressure (BP), heart rate (HR), and autonomic nervous function are thought to be at least in part responsible for the rapid reduction in the risk of cardiovascular diseases after quitting. Ex-smokers may adopt a healthier lifestyle, which may result in a decreased body weight, total cholesterol level or blood pressure. Changes in body weight will consequently alter serum total and HDL cholesterol level and blood pressure levels. Since CVD risk is associated with the duration of cessation and the

amount of smoking, changes in CVD-risk factors may also be associated with these characteristics of smoking behavior.

Arteriosclerotic lesions throughout the vascular system are more prevalent in smokers than in non-smokers. Cigarette smokers have also been shown to have significantly lower levels of high-density lipoprotein cholesterol (HDL-C) and higher low-density lipoprotein cholesterol (LDL-C) levels in their blood than non-smokers. Smoking intensity also has been associated with small, statistically significant increases in low-density lipoprotein cholesterol (LDL-C) and decreases in HDL-C. Some have described small dense LDL particles among current smokers and improvements in lipids after smoking cessation; however, these findings have been less consistent. To what extent the reduction in CVD risk after smoking cessation is due to smoking-related changes in other risk factors is not established yet in the general population. The effect of smoking cessation on body weight, total and HDL cholesterol levels and blood pressure was examined under controlled circumstances in several smoking intervention trials or in several cohort studies among special groups of the population. This is a matter of considerable importance, because smokers in the 21st century are significantly more overweight than those studied previously (Gossett *et al.*, 2009; Campbell *et al.*, 2008; Center for Disease Control and Prevention, 2009). Since smoking cessation is associated with weight gain (Eisenberg and Quinn, 2006; Johnson *et al.*, 2010; Flegal *et al.*, 1995) and weight gain affects lipoproteins (Dattilo *et al.*, 1992; Hession *et al.*, 2009) the effects of smoking cessation on lipoproteins remains unclear. Results obtained from the general population are of greater importance for public health policy makers, since they are derived from a sample of the total population under natural and uncontrolled conditions. In these general-population cohort studies, an unfavourable increase in body weight was observed after smoking cessation. Therefore, in the present study, we used longitudinal data from the general population to study the change in body weight, serum total and HDL cholesterol level and systolic and diastolic blood pressure in subjects who stopped smoking during follow up. Furthermore, we studied the effect of amount of smoking before cessation and duration of cessation on these changes in CVD risk factors. Finally, we examined whether these changes were influenced by other factors, including body weight change.

MATERIALS AND METHODS

Subject was examined on Cardiovascular Disease Risk Factors, of 20-59 year old men from Kurnool. Weight change due to smoking cessation may not be distinguishable from weight change due to a weight reducing diet or smoking-related chronic diseases, such as CVD, or COPD. 210 never smokers, ex-smokers, 83 and 200 current smokers at both examinations, quitters between baseline and re-examination, 137 ‘relapsers’ (ex-smokers at baseline who smoked at re-examination) and 160 subjects who were occasional smokers (less than 1 cigarette per day) or who had inconsistent smoking information across the two examinations. All subjects gave their written informed consent.

Measurements

1. Subjects were classified as persistent smokers if they reported to smoke 1 cigarette or more per day at both examinations, as quitters if they smoked 1 cigarette or more per day at baseline and reported to have quit smoking at the re-examination, and as never smokers if they did not smoke at both examinations.
2. For persistent smokers and quitters, information on the number of cigarettes smoked per day at baseline was collected and categorized as 1-9, 10-19 and > 20 cigarettes per day.
3. For quitters, the duration of cessation was established at the re-examination and categorized as 0-1 years, 2-3 years and 4-6 years.
4. After taking antiseptic precautions blood samples were drawn from the antecubital vein and collected into 3m1 EDTA vacutainers (Akuret, eastern medkit limited). The EDTA blood samples were processed by using MS-9 automated. Hematology cell counter for Haemoglobin. Samples were processed on the same day within 3-5 hours of collection. Data collected was subjected to standard statistical analysis by SPSS software.
5. Lipid profile was estimated by drawing 5ml of fasting blood sample. Serum lipids and lipoproteins cholesterol fractions were measured on fasting state. Total cholesterol and triglyceride level estimations

are carried out using enzymatic end point kit method. HDL cholesterol was estimated by precipitation of non HDL lipoproteins and estimations done using superannuant. Total and HDL cholesterol levels were determined at the Clinical Laboratory.

