Can the Smoke-Free Ratio be a Novel Indicator of the Cardiovascular and Metabolic Risk Reduction of Former Smokers? A Cross-Sectional Study

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Objective: The association of smoking with many diseases is well known, as well as are the benefits of smoking cessation. While mentioning these benefits, the duration that passes after quitting smoking is always stressed. However, former smokers' history of smoking exposure is usually ignored. This study aimed to investigate the possible effect of the pack-years history on several cardiovascular health parameters.

Methods: A cross-sectional study was conducted on 160 ex-smoker participants. A novel index was described and named the "smoke-free ratio" (SFR), which is the number of smoke-free years divided by the number of pack-years. The associations between the SFR and various laboratory values, as well as anthropometric and vital measurements, were investigated.

Results: The SFR was negatively correlated with body mass index, diastolic blood pressure, and pulse in women with diabetes. In the healthy sub-group, fasting plasma glucose was negatively and high-density lipoprotein cholesterol positively correlated with the SFR. A Mann–Whitney *U* test revealed that the cohort with metabolic syndrome had significantly lower SFR scores (Z = -2.11; *P* = .035). In binary grouping, the participants with low SFR scores had higher rates of metabolic syndrome.

Discussion: This study revealed some impressive features about the SFR, which is proposed as a novel tool for estimating metabolic and cardiovascular risk reduction in former smokers. Nevertheless, the actual clinical significance of this entity remains unclear. [*P R Health Sci J 2023;42(1):10-15*]

Key words: Smoke-free ratio, Cumulative smoking exposure, Benefits of smoking cessation

There is much research on the cumulative harm of smoking, and the pack-years formula has been a subject of interest to the scientific world for a long time (1,2). Many scientists considered the pack-years concept to be the most well-recognized and useful indicator for calculating cumulative exposure to tobacco-related toxins (3). Some studies have shown a stronger association of smoking duration with some diseases than has been seen with the composite of pack-years (4–6). It can be inferred that, for example, smoking 10 cigarettes per day for 40 years would produce a higher risk compared to that generated by smoking 20 cigarettes per day for 20 years, although both sets of circumstances yield 20 pack-years. However, some other studies have stressed the amount of smoking as an independent predictor of severe health issues (7,8). The shortcomings of the pack-years formula have been discussed recently, as well, such as that it does not take into account other types of tobacco products (e.g., loose tobacco) and ignores the independent effects of age and the type of tobacco (e.g., black vs. blonde tobacco) (9–11).

In addition to all those debates about the reliability of the pack-year concept, in the literature, the expected benefits of quitting smoking have only been linked to the length of time that the former smoker remains smoke-free (12). It was believed that the earlier you quit smoking (especially before age 35), the more benefits you would gain (13,14). All these assumptions reckon without the possible effect of the cumulative smoking history of former smokers. Considering the dose-response relationships between smoking and various diseases, we can assume that pack-year history and health benefits might be similarly related (15). Therefore, this study aimed to investigate the potential effect of pack-year history on the health benefits conferred on former smokers, combined with the time that passed after those individuals quit smoking.

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The author has no conflict of interest to disclose.

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Methods

Study design

A cross-sectional design was used for the study.

