Impact of Smoking and Excess Body Weight on Overall and Site-Specific Cancer Mortality Risk

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Abstract

Background: Smoking and excess body weight are major preventable risk factors for premature death. This study aimed at analyzing their single and combined association with site-specific cancer mortality.

Methods: Our study population comprised 35,784 men and women of ages 14 to 99 years, who participated in population-based health surveys conducted 1977–1993 in Switzerland and were followed up for mortality until 2008. Multivariable Cox proportional hazards models were calculated for different cancer sites, and population attributable fractions were derived.

Results: The hazard ratio of dying from cancer (all sites) was 2.32 (95% confidence interval, 2.04–2.63) for heavy smokers (vs. never smokers) and 1.15 (1.01–1.32) for obese [body mass index (BMI) ≥ 30 kg/m²] vs. normal weight individuals. Heavy smoking (≥ 20 cigarettes/day) was associated with increased mortality due to cancer of the lung, upper aero-digestive tract, pancreas, bladder, liver, and the total of remaining sites. Obesity was associated with higher risk of dying from cancer of the liver and the female genital tract (essentially corpus or cervix uteri and ovary). More than 20% of all cancer deaths in our population were attributable to ever smoking and overweight (BMI ≥ 25 kg/m²).

Conclusions: Smoking was a much stronger risk factor for cancer than excess body weight. For lung, liver, and pancreatic cancer, the combination of excess body weight and smoking lead to cumulated higher risks.

Impact: Our findings support recommendations for obese persons to quit smoking despite potential postcessation weight gain. Cancer Epidemiol Biomarkers Prev; 24(10); 1–7. ©2015 AACR.

Introduction

In Switzerland, as in other developed countries, age-standardized mortality due to cancer is declining, however less than that due to other causes of death. As a consequence of population aging, case counts are still increasing. Even more unfavorable are the figures for cancer incidence (1), underlining the importance of prevention. Smoking and obesity are two major risk factors for premature death in western populations (2–4). Their prevalence is still high in many countries. Although smoking has slowly decreased over the past decades (particularly in men), it is unclear whether the obesity epidemic has reached its peak (5). In Switzerland, recent trends suggest a continuation of the increase of the obesity prevalence over time in adults (6). Individuals, who accumulate these two risk factors, that is, smoking plus obesity, may have a particularly high risk of dying from cancer. Statistics from 2012 indicate that of the adult male and female permanent resident population of Switzerland, 49% and 29% had excess body weight [body mass index (BMI) ≥ 25 kg/m²], 29% and 22% smoked, and 13% and 6% had excess body weight and smoked, respectively (7).

The few studies looking at this co-occurrence provided, however, conflicting results (2, 8, 9). Freedman and colleagues (2) reported the cancer mortality risk to be higher in obese than in normal weight current smokers, but for older individuals (age ≥ 65 years) the association between obesity and cancer mortality was more apparent in never than in former or current smokers. In contrast, Kitsantas and colleagues (8) reported that younger women (41–50 years) who smoked had a higher cancer mortality risk if they were underweight. Hjellvik and colleagues (9) examined middle-age death risk and observed the combination of obesity and heavy smoking to increase the risk by the factor of 5.

Moreover, it is unclear whether specific cancer sites are more affected than others by smoking, obesity or both, and which BMI levels, that is, overweight and obesity vs. normal weight, are important.

Our aim was to examine the significance of smoking and increased BMI on overall and site-specific cancer mortality in terms of relative risks and population attributable fractions (PAF). For this purpose, we used data from general population-based studies conducted in Switzerland between 1977 and 1993, which were linked with the Swiss National Cohort (SNC) for a mortality follow-up until the end of 2008.

Materials and Methods

Study population

Details about the study population were described previously (10). In brief, it consists of 35,784 participants of ages 14 to 99 years at baseline who participated in one of four different...
population-based studies conducted between 1977 and 1993: NRP 1A [National Research Program 1A, 1977–79], SOMIPOPS (Socio-Medical Indicators for the POPopulation of Switzerland, 1982), Swiss MONICA (MONItoring of trends and determinants in CArdiovascular disease, three waves: I, 1984–86; II, 1988/89; III, 1992/93), and the first SHS (Swiss Health Survey, 1992/93). Cause-specific mortality follow-up until 2008 was obtained via anonymous record linkage with the SNC (10, 11).