6. Blood pressure was measured twice on the upper arm with the participant in sitting position, with a random-zero sphygmomanometer. Systolic and diastolic blood pressure was recorded at the first and fifth and fifth Korotk off phase, respectively. The average of the two blood pressure measurements was used in the analyses.
7. Height was measured with as wall-mounted stadiometer in subjects without shoes who stood upright against the wall.
8. Body weight was measured in light indoor clothing without shoes, and 1 kg was subtracted to correct for clothing.

Information was obtained by means of a questionnaire. Educational level at baseline, as an indicator of socio-economic status, was divided into three categories, according to the highest achieved level of education: low (intermediate secondary education or less), intermediate (intermediate vocational or higher secondary education) and high (higher vocational or university education).

Alcohol intake was reported in number of glasses of beer, wine or spirits per week at both examinations. We transposed this into the number of glasses alcohol per day, assuming equal amounts of alcohol intake between the two examinations.

Their previous history related with of myocardial infractions, cerebro-vascular accident, diabetes mellitus or cancer etc., respiratory symptoms, including chronic cough, chronic phlegm, and shortness of breath and wheezing are taken in to consideration

Statistical Analysis

Differences in characteristics between persistent smokers on the one hand and quitters and never smokers on the other hand were tested by means of a t-test for continuous variables or by means of x2 –test for categorical variables.

Multivariate analysis of variance was used to estimate changes in body weight, total and HDL cholesterol and blood pressure level between baseline and re-examination. These changes were estimated for persistent smokers, quitters and never smokers and for quitters in categories of duration of cessation. Differences in changes between categories were tested with the total group of persistent smokers as the reference group. Furthermore, changes were estimated for persistent smokers and quitters in categories of amount of smoking. In these analyses, differences in changes between quitters and persistent smokers were tested in subgroups of amount of smoking with persistent smokers as the reference group. A trend in amount of smoking with change in risk factors was tested using linear regression modeling. Haemoglobin Data collected was subjected to standard statistical analysis by SPSS software

Data were analysed using the GLM-procedure of SAS statistics version 6.12. Two-sided p-values below 0.05 were considered statistically significant.

RESULTS AND DISCUSSION

Results

There were no significant differences in baseline characteristics between persistent smokers and quitters, except for educational level and for body mass index (BMI) among men and number of cigarettes per day among women (Table 1). At re-examination, quitters reported a lower prevalence of asthma and COPD among men and of cancer and COPD among women.

Table 2 reflects the age-adjusted changes in body weight, total and HDL cholesterol level and blood pressure for categories of smoking behavior. Compared to persistent smokers, quitters experienced a larger age-adjusted increase in body weight, HDL cholesterol and diastolic blood pressure (DBP) between baseline and re-examination among both men and women (excess gain in quitters compared to persistent smokers: body weight 3.4 kg among men and 3.8 kg among women; HDL cholesterol 0..7 mmol/L among both men and women; DBP 2.8 mm Hg among men and 1.7 mm HG among women).

Table 1: Baseline characteristics of the study population (men (sd) or%)

	Men			**Women**		
	Persistent smokers	**Quitters**	**Never smokers**	**Persistent smokers**	**Quitters**	**Never smokers**
N	200	83	210	50	15	100
Age (years)	38.2(9.6)	37.0(10.3)	38.3(9.8)***	37.4(9.2)	37.0(10.0)	42.7(11..0)***
Educational level(%):						
Low	67.7	53.6	40.3	75.6	77.2	66.4
Intermediate	21.7	26.3**	33.8***	16.1	10.4*	20.3***
High	10.6	20.1	26.0	8.3	12.4	13.3
Nr of cigarettes per day	16.9(7.9)	15.7(8.0)	--	13.2(6.9)	10.5(6.1)***	---
Body weight (kg)	71.0(11.7)	74.5(10.2)	75.2(10.0)	62.6(10.1)	63.3(9.2)	66.9(10.6)***
BMI(kg/m^2)	24.7(3.2)	24.3(2.7)*	24.5(2.9)	23.4(3.4)	23.5(3.3.)	24.4(3.8)***
Total cholesterol (mmol/l)	5.65(1.19)	5.48(1.10)	5.30(1.04)***	5.41(1.06)	5.46(1.04)	5.32(1.00)
HDL cholesterol (mmol/l)	1.07(0.26)	1.10(0.26)	1.16(0.26)***	1.30(0.31)	1.29(0.29)	1.39(0.28)***
Systolic blood pressure(mm Hg)	124.8(12.8)	123.5(12.6)	124.5(12.9)	115.3(13.5)	114.4(13.6)	117.6(13.8)***
Diastolic blood pressure(mm Hg)	77.5(9.8)	76.0(9.8)	77.3(9.8)	72.7(9.4)	71.8(9.4)	74.5(9.4)***
Alcohol intake:						
% drinkers	73.5	75.8	62.2***	47.5	43.2	37.2***
No of glasses/day among drinkers	2.3(1.8)	2.1(1.6)	1.4(1.2)***	1.3(1.0)	1.1(0.9)	0.7(0.5)***