Data collection

From February through July 2015, 1165 patients without any acute complaints and who had been admitted to the family medicine polyclinics of Ankara Ataturk Training and Research Hospital for a routine check-up were studied. Among them, 160 participants who were ex-smokers, older than 18, and willing to participate in the study were enrolled (Figure 1). The anthropometric parameters collected were height, weight, and waist circumference (at the umbilical level). The vital signs (systolic and diastolic blood pressure [DBP] and pulse) were measured with an automatic device (Omron M2). Blood samples were collected for laboratory analysis. Fasting plasma glucose (FPG), hemoglobin A1c (HbA1c), and blood lipids (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol [HDL-C], and triglycerides) were analyzed. The time that passed after quitting smoking (years) and the cumulative smoking exposure history(pack-years) were elicited with questions asking how many years the participant had smoked and how many cigarettes he or she had smoked per day. Thus, the pack-years parameter was calculated using the following formula: (number of cigarettes smoked per day/pack size [20]) x smoking duration (years). The metabolic syndrome status of the participants was calculated according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP-III) criteria. The diagnosis of metabolic syndrome requires the presence of 3 or more of the following: 1) abdominal obesity (waist circumference >102 cm in men and >88 cm in women); 2) a high triglyceride level (≥150 mg/dL [≥1.69 mmol/L]); 3) a low HDL cholesterol level (<40 mg/dL [<1.03 mmol/L] for men and <50 mg/dL [<1.29 mmol/L] for women); 4) high blood pressure (systolic ≥130 mm Hg or diastolic ≥85 mm Hg); and 5) a high FPG concentration (≥110 mg/dL)(16).

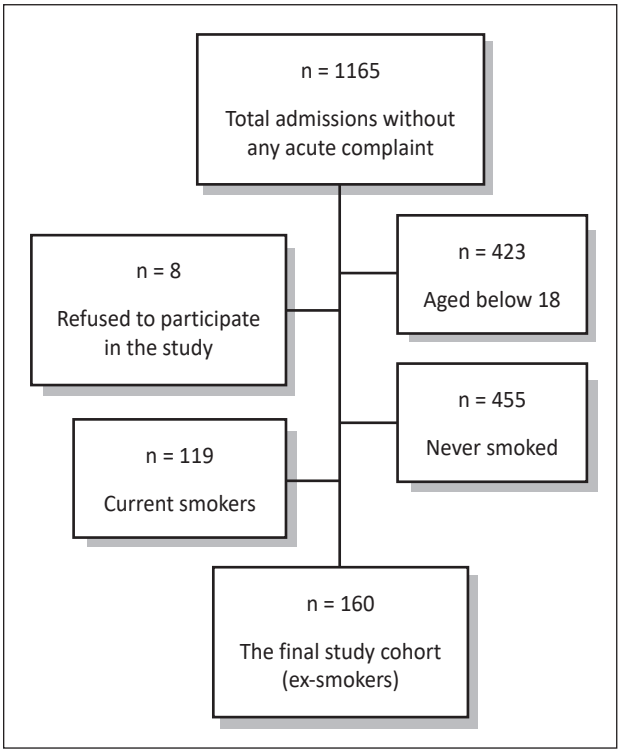

Figure 1. Flow diagram of the participants

The smoke-free ratio (SFR)

This novel index was generated by calculating the ratio of the time that had passed after quitting smoking (years) by the cumulative smoking exposure history (pack-years).

$$\text{SFR} = \frac{\textit{The time that had passed after smoking cessation (years)}}{\textit{The history of total exposure to smoking (pack-years)}}$$

Statistical analysis

The data obtained from the participants were transferred to IBM SPSS Statistics 20. The frequencies were obtained for descriptive data. The normal distribution of data was examined using the Shapiro–Wilk test. The demographic data were given in numbers and median, minimum, and maximum values. Spearman's rho correlation test was performed for correlation analysis between SFR and the laboratory parameters as well as the body measurements. The correlation analysis was performed separately for diabetic, non-diabetic, and healthy sub-groups. A Mann–Whitney *U* test was used to investigate the association between SFR and metabolic syndrome. A logarithmic transformation was done to obtain a normal distribution for the SFR. The chi-square test was used for comparing the categorical variables of metabolic syndrome and SFR binary grouping. All available analyses were grouped both by gender and by total. A P value less than .05 was accepted as the limit of significance.

Results

The study comprised 103 (64.4%) male and 57 (35.6%) female participants. Of them, 33 (20.6%) were healthy, while 127 (79.4%) had at least 1 chronic disease. The median amount of time that passed after smoking cessation was 10 years (IQR: 10), and the median cumulative smoking exposure history was 10 pack-years (IQR: 16). Further descriptive data are given in Table 1.