After exclusion of 81 participants because of missing values in covariates used for the fully adjusted model, our study population comprised 35,703 participants with a mean and maximum follow-up time of 18.9 and 31.9 years, respectively. More details about the study population stratified by survey can be found in Supplementary Table S1.

Body mass index
BMI was defined as weight (kg) divided by the square of height (m²) and classified into following categories: normal/underweight (reference category, BMI < 25.0 kg/m²), overweight (25.0 ≤ BMI < 30.0 kg/m²), and obesity (BMI ≥ 30.0 kg/m²). Only about 3.5% of the study population was underweight (BMI < 18.5 kg/m²). Moreover, this underweight population (irrespective of smoking status) had a similar cancer risk as normal weight persons (10). We therefore combined this category with normal weight in our analyses. In MONICA and NRP 1A, weight and height were measured at baseline, whereas in SOMIPOPS and SHS 92/93, they were reported by the participants (10).

Smoking
We defined the following smoking categories: never (reference category), former (reported having ever smoked for more than 6 months), current light (<20 cigarette equivalents a day), and current heavy (≥20) smokers. Thereby, one pipe or cigarillo equals two, and one cigar equals four cigarette equivalents. Unfortunately, we only had fragmentary information on smoking duration and intensity in former smokers.

Covariates
Besides the covariates smoking and BMI we chose age (in years), sex (where appropriate), survey, years of education, marital status, nationality, frequency of physical activity (PA), alcohol consumption, and a diet proxy variable for the fully adjusted model. For all covariates, we used baseline information.

The covariate survey had six categories: NRP 1A, SOMIPOPS, MONICA I-III, and SHS. Marital status had four categories: single, married, widowed, and divorced. Nationality was binary: Swiss and other nationalities. For frequency of PA, we used the categories daily, several times per week, once per week, and less. Alcohol was used as a binary variable and refers to the consumption the day before the interview (yes/no). As a proxy for a healthy diet, a binary variable was defined: for MONICA, NRP 1A, and SOMIPOPS as "regularly eating three main meals per day" and for SHS 92 of 93 as "eating fruits and vegetables at least once per day."

Outcome variables
Causes of death were coded according to the International Classification of Diseases (ICD). Until the end of 1994 the 8th (ICD-8) and since 1995 the 10th (ICD-10) revision was used. For our cancer-specific analyses, we aggregated them in the following 13 cancer groups: all cancers (ICD-8: 140–239; ICD-10: C00-C99; D00-D48), cancer of the lung (ICD-8: 162; ICD-10: C33-C34), colon or rectum (ICD-8: 153–154; ICD-10: C18-C21), upper aero-digestive tract (UADT, ICD-8: 140–150, 161; ICD-10: C0, C10-C15, C32), lymphatic and hematopoietic tissue (abbreviated as "blood cancer" in the following, ICD-8: 200–209; ICD-10: C81-C86, C88, C90-C96), pancreas (ICD-8: 157; ICD-10: C25), bladder (ICD-8: 188–189; ICD-10: C67-C68), liver (ICD-8: 155; ICD-10: C12), stomach (ICD-8: 151; ICD-10: C16), breast (ICD-8: 174; ICD-10: C50), female genital tract (ICD-8: 180–184; ICD-10: C51-C58), prostate (ICD-8: 185; ICD-10: C61), and the total of remaining cancer sites.

Statistical analysis
For descriptive analyses, we stratified the population by sex and smoking status and calculated counts, means, standard deviation, and proportions. To reduce the number of missing values, we imputed (12) the covariates BMI [number of missing values (n = 451), years of education (n = 577), and frequency of PA (n = 1,021). Multiple imputation was used for BMI and frequency of PA. As we had information on the education category [mandatory (modal value 9 years), further education (12 years), higher education (16 years), university (19 years)] for most of the missing values in the variable "years of education," we used the modal value (was the same for women and men in all categories) for each category as imputation value. For all analyses, we excluded 81 observations that after imputation still contained missing values for one of the covariates required for the fully adjusted model.