** p<0.05, **p<0.01, ***p<0.001 for a difference with persistent smokers*

Table 2: Chapt Age –adjusted change (95% confidence interval) between baseline and re-examination in body weight, total and HDL cholesterol, and systolic and diastolic blood pressure for the total groups of persistent smokers. Quitters and never smokers, and for quitters in categories of duration of cessation

	Body weight (kg)	Total cholesterol (mmol/L)	HDL cholesterol (mmol/L)	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg)
Men					
Persistent smokers	3.2(2.8-3.4)	-0.04(-0.10-0.02)	0.05(0.3-0.06)	2.3(1.1-3.0)	2.0(1.2-2.7)
Quitters	6.2(5.9-7.2)	0.13(0.01-0.24)*	0.12(0.10-0.15)***	4.6(2.9-6.6)*	4.3(3.2-6.1)**
Never smokers	3.5(3.5-4.1)**	0.07(0.01-0.13)**	0.06(0.05-0.08)	2.4(1.6-3.5)	2.4(1.8-3.3)
Duration of cessation (years)[a]					
0-1	5.3(3.9-7.1)***	0.10(-0.12-0.32)	0.09(0.03-0.15)	2.6(-0.7-6.3)	2.2(-0.2-5.1)
2-3	6.8(5.4-8.8)***	0.31(0.17-0.55)**	0.15(0.09-0.22)***	4.1(0.5-8.2)	3.1(0.4-6.2)
4-6	6.5(5.5-8.0)***	0.01(-0.17-0.18)	0.14(0.09-0.18)***	6.6(4.1-9.8)**	6.7(4.8-9.1)***
Women					
Persistent smokers	2.8(2.6-3.4)	0.05(-0.002-0.11)	0.12(0.10-0.14)	4.3(4.0-5.9)	2.7(2.2-3.6)
Quitters	6.5(6.1-7.5)***	0.03(-0.08-0.14)	0.19(0.15-0.23)***	5.3(3.8-7.7)	4.4(3.2-5.9)*
Never smokers	3.8.(3.7-4.4)***	0.12(0.07-0.16)	0.12(0.10-0.14)	3.9(3.5-5.2)	2.6(2.2-3.4)
Duration of cessation (years)[a]					
0-1	5.4(3.5-6.8)**	-0.02(-0.23-0.20)	0.22(0.15-0.29)**	5.6(1.9-9.8)	3.9(1.3-6.9)
2-3	7.6(6.3-9.6)***	0.02(-0.19-0.23)	0.19(0.13-0.26)*	4.9(1.4-9.1)	5.8(3.3-8.7)*
4-6	6.3(6.1-9.0)***	0.04(-0.15-0.23)	0.17(0.11-0.23)	3.9(0.8-7.8)	2.9(1.2-6.1)