Of the participants, 52 (32.5%) had metabolic syndrome (31 men and 21 women) according to the ATP-III criteria. The Mann–Whitney *U* test revealed that the cohort with metabolic syndrome had significantly lower SFR scores (Z = -2.11; *P* = .035) (Figure 2).

In the correlation analysis between SFR and various laboratory, anthropometric, and vital parameters, SFR was negatively correlated with body mass index (BMI), DBP, and pulse in women with diabetes. In the healthy sub-group, FPG was negatively and HDL cholesterol positively correlated with the SFR (Table 2).

Table 1. The descriptive analysis of the participants

		Total (both genders)	Total (n = 160)		Non-diabetic (n = 125)		Diabetic (n = 35)		Healthy (n = 33)	
			Women (n = 57)	Men (n = 103)	Women (n = 46)	Men (n = 79)	Women (n = 11)	Men (n = 24)	Women (n = 10)	Men (n = 23)
Age (years)	Median	57	51	60	50	59	62	61	50	60
	Min.	21	21	23	21	23	49	39	21	29
	Max.	84	76	84	76	84	68	83	59	74
SFR	Median	0.75	1.00	0.52	1.00	0.65	0.59	0.36	0.70	1.00
	Min.	0.03	0.09	0.03	0.11	0.03	0.09	0.07	0.20	0.03
	Max.	15.00	10.00	15.00	10.00	10.00	5.00	15.00	1.11	8.33
HbA1c (%)	Median	5.7	5.6	5.8	5.5	5.7	6.1	7.2	5.5	5.7
	Min.	4.5	4.7	4.5	4.7	4.5	5.3	5.5	5.2	4.9
	Max.	12.7	6.9	12.7	6.4	6.6	6.9	12.7	6.4	6.2
Fasting Plasma Glucose (mg/dL)	Median	98	94	100	91	97	110	138	89	96
	Min.	76	77	76	77	76	85	91	79	81
	Max.	311	153	311	111	128	153	311	108	112
LDL-C (mg/dL)	Median	124	133	115	139	115	106	114	150	115
	Min.	54	54	55	54	55	84	56	54	76
	Max.	217	198	217	198	203	178	217	175	178
HDL-C (mg/dL)	Median	50	57	47	57	49	58	43	63	52
	Min.	18	31	18	37	18	31	31	46	39
	Max.	110	110	89	110	88	83	89	101	88
Total Cholesterol (mg/dL)	Median	203	213	199	215	198	193	210	228	187
	Min.	104	131	104	131	104	148	130	167	143
	Max.	324	301	324	301	323	266	324	243	248
TG (mg/dL)	Median	130	110	139	110	129	129	163	85	95
	Min.	36	36	42	36	42	70	50	80	42
	Max.	512	465	512	465	470	205	512	110	131
Body Mass Index (kg/m2)	Median	27.6	26.8	27.8	25.7	27.7	34.1	28.3	26	27
	Min.	20.0	20.0	21.9	20.0	21.9	20.8	23.4	20	22.1
	Max.	40.0	40.0	39.1	34.3	39.1	40.0	38.6	33.3	38.1
SBP (mmHg)	Median	120	110	120	110	120	110	110	110	120
	Min.	90	90	90	90	90	110	100	90	100
	Max.	140	135	140	130	140	135	140	120	130
DBP (mmHg)	Median	80	70	80	70	80	70	80	70	80
	Min.	60	60	60	60	60	70	60	60	70
	Max.	90	90	90	90	90	85	90	85	80
Pulse (bpm)	Median	78	77	80	78	80	75	77	78	77
	Min.	60	60	60	60	68	70	60	70	68
	Max.	88	88	85	88	85	88	85	80	84

DBP: diastolic blood pressure; HbA1c: hemoglobin A1c; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; SBP: systolic blood pressure; SFR: smoke-free ratio; TG: triglyceride

Based on the distribution of SFR scores in the study population, a cut-off value of 0.20 was calculated. For this, a logarithmic transformation of the SFR scores was performed to obtain a normal distribution, and [*mean value – standard deviation*] formula is preferred. Since there is no upper limit for the SFR, theoretically, scores of 0.20 and lower were accepted as being low SFRs. The odds ratios of metabolic syndrome and its components in the group with low SFR scores are shown in Table 3.