To derive hazard ratios (HR), we calculated Cox proportional hazards models. We hereby used survival or censoring time from study entry as well as different cancer site-specific binary variables as outcomes. The models were calculated for both sexes separately and combined. Time to event or censoring was defined as time difference between study entry (date of baseline interview) and date of death from site-specific cancer, or the possible censoring dates of death from other causes, emigration, or end of the study (December 31, 2008), respectively. Model variables were selected according to Akaike’s information criterion (AIC). We calculated two Cox models for every cancer-specific outcome with different adjustment levels: besides smoking and BMI, the basic model was adjusted for age, sex (where appropriate), and survey. The multivariable-adjusted model additionally included years of education, marital status, nationality, frequency of PA, alcohol consumption, and healthy diet. We also tested for a multiplicative interaction effect of smoking with BMI in the models. In order to calculate cumulative HRs for different risk combinations of BMI and smoking categories, we used linear combinations of the estimated HRs from the Cox models. The proportional hazards assumption was tested using Schönfeld residuals. The assumption was sufficiently fulfilled for all sex-specific models and not fulfilled for three out of 10 sex-combined models, namely for all cancer deaths combined, lung cancer, and the total of remaining cancer deaths.

To calculate PAFs for smoking and BMI, we used the punafcc STATA package by Newson (13). Smoking (ever including former vs. never smoking) and BMI (cutoff point at 25 kg/m²) were dichotomized for this analysis. This cutoff point for BMI was selected because of small case numbers for obese participants.

All analyses were conducted in STATA 13 (StataCorp. 2013. Stata Statistical Software: Release 13. StataCorp LP).
Results

Baseline characteristics and figures for specific cancer sites are shown in Table 1. Heavy smokers exhibited higher proportions of persons being physically inactive and following a poor diet than individuals of the other smoking categories. The proportion of those who reported alcohol consumption on the previous day was

<table>
<thead>
<tr>
<th>Table 1. Baseline characteristics of the study population and figures of cancer mortality for specific cancer sites stratified by smoking status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking status</td>
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<tr>
<td>----------------</td>
</tr>
<tr>
<td>Observations, n</td>
</tr>
<tr>
<td>Observations, %</td>
</tr>
<tr>
<td>Deaths from all causes, n</td>
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<tr>
<td>% Deaths from all causesc</td>
</tr>
<tr>
<td>Person-years</td>
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<tr>
<td>Mean (SD) age in years</td>
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<tr>
<td>Mean (SD) BMI in kg/m²</td>
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<tr>
<td>BMI categories, %</td>
</tr>
<tr>
<td>BMI &lt; 25</td>
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<tr>
<td>25 ≤ BMI &lt; 30</td>
</tr>
<tr>
<td>BMI ≥ 30</td>
</tr>
<tr>
<td>Marital status, %</td>
</tr>
<tr>
<td>Single</td>
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<tr>
<td>Married</td>
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<tr>
<td>Widowed</td>
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<tr>
<td>Divorced</td>
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<tr>
<td>Frequency of PA, %</td>
</tr>
<tr>
<td>Daily</td>
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<tr>
<td>Several times per week</td>
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<td>Once per week</td>
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<tr>
<td>Less than once per week</td>
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<tr>
<td>Other covariates</td>
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<tr>
<td>Alcohol (% yes)</td>
</tr>
<tr>
<td>Healthy dietd (% yes)</td>
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<tr>
<td>Mean (SD) years of education</td>
</tr>
<tr>
<td>Nationality (% Swiss)</td>
</tr>
</tbody>
</table>