**P<0.05, **p<0.01, ***p<0.001 for difference with persistent smokers. [a] Among quitters*

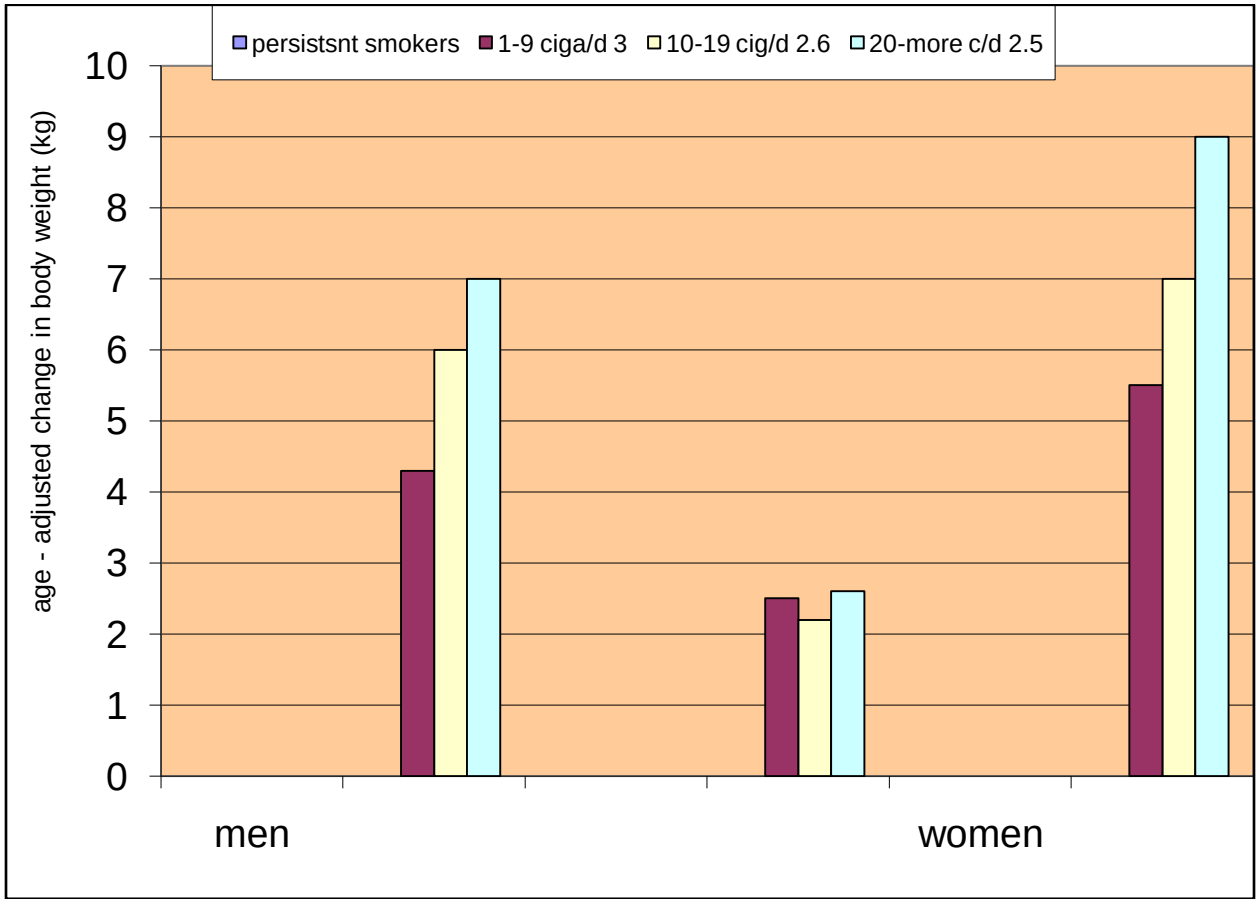

Figure: Age adjusted change in the body weight between baseline and re examination, for persistent smokers and quitters in categories of amount of smoking at base line *p for trend

Furthermore, in men only, quitters experienced a larger age-adjusted increase in total cholesterol and systolic blood pressure (SBP) (excess gain in quitters compared to persistent smokers: total cholesterol 0.17 mmol/L; SBP 2.7 mm Hg).

Quitters who quit smoking 2-6 years before re-examination tended to gain more weight than quitters who quit smoking within 2 years before re-examination, which was most pronounced among women (Table 2). Furthermore, the gain in body weight showed a positive trend with amount of smoking within quitters.

Figure Age-adjusted change in body weight between baseline and re-examination, for persistent smokers and quitters in categories of amount of smoking at baseline *p for trend.<0.01), but no trend within persistent smokers, which implies that heavy smokers who quit smoking gained more weight than light smokers who quit smoking (Figure). No clear effect of duration of cessation and amount of smoking was observed on the change in total and HDL cholesterol level and blood pressure between baseline and re-examination. Among men, but not among women, the increase in HDL cholesterol level tended to be largest after at least 2 years of cessation. Furthermore, in men, change in both systolic and diastolic blood pressure seemed to be larger among long-term quitters than among quitters who quit within 4 years before the re-examination, but this was not observed in women (Table 2). Among women, but not among men, HDL change among quitters tended to be largest from 10 cigarettes per day onward (data not shown).