Discussion

Of those studies focusing on the extent to which former smokers recover from the damage engendered by smoking after quitting, almost none consider the cumulative doses that these former smokers were exposed to, although it has been shown that with smoking cessation, the risks of disease and mortality decrease over time (14,17). The most important feature of this study is the fact that it is probably the first to explore this subject.

Theoretically, higher SFR scores may simply be predictors of the degree to which smoking-related toxins have been purged from the bodies of the former smokers. One of the most exciting results of this study is the association of the SFR with metabolic syndrome. In the literature, numerous studies emphasize the association of smoking with metabolic syndrome (18,19). Furthermore, risk reduction after quitting smoking has been usually linked to the time that passes after quitting, ignoring the pack-years history (20–23). This study revealed significantly higher SFR scores in participants without metabolic syndrome. Thus, we can assume that the cumulative smoking exposure history of former smokers might also be associated with the subsequent risk of metabolic syndrome.

Another remarkable result is the correlation between SFR scores and various laboratory parameters and anthropomorphic

measurements. The strong negative correlation of the SFR with BMI, DBP, and pulse measurements was seen only among diabetic women. This finding is interesting, although it was not observed in a similar male group, which may have been due to the limited number of patients in the diabetic sub-group. The FPG and HbA1c parameters were also negatively associated with the SFR scores, although the correlation was weak. However, in the binary grouping of the SFR scores, FPG was remarkably high in those participants with low SFRs. There is some critical evidence that smoking cessation may be associated with a short-term risk increase of type 2 diabetes, despite the net beneficial effect on reducing all-cause mortality (24). This phenomenon may be a confounder of the effect of the SFR on glucose metabolism. The actual clinical significance of this association may be clarified with studies conducted on larger populations.

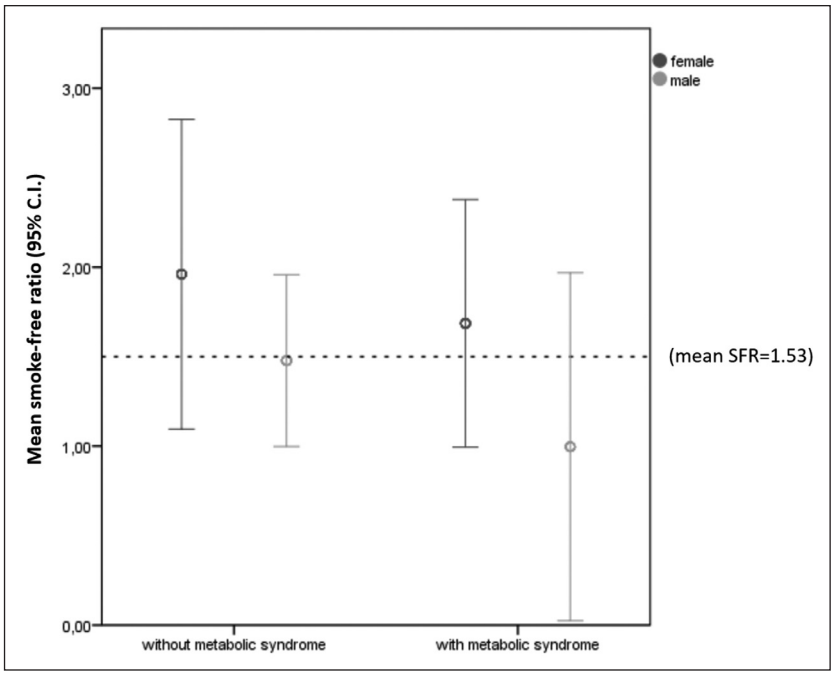

Figure 2. The distribution of SFR scores in terms of metabolic syndrome regarding sex.