Cancer mortality

| All cancer sites | n | 985 | 494 | 467 | 433 | 2,379 |
| Lung cancer | n | 76 | 79 | 93 | 178 | 426 |
| Colorectal cancer | n | 125 | 61 | 44 | 20 | 250 |
| UADT cancer | n | 27 | 24 | 31 | 43 | 125 |
| Blood cancer | n | 120 | 53 | 45 | 28 | 246 |
| Pancreatic cancer | n | 56 | 28 | 26 | 17 | 127 |
| Bladder cancer | n | 36 | 15 | 16 | 18 | 85 |
| Liver cancer | n | 24 | 22 | 16 | 19 | 81 |
| Stomach cancer | n | 37 | 19 | 8 | 10 | 74 |
| Breast cancera | n | 141 | 20 | 19 | 19 | 204 |
| Cancer of female genitalsa | n | 86 | 15 | 24 | 7 | 132 |
| Prostate cancerb | n | 49 | 57 | 52 | 12 | 170 |
| Remaining cancer cases | n | 208 | 102 | 89 | 63 | 462 |

NOTE: Sex-specific results can be found in Supplementary Table S2.

*a*Current light smokers: smoking less than 20 cigarette equivalents a day. One pipe or cigarillo corresponds to two and one cigar to four cigarette equivalents.

*b*Current heavy smokers: smoking at least 20 cigarette equivalents a day.

*c*Percent of all observations in this smoking category.

*d*Defined as follows: MONICA, NRP 1A, and SOMIPOPS: regularly eating three main meals per day; SHS 92/93: eating fruits and vegetables at least once per day.

*e*Percent of all deaths in this smoking category.

*f*Percent of all cancer deaths in this smoking category.

*g*Only women.

*h*Only men.
lower in never smokers compared with all others. The mean age at baseline was lower for light and heavy smokers than for never and former smokers. Mean BMI was highest for former smokers. Sex-specific values are shown in Supplementary Table S2. Of the 7,383 persons who died in 676,312 person-years, 2,379 died of cancer. The proportion was higher in ever smokers (35.9%) than in never smokers (28.3%). This was mainly due to lung cancer and—to a smaller extent—to UADT cancer. In heavy smokers, about 41% and 10% of cancer deaths were from lung and UADT cancers as opposed to 8% and 3% in never smokers, respectively. On the other hand, deaths from other cancers, such as colorectal, were less frequent in heavy smokers compared with the other smoking categories.

Table 2 shows HRs with 95% CIs for different BMI and smoking categories resulting from the fully adjusted model (statistically significant values are indicated in bold).

### Table 2. HRs with 95% CIs for different BMI and smoking categories resulting from the fully adjusted model (statistically significant values are indicated in bold)

