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Is the ongoing obesity epidemic partly explained by concurrent decline in cigarette smoking? Insights from a longitudinal population study. The Tromsø Study 1994–2016

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ABSTRACT

The increase of obesity coincides with a substantial decrease in cigarette smoking. We assessed post-cessation weight change and its contribution to the obesity epidemic in a general population in Norway. A total of 14,453 participants (52.6% women), aged 25–54 years in 1994, who attended at least two of four surveys in the Tromsø Study between 1994 and 2016, were included in the analysis. Hereof 77% participated in both the first and the last survey.

Temporal trends in mean body mass index (BMI), prevalence of obesity (BMI ≥ 30 kg/m^2) and daily smoking were estimated with generalized estimation equations. We assessed BMI change by smoking status (ex-smoker, quitter, never smoker, daily smoker), and also under a scenario where none quit smoking.

In total, the prevalence of daily smoking was reduced over the 21 years between Tromsø 4 (1994–1995) and Tromsø 7 (2015–2016) by 22 percentage points. Prevalence of obesity increased from 5 – 12% in 1994–1995 to 21–26% in 2015–2016, where obesity in the youngest (age 25–44 in 1994) increased more than in the oldest ($p < 0.0001$). Those who quit smoking had a larger BMI gain compared to the other three smoking subgroups over the 21 years ($p < 0.0001$). The scenario where none quit smoking would imply a 13% reduction in BMI gain in the population, though substantial age-related differences were noted.

We conclude that smoking cessation contributed to the increase in obesity in the population, but was probably not the most important factor. Public health interventions should continue to target smoking cessation, and also target obesity prevention.

1. Introduction

Tobacco smoking is the cause of millions of deaths and cases of chronic diseases worldwide every year (World Health Organization, 2020). The Global Burden of Disease project estimated that 7.1 million deaths and 182 million disability-adjusted life years (DALYs) could be attributed to this single risk factor in 2017, with only high systolic blood pressure causing more life years lost (Stanaway et al., 2018).

Obesity is a risk factor for diabetes, musculoskeletal disorders, some cancers, cardiovascular and kidney diseases (NCD Risk Factor Collaboration, 2016). There is some evidence that obesity also increases the risk for hospitalization and death from the infectious diseases H1N1 (swine influenza) and COVID-19 (Dietz and Santos-Burgoa, 2020). In a recent Mendelian randomization study, tobacco smoking and elevated body mass index (BMI) were found to be associated with an increased risk of severe COVID-19 (Ponsford et al., 2020).

Progress has been made to reduce the number of smokers, with a decline in every region of the world (World Health Organization, 2019). At the same time, there has been an increase in the prevalence of obesity. The fact that the prevalence of these two risk factors have moved in opposite directions motivated the research question "Is the ongoing obesity epidemic partly explained by concurrent decline in cigarette smoking?"

Several longitudinal population studies, with a follow-up time of 4 to

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11 years, have found that quitting smoking is associated with an excessive weight gain (Clair et al., 2013; Janzon et al., 2004; Lycett et al., 2011; Sneve and Jorde, 2008; Tamura et al., 2010; Travier et al., 2012; Williamson et al., 1991). We assessed this hypothesis over 21 years of follow-up. A time scale of two decades has previously been used in only one study (Jain et al., 2016), though the focus of Jain et al. was on long-term post-cessation weight, whereas we also aim to quantify the impact of smoking cessation on the obesity epidemic. Furthermore, we will also assess the short-term (a few years) post-cessation weight change, using data from a large longitudinal population study.

2. Materials and methods

2.1. Study population

The Tromsø Study is an ongoing population based study, established in 1974, with participants recruited from Tromsø, the largest populated municipality in Northern Norway, consisting of both urban and rural living areas.

We used data from the fourth to seventh survey: Tromsø 4 was conducted in 1994–1995, Tromsø 5 in 2001, Tromsø 6 in 2007–2008 and Tromsø 7 in 2015–2016. A total of 36,929 men and women participated in at least one of these four surveys, 19,965 in at least two. The attendance ranged from 65% to 79%. All inhabitants above a certain age were invited to Tromsø 4 (aged 25 and above) and Tromsø 7 (aged 40 and above). Representative samples were invited to Tromsø 5 and 6 (Eggen et al., 2013; Jacobsen et al., 2012).