This study also points out some riveting associations between the SFR and various lipid parameters, such as HDL-C and triglyceride levels. A weak positive correlation of the SFR with HDL-C in the total cohort was also observed in the general male group and the non-diabetic male sub-group, whereas the correlation was strong in the healthy female sub-group. A study conducted on a large cohort consisting of the Framingham offspring found that smoking cessation was associated with a lower risk of cardiovascular disease in non-diabetic participants, despite subsequent weight gain (25). Previous studies have also shown that smoking decreases HDL-C, which is known to be a protective factor for cardiovascular disease (26). The average values of HDL-C are well known to be higher in women. Some confounding factors in women with chronic diseases may be causing a lack of association with SFR scores, as these factors may have an inhibiting effect against HDL-C increase. This hypothesis seems to be sustained by the fact that the positive correlation of the SFR with HDL-C was high in the healthy female sub-group, notwithstanding the fact that further study is required.

Additionally, in almost all the sub-groups in which HDL-C increased with the SFR (except in the healthy women sub-group), the triglyceride levels were also negatively correlated with the SFR scores. High triglyceride levels were found remarkably frequently in the low SFR group, too. Thus, it can be inferred that the lipid parameters of HDL-C and triglycerides, which are among the parameters of metabolic syndrome, are directly associated with the SFR. The lack of a significant association between the SFR and total cholesterol can be explained by the counter-directional change of HDL-C and triglycerides, which has a balancing effect on total cholesterol.

In conclusion, the SFR may be a promising indicator of several parameters associated with cardiovascular risk reduction in former smokers, for it may be a more accurate predictor than just smoke-free duration. Despite its limited number of participants, this study has uncovered several significant relationships that are causing excitement and that promise to be significant avenues of extensive future research. Besides its predictive function, the SFR may be a valuable tool for motivating the patients who need support in the smoking-cessation process. Considering the high relapse rates after smoking cessation (even after 10 years or more), the motivation for increasing SFR scores may help ex-smokers maintain long-term abstinence.

Limitations

This study was conducted on a limited cohort. Thus, the findings cannot be generalized to the greater community. Furthermore, the data used to calculate the SFRs were based on the participants' declarations. It was shown that the calculation of pack-year history may vary between prospective and retrospective methods (27). It is also possible that the SFR calculations, based as they are on self-reported data, may suffer from recall bias.

Another limitation of this study is that the SFR was investigated in terms of only a limited number of laboratory and anthropometric parameters, most of which are usually considered to be indicators of cardiovascular health. The clinical usability of the SFR may become more evident in future studies that specifically focus on conditions such as cancer and chronic obstructive pulmonary disease, which are directly associated with smoking.