<table>
<thead>
<tr>
<th>Cancer site</th>
<th>Deaths (n)</th>
<th>BMI &lt; 25</th>
<th>BMI 25–30</th>
<th>BMI &gt; 30</th>
<th>Former Smoking 1 (cigarette equivalents per day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All sites</td>
<td>2,379</td>
<td>1.01 (0.92–1.10)</td>
<td>1.15 (1.01–1.32)</td>
<td>1.31 (1.16–1.47)</td>
<td>1.37 (1.22–1.53)</td>
</tr>
<tr>
<td>Lung</td>
<td>426</td>
<td>0.95 (0.77–1.17)</td>
<td>0.80 (0.57–1.14)</td>
<td>2.29 (1.64–3.20)</td>
<td>3.31 (2.26–4.26)</td>
</tr>
<tr>
<td>Colon or rectum</td>
<td>250</td>
<td>1.11 (0.84–1.48)</td>
<td>1.30 (0.87–1.95)</td>
<td>1.13 (0.80–1.58)</td>
<td>0.99 (0.69–1.42)</td>
</tr>
<tr>
<td>UADT 2</td>
<td>125</td>
<td>0.71 (0.48–1.05)</td>
<td>0.78 (0.42–1.43)</td>
<td>1.14 (0.80–1.62)</td>
<td>1.00 (0.70–1.43)</td>
</tr>
<tr>
<td>Blood</td>
<td>246</td>
<td>1.02 (0.77–1.35)</td>
<td>1.13 (0.74–1.74)</td>
<td>1.14 (0.89–2.40)</td>
<td>1.46 (0.89–2.39)</td>
</tr>
<tr>
<td>Pancreas</td>
<td>127</td>
<td>1.20 (0.81–1.78)</td>
<td>1.60 (0.93–2.75)</td>
<td>1.01 (0.55–1.95)</td>
<td>1.22 (0.65–2.29)</td>
</tr>
<tr>
<td>Bladder</td>
<td>85</td>
<td>0.62 (0.38–1.02)</td>
<td>1.16 (0.62–2.18)</td>
<td>2.14 (1.14–4.01)</td>
<td>1.76 (0.91–3.42)</td>
</tr>
<tr>
<td>Liver</td>
<td>81</td>
<td>1.28 (0.77–2.12)</td>
<td>2.21 (1.18–4.15)</td>
<td>1.23 (0.66–2.27)</td>
<td>0.60 (0.27–1.34)</td>
</tr>
<tr>
<td>Stomach</td>
<td>74</td>
<td>1.19 (0.72–1.98)</td>
<td>1.45 (0.71–2.95)</td>
<td>0.79 (0.49–1.29)</td>
<td>0.77 (0.49–1.21)</td>
</tr>
<tr>
<td>Female breast</td>
<td>201</td>
<td>1.16 (0.84–1.60)</td>
<td>1.07 (0.64–1.79)</td>
<td>1.21 (0.69–2.12)</td>
<td>1.48 (0.92–2.36)</td>
</tr>
<tr>
<td>Female genitals 3</td>
<td>132</td>
<td>1.31 (0.88–1.95)</td>
<td>1.88 (1.10–3.20)</td>
<td>1.12 (0.76–1.66)</td>
<td>1.38 (0.93–2.05)</td>
</tr>
<tr>
<td>Prostate</td>
<td>170</td>
<td>1.45 (1.03–2.04)</td>
<td>1.54 (0.93–2.55)</td>
<td>1.22 (0.94–1.58)</td>
<td>1.18 (0.91–1.54)</td>
</tr>
<tr>
<td>Remaining</td>
<td>462</td>
<td>0.89 (0.73–1.09)</td>
<td>1.08 (0.80–1.46)</td>
<td>1.22 (0.94–1.58)</td>
<td>1.18 (0.91–1.54)</td>
</tr>
</tbody>
</table>

NOTE: Models are adjusted for sex (where appropriate), age, survey, alcohol consumption, frequency of PA, civil status, years of education, nationality, and healthy diet.

Sex-specific results can be found in Supplementary Table S3.

1Reference: BMI < 25 kg/m².

2Reference: smoking BMI interaction term did not significantly improve our models for all 13 cancer sites. In a sensitivity analysis, we calculated the Cox models in participants with measured BMI (NRP1A, MONICA I-III). The associations with all sites combined and liver cancer mortality were very similar. However, the association between obesity and pancreas cancer was statistically significant (HR, 2.37; 95% CI, 1.25–4.48) despite fewer mortality cases (77 vs. 127).

As shown in Fig. 1, there is a suggestive cumulative association of BMI and smoking with specific cancer sites, such as pancreas or liver. For the other cancer sites, there was no significant cumulative association seen. Exact figures for the cumulative HRs including 95% CIs are shown in the Supplementary Table S4 for all specific cancer sites.

Discussed HRs and 95% CIs from the fully adjusted model are shown in Supplementary Table S5. About 22% of prostate cancer deaths attributable to ever (including former) smoking and BMI ≥ 25 kg/m² are shown in Fig. 2. Exact values of the PAFs including 95% CIs, also for other relevant cancer sites and sex-specific, are shown in Supplementary Table S5. About 22% of prostate cancer deaths were attributable to BMI ≥ 25 kg/m². For total, lung, and liver cancer, 13%, 61%, and 39% of deaths were attributable to ever smoking, respectively. Of all deaths, deaths due to liver, pancreatic, and prostate cancer 50%, 29%, and 29% were attributable to ever smoking and overweight combined, respectively.