In the present analyses we included participants born in 1940–1969, aged 25–54 years in 1994. We included subjects that attended at least two surveys ($n = 14{,}789$), excluding those who withdrew their consent to research ($n = 19$) and those with missing data on smoking ($n = 112$), BMI ($n = 202$) or both ($n = 3$), resulting in a final sample of 14,453 participants (52.6% women). Herein 52% participated in two, 37% in three and 11% in all four surveys, 77% participated in both Tromsø 4 and Tromsø 7, 20% in Tromsø 4 and 5, 14% in Tromsø 5 and 6 and 50% in Tromsø 6 and 7. A lasagna plot (Jones et al., 2014) of the joint distribution of attendance is found in Supplementary Fig. 1. Supplementary Table 1 shows the sample size by birth cohort and sex.

The Regional Committee of Medical and Health Research Ethics (REK Nord 2014/940) and the Norwegian Data Protection Authority approved the Tromsø Study, and the participants gave written informed consent.

2.2. Anthropometric measurements

Height and weight were measured with light clothing and no footwear, to the nearest 1 cm and 0.5 kg in Tromsø 4, and to the nearest 0.1 cm and 0.1 kg in Tromsø 5–7, respectively. All measurements with remarks, e.g., pregnancy, scoliosis or measured with shoes, were set to missing. BMI was calculated as weight/height^2 (kg/m^2), and obesity defined as BMI ≥ 30 kg/m^2.

2.3. Smoking status

In Tromsø 5–7 the question "Do you/did you smoke daily?" was to be answered by one of the three alternatives; "Yes, now", "Yes, previously", "Never". For Tromsø 4 we constructed a binary variable about daily smoking (yes/no) from the three questions; "Do you smoke: Cigarettes daily? Cigars/cigarillos daily? A pipe daily?". If none of these three questions were answered, daily smoking (in Tromsø 4) was set to missing. If at least one of the answers was yes, then daily smoking was set to yes, otherwise no.

Smoking status at Tromsø 4 and Tromsø 7 were combined into one variable with four categories: Ex-smoker (No - Yes, previously), daily smoker (Yes - Yes, now), never smoker (No - Never) and quitter (Yes - Yes, previously). We excluded participants in the two remaining categories, those who started smoking or relapsed (No - Yes, now) and those who identified themselves as a daily smoker in Tromsø 4 and never smoker in Tromsø 7. In the analysis of two consecutive surveys, smoking status was defined similarly.

2.4. Statistical methods

In all analyses, sex and ten-year birth cohorts were used as categorical variables. Generalized estimation equations (GEE), with an exchangeable co-variance matrix, were used for estimation of mean BMI. GEE and logistic regression were used for estimation of prevalence of obesity and daily smoking. Robust standard deviation and asymptotic normality of the estimators were used to derive confidence intervals. The probability density functions of BMI were estimated by kernel density estimation, using Gaussian kernels with Silverman's rule of thumb bandwidth selectors.

To compute BMI change under a scenario where no one quit smoking, we first note that the mean BMI change μ can be written as

$$\mu = \sum p_i \, \mu_i, \tag{1}$$

where p_i and μ_i is the proportion and mean BMI change, respectively, in smoking status group i. The formula (1) follows from the law of double expectation. The proportion of quitters, say p_1, is distributed to one or more of the other groups ($i \neq 1$) and then set equal to zero, i.e., $p_1 = 0$ and $\sum p_i = 1$. We chose to distribute the proportion of quitters to smokers. We performed a sensitive analysis with educational level and body mass index at baseline included as confounders (Supplementary Tables 2 and 3) and found that the adjusted estimates were within the confidence intervals of the corresponding unadjusted estimates.

All of the analyses were conducted in R (R Core Team, 2020), and most figures were made with the R-package ggplot2 (Wickham, 2016). The p-values are for two-sided tests.

3. Results

3.1. Daily smoking

In total, the prevalence of daily smoking was reduced over the 21 years between Tromsø 4 and Tromsø 7 by 22 percentage points. This figure ranged from 21% to 24% in the six groups defined by birth cohorts and gender (Table 1). We observed a larger decline from Tromsø 5 (year 2001) and onwards, which can also be seen in Supplementary Table 4 and Supplementary Fig. 2.

