Original article

Smoking in relation to anxiety and depression: Evidence from a large population survey: The HUNT study

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Abstract

Smoking is reported to be associated with depression and anxiety. The present study (a) examines these associations taking comorbidity into account, (b) investigates possible confounders, (c) examines how former smokers compared to current and never-smokers in terms of anxiety and depression, and if anxiety and depression decline by time since cessation. Participants (66%) aged 20−89 years in a population-based health survey ($N = 60,814$) were screened employing the HADS. (a) The association with smoking was strongest in comorbid anxiety depression, followed by anxiety, and only marginal in depression. Associations were stronger in females and younger participants. (b) Variables partly accounting for the association comprised somatic symptoms, socio-demographics, alcohol problems, and low physical activity. (c) Anxiety and depression were most common in current smokers, followed by quitters, and then never-smokers. No decline in anxiety or depression was found with time since cessation. Previous studies of associations between depression and smoking might have overestimated the association when ignoring comorbid anxiety.
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1. Introduction

The detrimental effects from smoking on health are well known and mortality attributable to smoking is high [13]. Smoking has been found to be associated with a wide range of mental disorders [8,16,26,29,30,35,41,45,46]. In World Health Organisation surveys daily smoking is reported by 30% of the population in the western world [44]. Epidemiological studies have found that people with a mental disorder are twice as likely to smoke compared to the general population [33], and are high consumers of cigarettes [22]. Considering the most prevalent mental disorders, associations between depression and smoking have been shown repeatedly [8,11,18,20], but more recent work has also highlighted the importance of anxiety disorders [5,16,21,28,30,34,35,41,46,47]. Anxiety and depression are commonly comorbid [38]. However, the degree to which smoking is uniquely associated with anxiety, as opposed to be associated with co-occurring depression, is unclear [35].

Other factors may confound associations between smoking and anxiety/depression, including somatic health, other health-related behaviours, socio-economic status, age and gender. A recent literature review on anxiety and smoking call for more focus on moderators and mechanisms for improved understanding of aetiology [35]. Psychosocial factors and comorbid mental disorders have been implicated as important confounders in young adults [2,17]. Failure to take such confounders into account may have led to ungrounded optimism

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in causal theories on the link between smoking and specific mental disorders.

The public health response to smoking has usually consisted of campaigns directed at cessation or discouraging initiation of smoking. Previous research supports these policies, suggesting improved mood and stress-relief post cessation [39,40], and authors have emphasised the need to focus on cessation in people with mental disorders [22,23,33]. On the other hand, abstaining smokers with a history of major depression disorder (MDD) have an increased risk of developing a new episode of MDD over a 4 week [10] to 6 month [19] period. Beyond that time frame little is known about the long-term effects of quitting smoking in people with mental disorders. There are also speculations and some inconclusive empirical evidence that anxiety precludes cessation [35].

Finally, as to causality, there is evidence that smoking increases the risk of depression [29,31]. As to certain anxiety disorders [35], and in particular post traumatic stress disorder [3,16], evidence suggest the opposite causality. There is evidence for shared genetic effects in smoking and depression [31], and also in nicotine dependency and post traumatic stress disorder [30]. Quitters are reported to have non-increased risk for a new depressive episode compared to never-smokers [29], but there are power issues in most such studies. Comparisons of quitters and never-smokers (and time since cessation in quitters) in terms of mental illness might shed light on these issues of causality.

The aim of this study was to address these issues in a secondary analysis of data from a large community population. Specific objectives were as follows: (a) to investigate the association between smoking, anxiety and depression taking into account comorbidity between the two mental disorders; (b) to investigate the extent of confounding by other health-related behaviour, somatic health complaints, and socio-demographic factors; (c) to investigate anxiety and depression in former smokers: in particular associations with time since cessation. We hypothesized (a) increased anxiety, depression and comorbid anxiety depression in smokers compared to non-smokers, (b) and that these associations are not entirely explained by confounding factors. (c) Further, we hypothesized that there would be increased anxiety and depression in quitters compared to never-smokers (hypothesizing a trait-association), but also a normalization over anxiety and depression to never-smoker levels by elapsing time (hypothesizing a state-association).

2. Methods

2.1. Design, participants and procedures

In the cross-sectional Health Study of Nord-Trøndelag County (HUNT-II), self-reported data on smoking, mental and physical health, and demographic information and socio-economic status was collected. In addition, body mass index (BMI) and blood pressure were measured by trained community nurses [25]. Of 92,100 eligible inhabitants aged 20–89, 60,814 (66%) participated with completed variables relevant for the present study. The female proportion of the sample was 52.7%, and the ethnic diversity was minimal [25].