So far, we expressed the change in risk factors as the age-adjusted, actual observed change. This change in risk factors after smoking cessation may be explained by several factors. We examined the effect of several demographic and lifestyle factors, disease history, and weight gain, which are shown in Table 3. Adjustment for age, educational level, number of cigarettes per day at baseline, change in alcohol consumption and disease history did not substantially alter our adjusted results (Adjustment I).

Table 3: Change (95% confidence interval) between baseline and re-examination in body weight, total and HDL cholesterol, and systolic and diastolic blood pressure, adjusted for several factors, [a] for persistent smokers, quitters and never smokers

	Body weight (kg)	Total cholesterol (mmol/L)	HDL cholesterol (mmol/L)	Systolic blood pressure (mm Hg)	Diastolic blood pressure (mm Hg)
Men					
Adjustment 1					
Persistent smokers	2.9(2.6-3.5)	-0.002(-0.08-0.07)	0.05(0.09-0.07	1.4(0.2-2.7	1.5(0.5-2.5)
Quitters	5.9(5.8-7.2)***	0.14(0.02-0.26)*	0.12(0.09-0.15)***	4.2(2.2-6.2)*	4.2(2.9-6.0)***
Never smokers	3.9(3.4-4.3)*	0.03(-0.05-0.11)	0.06(0.04-0.08)	3.0(1.7-4.3)	2.6(1.9-4.0)
Adjustment II					
Persistent smokers		0.02(-0.05-0.10)	0.04(0.02-0.06)	1.9(0.7-3.01)	1.7(0.9-2.8)
Quitters	-	0.04(-0.08-0.16)	0.15(0.12-0.18)***	2.6(0.6-4.6)	3.1(1.6-4.8)
Never smokers		0.03(-0.05-0.11)	0.06(0.04-0.08)	3.0(1.7-4.2)	2.5(1.9-3.9)
Women					
Adjustment I					
Persistent smokers	2.6(2.2-3.3)	0.07(-0.01-0.15)	0.11(0.09-0.14)	4.2(2.8-5.6)	2.1(1.3-3.3)
Quitters	6.4(5.8-7.4)***	0.14(-0.08-0.16)	0.18(0.14-0.22)**	5.3(3.2-7.4)	4.1(2.9-5.9)*
Never smokers	4.2(3.8-4.7)***	0.11(0.04-0.18)	0.13(0.10-0.15)	4.7(3.5-5.9)	3.1(2.5-4.2)
Adjustment II					
Persistent smokers		0.09(0.02-0.17)	0.10(0.08-0.13)	5.0(3.6-6.3)	2.6(1.8-3.8)
Quitters		-0.02(-0.13-0.10)	0.20(0.17-0.24)***	3.6(1.5-5.6)	3.1(1.8-4.7)
Never smokers		0.10(0.03-0.17)	0.13(0.11-0.15)	4.5(3.3-5.6)	3.0(2.3-4.0)

**p<0.05, **p<0.001 for difference with persistent smokers. [a] Adjustment I includes age, educational level and number of cigarettes per day at baseline, change in alcohol consumption between baseline and re-examination and history of myocardial infarction, cerebrovascular accident, diabetes, cancer, asthma and COPD at re-examination; adjustment II includes adjustment I plus changes in body weight between baseline and re-examination.*

Table 4 depicts the effect of smoking on haemoglobin concentration. The mean value of haemoglobin in smokers was 14.22±0.79, while in non-smokers was 13.27±1.32 and the difference was statistically highly significant (p = 0.001).

Haemoglobin concentration of smokers and nonsmokers

	Smoking	Mean	p-value
Haemoglobin	**Smokers**	14.22±0.79	0.001
	Non Smokers	13.27±1.32	

After adjustment for weight gain, the increase in total cholesterol level, systolic and diastolic blood pressure among quitters tended to become smaller for both men and women, and the difference in total cholesterol (among males) and blood pressure change between quitters and persistent smokers lost significance (Adjustment II). The increase in HDL cholesterol level tended to become slightly larger after adjustment for weight gain.