Table 2. The correlation analysis of smoke-free ratio with various parameters

		Total (both genders)	Smoke-Free Ratio							
			Total (n = 160)		Non-diabetic (n = 125)		Diabetic (n = 35)		Healthy (n = 33)	
			Women (n = 57)	Men (n = 103)	Women (n = 46)	Men (n = 24)	Women (n = 79)	Men (n = 11)	Women (n = 10)	Men (n = 23)
HbA1c (%)	Rho	-0.203	-0.115	-0.165	0.019	-0.196	0.101	0.010	-0.143	-0.231
	P	.023	.480	.131	.923	.128	.768	.964	.760	.327
	n	125	40	85	29	62	11	23	7	20
Fasting Plasma Glucose (mg/dL)	Rho	-0.245	-0.221	-0.207	-0.208	-0.198	-0.074	0.153	-0.748	-0.351
	P	.002	.102	.036	.170	.080	.829	.476	.013	.110
	n	159	56	103	45	79	11	24	10	22
LDL-C (mg/dL)	Rho	-0.077	-0.148	-0.067	-0.236	-0.048	-0.237	-0.223	-0.500	-0.276
	P	.345	.301	.506	.143	.681	.482	.295	.253	.213
	n	152	51	101	40	77	11	24	7	22
HDL-C (mg/dL)	Rho	0.187	-0.142	0.254	-0.100	0.247	-0.256	0.258	0.857	0.091
	P	.021	.320	.010	.540	.031	.448	.223	.007	.688
	n	152	51	101	40	77	11	24	8	22
Total Cholesterol (mg/dL)	Rho	-0.073	-0.141	-0.059	-0.184	-0.069	-0.215	-0.068	-0.464	-0.183
	P	.369	.323	.557	.255	.551	.526	.753	.294	.416
	n	152	51	101	40	77	11	24	7	22
TG (mg/dL)	Rho	-0.199	0.079	-0.254	0.092	-0.311	0.237	-0.026	-0.321	-0.203
	P	.014	.583	.011	.573	.006	.482	.903	.482	.364
	n	152	51	100	40	76	11	24	7	22
Body Mass Index (kg/m^2)	Rho	-0.173	-0.190	-0.095	-0.010	-0.075	-0.607	-0.188	-0.017	-0.177
	P	.034	.161	.359	.948	.526	.048	.389	.966	.442
	n	152	56	96	45	73	11	23	9	21
SBP (mmHG)	Rho	0.046	-0.100	0.148	0.029	0.186	-0.511	-0.102	-0.426	0.094
	P	.569	.471	.139	.852	.105	.108	.634	.253	.686
	n	155	54	101	43	77	11	24	9	21
DBP (mmHg)	Rho	-0.081	-0.212	0.021	-0.092	0.031	-0.728	0.017	0.248	-0.144
	P	.315	.124	.835	.557	.788	.011	.939	.520	.534
	n	155	54	101	43	77	11	24	9	21
Pulse (bpm)	Rho	-0.130	-0.288	-0.028	-0.203	0.033	-0.692	-0.325	0.266	-0.146
	P	.108	.035	.784	.192	.772	.018	.121	.489	.527
	n	155	54	101	43	77	11	24	9	21

DBP: diastolic blood pressure; HbA1c: hemoglobin A1c; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; OR: odds ratio Rho: Spearman's rho; SBP: systolic blood pressure; TG: triglyceride

Table 3. Odds ratios for metabolic syndrome and its components in terms of low smoke-free ratio scores (≤0.20)

	Chi-square	*P*	OR (95% CI)
Metabolic syndrome	8.981	.003	3.58 (1.51–8.49)
High blood pressure	0.295	.587	0.78 (0.33–1.88)
High triglycerides	6.388	.011	2.96 (1.25–7.04)
Low HDL-C	3.414	.065	2.24 (0.94–5.37)
High fasting plasma glucose	13.252	<.001	5.85 (2.08–16.45)
Abdominal obesity	1.286	.257	1.63 (0.70–3.78)

HDL-C: high-density lipoprotein cholesterol; OR: odds ratio

Finally, there may be some bias originating from ignoring the effect of age. However, this bias is also found in the pack-years concept, which is still a widely accepted tool for predicting the cumulative harm of smoking.

Key points

- The SFR (smoke-free ratio) consists of the number of smoke-free years divided by the number of pack-years history.
- The SFR may be a new tool for predicting cardiovascular and metabolic risk reduction in former smokers.
- Higher SFR scores were associated with lower BMIs, DBPs, pulse rates, and metabolic syndrome rates in some sub-groups.
- The SFR can motivate those who quit smoking not to start again.