3.2. Mean BMI and obesity

Prevalence of obesity increased from the range of 5% -12% in Tromsø 4 to the range of 21% -26% in Tromsø 7 (Table 1, Supplementary Fig. 3). In Tromsø 4, the oldest birth cohort (1940–1949) had a higher prevalence of obesity compared to those born 1950–1969. However, the oldest had a smaller change over the 21 years ($p < 0.0001$), resulting in only minor differences in BMI between the birth cohorts in Tromsø 7.

Mean BMI increased at each consecutive survey for all groups and surveys ($p < 0.0001$), except for men born 1940–1949 in Tromsø 5–6 where the increase of 0.1 kg/m^2 was not statistically significant ($p = 0.125$) (Table 1). The magnitude of change in mean BMI over 21 years (ΔBMI) decreased with age: those born 1940–1949 had smaller ΔBMI than participants born 1950–1959 ($p < 0.0001$ for both genders) and those born 1950–1959 had smaller ΔBMI than participants born 1960–1969 ($p < 0.0001$ for both genders).

The increase in obesity and mean BMI between Tromsø 4 (1994–1995) and Tromsø 7 (2015–2016) can also be seen from the distributions in Fig. 1.

Table 1
Prevalence of daily smoking, obesity and mean body mass index according to survey and birth cohort. The Tromsø Study 1994–2016.

Sex	Year born	Survey years				Δ21 years[a]
		Tromsø 4	Tromsø 5	Tromsø 6	Tromsø 7	
		1994–1995	2001	2007–2008	2015–2016	
		Daily smoking (%)				
Women	1940–1949	36	30	21	14	−22
	1950–1959	41	36	26	18	−24
	1960–1969	38	34	25	18	−20
Men	1940–1949	36	32	19	12	−24
	1950–1959	36	34	20	15	−21
	1960–1969	37	34	21	15	−22
		Obesity (%)				
Women	1940–1949	12	21	22	25	14
	1950–1959	6	10	16	21	15
	1960–1969	5	12	17	24	18
Men	1940–1949	12	24	23	25	14
	1950–1959	8	15	19	25	18
	1960–1969	6	14	20	26	19
		Mean body mass index (kg/m^2)				
Women	1940–1949	25.1	26.6	27.0	27.4	2.3
	1950–1959	23.8	25.3	26.0	26.8	3.0
	1960–1969	23.3	25.1	25.9	26.9	3.6
Men	1940–1949	26.2	27.5	27.6	27.8	1.6
	1950–1959	25.4	26.6	27.3	27.8	2.4
	1960–1969	24.9	26.4	27.2	28.0	3.2

[a] Tromsø 7–Tromsø 4.

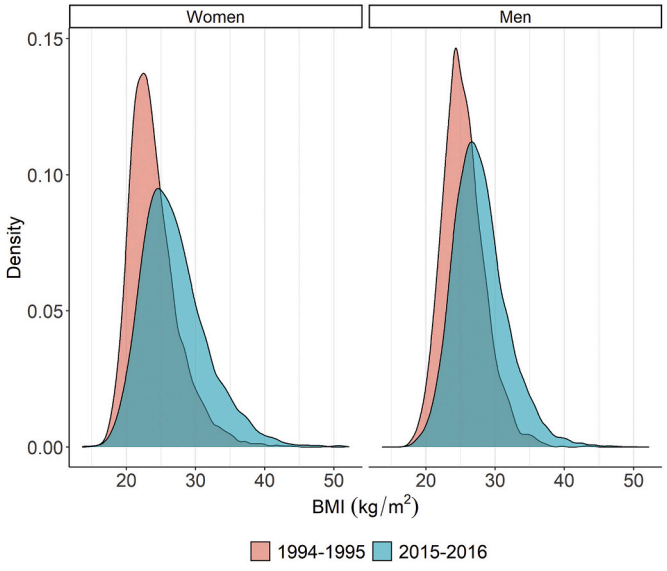

Fig. 1. Distributions of body mass index (BMI) among participants, born 1940–1969, attending both Tromsø 4 (1994–1995) and Tromsø 7 (2015–2016). Survey years indicated in legend. The Tromsø Study.