**Discussion**

Our results show that smoking was more strongly associated with cancer-related mortality than obesity. Heavy smokers had higher risks of dying from total, lung, UADT, pancreatic, and liver cancer compared with never smokers. Moreover, former and light smokers had an increased risk of dying from any cancer, mainly due to lung and UADT cancer. Being obese compared with normal weight was associated with a small increased overall cancer risk and an elevated site-specific risk for cancer of the liver, the female genital tract as well as pancreatic cancer (only in
participants with measured BMI). We did not observe a significant interaction between increased BMI and smoking on cancer-related mortality risk, which speaks against a substantial cumulative association with smoking and BMI. When calculating the PAF of ever smoking and overweight combined, there was a suggestive cumulative association with cancer-related mortality risk for some cancer sites. More than 20% of all cancer deaths, in particular 50%, 29%, and 29% of liver, pancreatic, and prostate cancer-related mortality cases were attributable to ever smoking and excess weight (BMI ≥ 25 kg/m²).

On the basis of previous reviews (14–17) on cancer incidence, regarding the sites analyzed in this study, an association with smoking has been reported for lung, stomach, pancreatic, bladder, UADT, and liver cancer. In our analysis, we confirmed all these associations except for stomach cancer, which could be due to the relatively small number of cases (n = 74). However, the results from the basic model showed a significantly increased stomach cancer risk in heavy smoking men (HR, 2.69; 95% CI, 1.08–6.73; n = 10). On the basis of the same reviews, for colorectal, breast, and prostate cancer, an association with smoking was uncertain or unlikely. This is in line with our results. A more recent review, however, has shown that current smoking is related to progression in prostate cancer leading to more aggressive and fatal cases (18).

For incidence and mortality of colorectal cancer, smoking was reported to be a risk factor (14, 19). In previous studies, an association with smoking was shown for myeloid but not for lymphatic leukemia (15). We did not account for this difference in our analysis and did indeed not see an association with smoking on overall blood cancer risk. For cancer of the female genital tract, we did not detect any association with smoking. Our definition of this site is a mix of different female cancers. Others suggested that smoking has no association with ovarian (15), an inverse association with endometrial (15, 20), and doubles the risk for cervical cancer (15).

Concerning incidence, the following of the cancer sites analyzed in our study were shown to be positively associated with obesity: colon, postmenopausal breast, adenocarcinoma of the esophagus (included in our definition of UADT cancer), endometrium, pancreas, advanced-stage prostate, and liver (21). For the remaining sites, evidence is conflicting. Accordingly, we observed an association of obesity with liver cancer and in participants with measured BMI values with pancreatic cancer. For prostate cancer, we observed a statistically significant association with overweight but not obesity. A possible explanation is the low power, as our population comprised only 23 obese prostate cancer deaths. In our data, obesity was also positively
associated with death due to cancer of the female genital tract. In the Million Women Study (UK), an association between elevated BMI and endometrial as well as ovarian but not cervical cancer incidence was observed (22). In contrast to our results, in the same study, an association between obesity and leukemia was reported. Again, there are discordant definitions and also the power might have been too low (14 blood cancer deaths among obese women). For breast cancer, we did not detect an association with BMI. Because of lack of information, we were unable to accurately classify deaths by menopausal status. Again, probably due to lack of statistical power, we could not confirm the association of obesity with pancreatic cancer (n = 19). Furthermore, this association appears to be generally weak (23). We did see, however, that the mean BMI in female and male cases was significantly about 1.9 and 1.3 BMI units higher, than in non-cases, respectively. Moreover, there may be a cumulative association of smoking plus excess body weight with pancreatic cancer mortality risk (Figs. 1 and 2). Relatively few studies examined the interaction of these two risk factors. Previous publications corroborate our findings of a stronger impact of smoking (vs. obesity) on cancer risk (2, 3, 8). This might be explained by residual confounding of unobserved lifestyle habits, as smokers tend to exhibit an unhealthier overall lifestyle (24, 25). Besides, a lack of a general BMI–smoking interaction on mortality was reported previously (26, 27). However, as suggested by our results, this joint association may depend on the respective cancer site. We could identify one study that also suggested a cumulative association of obesity and smoking with pancreatic cancer risk (28). In contrast to our results, Rauscher and colleagues (29) observed that obesity was associated with lung cancer risk in a nonsmoking population. Furthermore, Luo and colleagues (30) observed that the association of smoking with breast cancer risk is modified by obesity in postmenopausal women, which was also not apparent in our population.