Discussion

In this general-population study, quitters experienced a larger increase than persistent smokers in body weight. Men experienced a large increase in total cholesterol level and systolic blood pressure. Heavy smokers gained more weight after cessation than light smokers. Weight gain largely explained the larger increase in total cholesterol and blood pressure among quitters compared with persistent smokers and slightly counteracted the favourable increase in HDL cholesterol among quitters.

Study, shows that age-adjusted, observed changes in CVD risk factors after smoking cessation and examined to what extent confounding factors such as demographic and lifestyle factors, were responsible for these changes.

Limitations of our study include the possibility that quitters may have adopted other healthy lifestyle habits affecting body weight, cholesterol level and blood pressure, which we were not able to account for. For instance, non-smokers usually are more physically active than current smokers. Furthermore, non-smokers generally consume a healthier diet compared to smokers, especially heavy smokers. Dietary intake at both baseline and re-examination did not substantially differ between quitters and persistent smokers. Information on intake of alcoholic beverages, which also differs between heavy smokers and ex-smokers, was comparable between the two examinations and the change in alcohol consumption between baseline and re-examination was included in our analyses.

Hemoglobin concentration increases in smokers because the inhaled carbon monoxide results in increased carboxyhemoglobin, which has no oxygen-carrying capacity. Impaired tissue oxygen supply results from decreased oxygen carrying capacity and increased oxygen-haemoglobin affinity caused by carboxyhaemoglobin (COHb) (Sagone *et al.,* 1973). To compensate, hemoglobin levels increase.

A gain in body weight following smoking cessation has been widely reported. On average, male and female quitters gained 3.4 kg and 3.8 kg respectively more than persistent smokers in our study with six years of follow-up, which did not substantially change after adjustment for relevant variables. It is clear that smokers weigh less than non-smokers after many years of smoking. However, among adolescents and young adults, weight differences between smokers and non-smokers are small or non-existent, and smoking initiation is *not* associated with weight loss. In contrast, smoking cessation reliably produces weight gain. In several large prospective studies, weight gain attributable to smoking cessation has averaged

2 to 4 kg, and has been greater in women These excess gains were smaller than those found in a smoking cessation trial (6.3 kg and 6.8 kg for men and women respectively after 5 years of follow-up), but larger than those found in other smoking cessation trials, in cohort studies among specific groups of the population, or in other cohort studies among the general population, which reported excess gains from around 2 kg to 3.8 kg after 4-16 years of follow up. Larger excess gains in our study may be explained by

the longer duration of follow-up in some other studies, since it is suggested that, after an initial increase, body weight may decrease later in the follow-up period. .

The Nicotine in tobacco smoke stimulates the adrenal cortex, leading to consecutive releases of free fatty acids and very low density lipoprotein, which in turn results in lower HDL cholesterol levels. The absence of Nicotine after smoking cessation may reverse this process. HDL cholesterol has been identified as a protective factor against Cardiovascular diseases. It is estimated that the excess gain of quitters compared to persistent smokers of 0.07 mmol/L, as was found in our study, would decrease CVD mortality with around 5.5% among men and 8% among women. This indicates that this post cessation change in HDL cholesterol may contribute to the positive effects of smoking cessation on CVD.

Smoking cessation increased total cholesterol levels in men and systolic (significant in men only) and diastolic blood pressure levels in both men and women. Weight gain is known to increase total cholesterol and blood pressure levels. This indicates that the increase in body weight may mediate between smoking cessation on the one hand and total cholesterol and blood pressure change on the other hand.

These results suggest that quitters should prevent post cessation weight gain as much as possible in order to gain the maximum health benefits from smoking cessation.

In this large, prospective, contemporary study of current smokers, smoking cessation improved HDL-C, total HDL, and large HDL particle concentrations, despite weight gain. These findings were especially strong in women. Smoking cessation, not baseline smoking intensity, predicted increased HDL parameters. These findings suggest that an increase in HDL may mediate some of the reduced CVD risk observed after smoking cessation. Quitting smoking is clearly associated with an increase in HDL-C concentrations. Generally the increase occurs rapidly, in less than three weeks, with no clear pattern of change thereafter. This emphasises that some, at least, of the adverse effects of smoking appear to be rapidly reversible on quitting, strengthening the argument for encouraging smokers to quit. The high prevalence of an atherogenic lipid profile in smokers makes them prone to develop premature atherosclerosis and the changes become more marked with the number of cigarette/day smoked.

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