3.3. Change in BMI by smoking status and scenario

The change in mean BMI over 21 years by smoking status is shown in Fig. 2. In each group (gender and birth cohort) quitters had a larger increase in mean BMI compared to the other three smoking subgroups ($p < 0.0001$). In particular, compared to never-smokers, the excessive BMI gain ranged from 1.3 to 1.9 kg/m^2 over 21 years (Supplementary Table 5).

Quitters between consecutive surveys also had an excessive BMI gain (Supplementary Fig. 4 and Supplementary Table 6). Table 2 shows the difference in BMI gain between never-smokers and quitters, over 21 years and between consecutive surveys.

The mean BMI gain in the population between Tromsø 4 and Tromsø 7 was 2.7 kg/m^2. The scenario where none quit smoking would imply a 13% reduction, to a hypothetical BMI gain of 2.3 kg/m^2. We observed differences in the attributable fractions between the birth cohorts, e.g., 6% and 7% in the birth cohort 1960–1969 and 24% and 28% in the birth cohort 1940–1949, in women and men, respectively (Table 3).

4. Discussion

In this longitudinal population study with a follow-up time of 21 years, we found that the prevalence of daily smoking was reduced by 22 percentage points in the study population while the prevalence of obesity increased substantially. The youngest had the largest increase in the prevalence of obesity with 4–5 times more obese people in 2015–2016 than in 1994–1995. Furthermore, those who quit smoking between two surveys had a significant excessive BMI gain compared to the other three smoking subgroups. Our results indicate that the major part of the excessive gain occurred during the first six to eight years after quitting, and also that quitting smoking contributed to the increased BMI at the population level, though the attributable fraction was relative low (13% in total). However, substantial age-related differences were noted and in the older part of the study population, approximately 25% of the increase in BMI could be attributed to smoking cessation.

This study confirms findings from the Framingham Heart Study (Jain et al. (2016), of an excessive body weight gain over two decades following smoking cessation. Jain et al. only compared quitters to smokers, but smokers were further split into subgroups based on number of cigarettes per day. In contrast, we treated smokers as one group, but also included ex-smokers and never-smokers as there is no reason to a priori assume that they did not contribute to the obesity epidemic. Indeed, we found that all smoking subgroups in our study (including ex-smokers and never-smokers) had a significant BMI gain, which point towards other factors to explain the obesity epidemic in addition to smoking cessation. The fact that the youngest had the largest increase in BMI is in line with findings from other population-based cohort studies (Løvsletten et al., 2020). Our study on smoking related weight gain adds to previous findings on longitudinal changes in BMI in the Tromsø Study