Resumen

Antecedentes: la asociación del tabaquismo con muchas enfermedades es bien conocida, así como los beneficios de dejar de fumar. Si bien se mencionan los beneficios, siempre se destaca el tiempo transcurrido después de dejar de fumar. Sin embargo, se ignoró el historial de dosis acumuladas de exposición al tabaco en exfumadores. Este estudio tiene como objetivo investigar el posible efecto del historial de paquetes-año sobre algunos parámetros de salud cardiovascular. Métodos: Se realizó un estudio transversal en 160 exfumadores participantes. Se describió un índice novedoso y se denominó como la proporción libre de fumar (SFR, por sus siglas en inglés), que se refería

a la duración de los años sin fumar dividida por el historial de paquetes-años. Se investigaron las asociaciones entre SFR y algunos parámetros de laboratorio, así como mediciones antropométricas y vitales. Resultados: SFR se correlacionó negativamente con el IMC, la PAD y el pulso en mujeres con diabetes. En el subgrupo sano, la glucosa plasmática en ayunas fue negativa y el colesterol HDL se correlacionó positivamente con SFR. La prueba U de Mann-Whitney reveló que la cohorte con síndrome metabólico tenía puntuaciones SFR significativamente más bajas (Z = -2,11; *P* = 0,035). En la agrupación binaria, los participantes con puntuaciones de SFR bajas tenían tasas de síndrome metabólico más altas. Discusión: Este estudio reveló algunos resultados impresionantes sobre el SFR, que se propone como un concepto novedoso para estimar la reducción del riesgo metabólico y cardiovascular en exfumadores. Sin embargo, la importancia clínica real de esta entidad aún no está clara.

Acknowledgments

I am grateful to all my colleagues in my department for their valuable efforts in collecting data for this study. I want to thank the volunteer participants for kindly agreeing to participate in my study. Ethical approval was given by the local ethics committee (approval number 29.01.2015/38). The informed consent forms were collected from participants.