Our study comprised a large general population sample with a follow-up time of up to 32 years. The data stem from Switzerland, a country with a highly developed health care system allowing reliable diagnoses. Our data also included a wide range of lifestyle factors allowing to account for potential confounding. Another strength of our study is that we were able to calculate the impact of smoking and BMI on specific cancer sites. Our study has several limitations. First, we had data only on cancer-related mortality and no information on cancer incidence. However, Reeves and colleagues (22) reported that the relationship between BMI and cancer mortality was similar to that of cancer incidence in women. Second, we only had baseline information on smoking and BMI. In light of the long follow-up time, participants might have changed their behavior over time. Third, the PAFs were calculated only on the basis of our baseline and mortality data, which stem from up to 39 years ago. Therefore, the results should be interpreted with caution, as the prevalence, for example, of tobacco use or lung cancer-related mortality rates changed over the years. Fourth, the exposure measurement in our study was crude and, for some variables, not uniform as we pooled different samples to obtain a sufficient number of specific cancer cases. For example, BMI was partly measured and partly self-reported and the definitions for healthy diet were different. The different BMI measurements, however, should not have a substantial impact on the relative mortality risks as shown previously by our group (10). In this publication, the same pooled dataset was used to analyze all-cause mortality and a sensitivity analysis using a mixed effects Cox model with random effects for survey was performed. The results of that sensitivity analysis showed that adjusting for survey sufficed. Accordingly, we adjusted for survey in all our Cox models to compensate for heterogeneity. Finally, our study population is presumably healthier than the general population, which might influence prevalence rates (31). However, in the same study, there was no difference in cancer-related mortality risk between the MONICA sample and the general population detected. We would expect that this applies also to the other samples included in our study.

Conclusion

In Switzerland, 19.7% of fatal cancers were attributable to ever smoking. When adding overweight, this proportion increased slightly to 20.6%. In this country, as in other developed nations, cancer as cause of death is gaining relative significance. Therefore, the government, public health authorities, and primary care providers need to intensify their efforts aimed at reducing the modifiable proportion of cancer risk. Our results suggest that tobacco control is by far the most efficient measure to prevent cancer deaths. The overall impact of excess weight on cancer-related mortality risk was comparably small, which underlines the importance of recommendations for obese persons to quit smoking despite potential postcessation weight gain.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Authors’ Contributions

Conception and design: S. Rohrmann, M. Bopp, D. Faeh
Development of methodology: J. Meyer, S. Rohrmann, D. Faeh
Acquisition of data (provided animals, acquired and managed patients, provided facilities, etc.): M. Bopp
Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): J. Meyer
Writing, review, and/or revision of the manuscript: J. Meyer, S. Rohrmann, M. Bopp, D. Faeh
Administrative, technical, or material support (i.e., reporting or organizing data, constructing databases): M. Bopp
Study supervision: M. Bopp, D. Faeh

Acknowledgments

The authors thank the Swiss Cancer Research foundation for supporting our study. The authors also thank the Swiss Federal Statistical Office for providing mortality and census data and for the support, which made the SNC and this study possible. The members of the SNC Study Group are Matthias Egger (Chairman of the Executive Board), Adrian Spoerri and Marcel Zwahlen (all Bern), Milo Puhan (Chairman of the Scientific Board), Matthias Bopp (both Zurich), Nino Künzli (Basel), Fred Paccaud (Lausanne), and Michel Oris (Geneva). Finally, the authors thank Julia Braun and Tina Lohse for their input.

Grant Support

This work was supported by the Swiss Cancer Research foundation (grant no. KFS-3048-08-2012, to S. Rohrmann). The SNC was supported by the Swiss National Science Foundation (grant nos. 3347CO-108806, 33CS30_134273, and 33CS30_148415).

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Received April 22, 2015; revised July 8, 2015; accepted July 22, 2015; published OnlineFirst July 27, 2015.
Impact of Smoking and Excess Body Weight on Cancer Mortality

References


Impact of Smoking and Excess Body Weight on Overall and Site-Specific Cancer Mortality Risk


Cancer Epidemiol Biomarkers Prev Published OnlineFirst July 27, 2015.

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