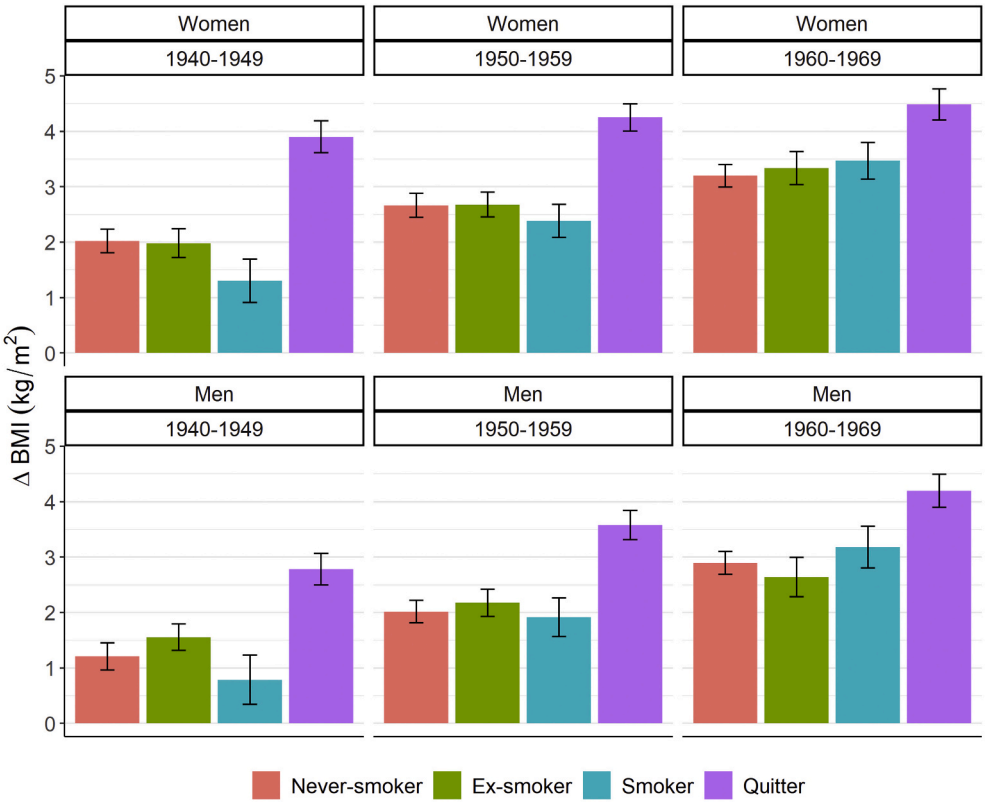

Fig. 2. Changes in body mass index over 21 years, by smoking status, sex, and year born, with 95% confidence intervals. The Tromsø Study 1994–2016.

Table 2
Excessive body mass index gain (kg/m^2) for those who quit smoking, compared to never-smokers. 95% confidence intervals in parentheses. The Tromsø Study 1994–2016.

Sex	Year born	Tromsø 4–5	Tromsø 5–6	Tromsø 6–7	Tromsø 4–7
		$\Delta t = 7$[a]	$\Delta t = 6$[a]	$\Delta t = 8$[a]	$\Delta t = 21$[a]
Women	1940–1949	1.2 (0.7, 1.6)	1.9 (1.5, 2.4)	2.0 (1.6, 2.4)	1.9 (1.5, 2.2)
	1950–1959	1.6 (0.8, 2.4)	1.2 (0.4, 2.0)	1.6 (1.2, 2.1)	1.6 (1.3, 1.9)
	1960–1969	0.8 (0.0, 1.5)	0.4 (−0.5, 1.3)	1.8 (1.4, 2.2)	1.3 (0.9, 1.6)
Men	1940–1949	0.5 (−0.1, 1.2)	1.1 (0.5, 1.7)	1.7 (1.3, 2.1)	1.6 (1.2, 1.9)
	1950–1959	1.2 (0.4, 2.0)	0.6 (−0.3, 1.5)	1.6 (1.1, 2.1)	1.6 (1.2, 1.9)
	1960–1969	1.2 (0.3, 2.1)	1.6 (0.6, 2.6)	0.7 (0.3, 1.1)	1.3 (0.9, 1.7)

[a] Years between surveys.

(Jacobsen and Aars, 2015, and Løvsletten et al., 2020). Changes in BMI by smoking status has been published for a shorter time interval between Tromsø 4 and Tromsø 5 (Sneve and Jorde, 2008).

The small attributable fraction of smoking cessation on BMI gain in the youngest age group can be understood from i) Eq. (1) together with the fact that quitters are only a minority of the population and ii) the other smoking subgroups also gained considerable weight. The proportions of quitters were similar across age groups. However, a greater relative difference between quitters and the other subgroups, implied a higher attributable fraction in the oldest birth cohorts, which may partly be explained by survival bias. Our reasoning is that smokers with high BMI have an increased mortality, which is followed by a lower BMI in the smoking group and, in turn, a higher attributable fraction.

Table 3
Change in body mass index (BMI, kg/m^2) over 21 years; observed and in a scenario where none quit smoking. 95% confidence intervals in parentheses. The Tromsø Study 1994–2016.