References

1. Hammond EC, Horn D. The relationship between human smoking habits and death rates: a follow-up study of 187,766 men. J Am Med Assoc. 1954;155(15):1316-1328. doi:10.1001/jama.1954.03690330020006
2. Franklin W, Lowell FC, Michelson AL, Schiller IW. Chronic obstructive pulmonary emphysema; a disease of smokers. Ann Intern Med. 1956;45(2):268-274. doi:10.7326/0003-4819-45-2-268
3. Freund KM, Belanger AJ, D'Agostino RB, Kannel WB. The health risks of smoking. The Framingham Study: 34 years of follow-up. Ann Epidemiol. 1993;3(4):417-424. doi:10.1016/1047-2797(93)90070-k
4. Bhatt SP, Kim YI, Harrington KF, et al. Smoking duration alone provides stronger risk estimates of chronic obstructive pulmonary disease than pack-years. Thorax. 2018;73(5):414-421. doi:10.1136/thoraxjnl-2017-210722
5. Whisnant JP, Homer D, Ingall TJ, Baker HL Jr, O'Fallon WM, Wievers DO. Duration of cigarette smoking is the strongest predictor of severe extracranial carotid artery atherosclerosis. Stroke. 1990;21(5):707-714. doi:10.1161/01.str.21.5.707
6. Pirie K, Peto R, Reeves GK, Green J, Beral V; Million Women Study Collaborators. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. Lancet. 2013;381(9861):133-141. doi:10.1016/S0140-6736(12)61720-6
7. Dempsey RJ, Moore RW. Amount of smoking independently predicts carotid artery atherosclerosis severity. Stroke. 1992;23(5):693-696. doi:10.1161/01.str.23.5.693
8. Liaw KM, Chen CJ. Mortality attributable to cigarette smoking in Taiwan: a 12-year follow-up study. Tob Control. 1998;7(2):141-148. doi:10.1136/tc.7.2.141
9. Wood DM, Mould MG, Ong SB, Baker EH. "Pack year" smoking histories: what about patients who use loose tobacco?. Tob Control. 2005;14(2):141-142. doi:10.1136/tc.2004.009977
10. Peto J. That the effects of smoking should be measured in pack-years: misconceptions 4. Br J Cancer. 2012;107(3):406-407. doi:10.1038/bjc.2012.97
11. Armadans-Gil L, Vaqué-Rafart J, Rosselló J, Olona M, Alseda M. Cigarette smoking and male lung cancer risk with special regard to type of tobacco. Int J Epidemiol. 1999;28(4):614-619. doi:10.1093/ije/28.4.614
12. Jaspers NEM, Blaha MJ, Matsushita K, et al. Prediction of individualized lifetime benefit from cholesterol lowering, blood pressure lowering, antithrombotic therapy, and smoking cessation in apparently healthy people [published correction appears in Eur Heart J. 2020 Dec 21;41(48):4579]. Eur Heart J. 2020;41(11):1190-1199. doi:10.1093/eurheartj/ehz239
13. Godtfredsen NS, Prescott E. Benefits of smoking cessation with focus on cardiovascular and respiratory comorbidities. Clin Respir J. 2011;5(4):187-194. doi:10.1111/j.1752-699X.2011.00262.x
14. Taylor DH Jr, Hasselblad V, Henley SJ, Thun MJ, Sloan FA. Benefits of smoking cessation for longevity [published correction appears in Am J Public Health 2002 Sep;92(9):1389]. Am J Public Health. 2002;92(6):990-996. doi:10.2105/ajph.92.6.990
15. Law MR, Morris JK, Watt HC, Wald NJ. The dose-response relationship between cigarette consumption, biochemical markers and risk of lung cancer. Br J Cancer. 1997;75(11):1690-1693. doi:10.1038/bjc.1997.287
16. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA. 2001;285(19):2486-2497. doi:10.1001/jama.285.19.2486
17. Samet JM. The health benefits of smoking cessation. Med Clin North Am. 1992;76(2):399-414. doi:10.1016/s0025-7125(16)30359-5
18. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. Arch Intern Med. 2003;163(4):427-436. doi:10.1001/archinte.163.4.427
19. Oh SW, Yoon YS, Lee ES, et al. Association between cigarette smoking and metabolic syndrome: the Korea National Health and Nutrition Examination Survey. Diabetes Care. 2005;28(8):2064-2066. doi:10.2337/diacare.28.8.2064
20. Novello AC. Surgeon General's report on the health benefits of smoking cessation. Public Health Rep. 1990;105(6):545-548.
21. World Health Organization. Tobacco: Health benefits of smoking cessation. February 25, 2020. Accessed April 15, 2020. https://www.who.int/tobacco/quitting/benefits/en/
22. U.S. Department of Health and Human Services. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health;2014.
23. U.S. Department of Health and Human Services. Smoking Cessation. A Report of the Surgeon General. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health;2020.
24. Hu Y, Zong G, Liu G, et al. Smoking Cessation, Weight Change, Type 2 Diabetes, and Mortality. N Engl J Med. 2018;379(7):623-632. doi:10.1056/NEJMoa1803626
25. Clair C, Rigotti NA, Porneala B, et al. Association of smoking cessation and weight change with cardiovascular disease among adults with and without diabetes. JAMA. 2013;309(10):1014-1021. doi:10.1001/jama.2013.164426
26. Kim SK, Kim HC, Shim JS, Kim DJ. Effects of cigarette smoking on blood lipids in Korean men: Cardiovascular and Metabolic Diseases Etiology Research Center cohort. Korean J Intern Med. 2020;35(2):369-382. doi:10.3904/kjim.2019.133
27. Bernaards CM, Twisk JW, Snel J, Van Mechelen W, Kemper HC. Is calculating pack-years retrospectively a valid method to estimate lifetime tobacco smoking? A comparison between prospectively calculated pack-years and retrospectively calculated pack-years. Addiction. 2001;96(11):1653-1661. doi:10.1046/j.1360-0443.2001.9611165311.x