Sex	Year born	ΔBMI	ΔBMI scenario	% reduction[a]
Women	1940–1949	2.3 (2.1, 2.6)	1.8 (1.5, 2.1)	24 (21, 28)
	1950–1959	3.0 (2.8, 3.2)	2.6 (2.3, 2.8)	15 (14, 17)
	1960–1969	3.6 (3.3, 3.8)	3.3 (3.1, 3.6)	6 (6, 7)
Men	1940–1949	1.7 (1.4, 1.9)	1.2 (0.9, 1.5)	28 (22, 37)
	1950–1959	2.4 (2.2, 2.6)	2.0 (1.8, 2.3)	16 (14, 18)
	1960–1969	3.2 (2.9, 3.5)	3.0 (2.7, 3.3)	7 (6, 9)

[a] $\frac{\Delta\text{BMI} - \Delta\text{BMI scenario}}{\Delta\text{BMI}} \times 100$

The BMI gain associated with smoking cessation may be limited to the first years following abstinence. Our results (Table 2), though limited to the sampling scale of 6–8 years, are in accordance with this hypothesis. The group of ex-smokers had BMI gain similar to never-smokers. Ex-smokers reported to be former daily smokers, but did not smoke at any of the two surveys, whereas quitters were daily smokers in the first survey and had quit in the second survey. This could be interpreted as ex-smokers quit daily smoking prior to the first survey which further strengthens the evidence that the excessive BMI gain following smoking cessation took place the first years after abstinence (Travier et al., 2012). The exact mechanism through which nicotine regulates weight is still not clearly understood. Post-cessation weight gain, as found in this and other population-based cohort studies, are also

observed in rodent models where both rats and mice experience weight gain during nicotine withdrawal (Calarco and Picciotto, 2020). Previous studies have found an indirect effect through increased energy intake following cessation, and possibly also physical activity change, as well as a direct effect of nicotine-related metabolic changes (Bush et al., 2016). In this study we do not have access to data on physical activity and energy intake, so our results are limited to the total effect of smoking cessation on BMI change.

It has been suggested that weight concerns should be taken into account in tobacco dependence treatment (Luostarinen et al., 2013). The initial weight gain following abstinence may cause some to relapse (Pisinger and Jorgensen, 2007). Thus, offering information and support to maintain body weight after smoking cessation may be of importance in order to reduce the probability of relapse. It is important to communicate that a possible post-cessation weight gain is much healthier compared to continuing smoking (Siahpush et al., 2014).

This study shows a substantial decrease in the prevalence of daily smokers, which we believe is due to a variety of legislative measures that have been implemented since the Norwegian Tobacco Act entered into force in 1975 (Helsedirektoratet, 2020). While appreciating that we have come a long way in fighting tobacco smoking, targeting cessation for the remaining group of smokers should remain a priority.

4.1. Strengths and limitations

We acknowledge that the results presented, in particular the prevalences, may not be representative for the group of non-participants. In the population based HUNT study Langhammer et al. (2012) found that non-participants were likely to be more unhealthy, with a higher prevalence of daily smoking though with a lower BMI. We have previously found that consistent attendees to Tromsø 2 – Tromsø 4 had lower mortality than those who only attended Tromsø 4, although had been invited to all three surveys (Jacobsen et al., 2012). However, the Tromsø Study has a high attendance (range 65–79% in Tromsø 4–7), which is a significant strength of this study. Another strength is that height and weight were measured, as opposed to self-reported which is prone to bias (Gorber et al., 2007). Participants in the Tromsø Study reported smoking habits on self-administered questionnaires which probably means that the number of daily smokers are under-reported (Løchen et al., 2017) and also that there may be some misclassification in the smoking subgroups. Validation studies in Finland and Norway have shown a high degree of concordance between self-reported smoking and biological markers of smoking (Foss et al., 1998; Vartiainen et al., 2002). Thus, there is little reason to believe that the information bias from self-reported smoking has had any substantial effect on the results presented.

In the analysis of BMI change by smoking status we did not model all four surveys jointly, which can be seen as a limitation. On the other hand, the statistical methods we used are simple and well-known. A possible challenge in using all four surveys is the number of possible smoking status combinations ($2 \times 3^3 = 54$) and missing data.

5. Conclusion

We conclude that smoking cessation contributed to an observed increase in obesity in the population, but was probably not the most important factor. Public health interventions should continue to target smoking cessation, and also target obesity prevention.

Declaration of Competing Interest

The authors have no conflicts of interest to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ypmed.2021.106533.

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