2.2. Definition of depression and anxiety

The Hospital Anxiety and Depression scale (HADS) is a self-report questionnaire comprising 14 four-point Likert-scaled items: seven for anxiety (HADS-A) and seven for depression (HADS-D) with reference to the two preceding weeks [38]. Somatic symptoms and sleep/appetite disturbance are specifically excluded to avoid false positive cases in individuals with physical disorders. A cut-off score of 8 on each subscale was found to give an optimal balance between sensitivity and specificity (both about 0.8) for depression and anxiety according to DSM-III and IV, or ICD-8 and 9 [3]. Applying these cut-offs, two dichotomies for case-level anxiety and depression were computed. For the purpose of examining pure and comorbid conditions of anxiety depression, an additional variable was computed with four groups for anxiety only, depression only, comorbid anxiety and depression, and a reference group with no case-level disorder [43]. In accordance to previous publications employing HADS [36], we performed secondary analyses using anxiety and depression as dimensional scores.

2.3. Smoking

To investigate the association between current smoking and anxiety/depression, current smokers were compared to all other participants. Smoking was defined as current daily smoking of cigarettes, cigars or pipe. Next, comparisons were made between never-smokers and both current and former smokers. Finally, prevalence rates of anxiety and depression among former smokers were compared according to time since cessation.

2.4. Confounding variables

Confounding factors were operationalized according to previous analyses carried out on the HUNT database [36,37], and the variables are described in more detail in previous publications [25]. Information on age and gender was obtained from the national population registry [25]. Age was encoded in decades with 20–29 years as reference group. As in previous publications, physical health was assessed with one variable on the number of organ systems from which *somatic symptoms* were reported (symptoms) and one on number of *somatic diagnoses* (diagnoses) [36,37]. The index for somatic symptoms was computed as the number of (up to six) organ systems from which symptoms were reported [36,37]. The index for somatic diagnoses was computed as the number of (up to 15) reported diagnoses enquired about as part of the survey. Both indexes were entered with a range of 0–4 (values >4 truncated to 4) [36,37]. Socio-demographic factors consisted of educational level (primary school, high school, and university level), marital status (living with spouse or partner versus not) [36,37], and socio-economic status derived from current

occupation (seven categories) [32]. *Alcohol problems* were measured with the CAGE questionnaire, which comprises four questions on drinking behaviour, giving a sum-score with range 0−4 included as dummy-categories, with a separate category for missing responses [1]. Frequency of *physical activity* was measured with two questions enquiring about light and heavy activity (four categories from 'no activity' to '>2 h per week') [36,37]. *Physical measures* of BMI, blood pressure and total cholesterol level were also included as continuous variables [25]. HUNT was approved by the Board of Research Ethics in Health Region IV of Norway [25].

2.5. Data analysis

Employing logistic regression analyses, we analyzed smoking (independent variable) in relation to case-level anxiety, case-level depression, and comorbid case-level anxiety depression in separate analyses with non-cases as common reference group.

Candidate confounding/mediating factors were chosen on the basis on well-recognized associations to both common mental disorder and smoking. However, the etiology in many of these well-recognized associations are still unknown, thus we are unable to disentangle confounding from mediating factors in this cross-sectional analysis. Our list of covariates is extensive, but is obviously not complete.

We also compared the association between current smoking and anxiety with depression employing linear regression analysis with z-scored scale-scores for symptom load of anxiety and depression. Beyond adjustments for age and gender, in the analysis of the association between symptoms of anxiety and depression we adjusted for depression, and vice versa.

Further, we compared the prevalences of anxiety and depression between current smokers, quitters and never-smokers, with the latter as reference group, again employing logistic regression models and case-levels for anxiety and depression. Employing the same statistics, including quitters only, we analyzed the prevalences of anxiety and depression across groups defined by time since cessation.

Finally, we analyzed the smoking anxiety/depression association stratified for age (decades) and sex, and employing logistic regression models, we tested for interactions for age and sex.

3. Results

Current smoking was reported by 29% of the sample. A further 29% reported having ceased smoking, and 42.0% reported never having smoked. The prevalence rates of case-level anxiety and depression were 15.5% and 10.8%, respectively; 9.6% had anxiety without depression, 4.9% had depression without anxiety and 5.9% had both conditions. Further characteristics of the sample are reported in Table 1.

After adjustment for age and gender, the associations between smoking and anxiety (OR 1.56, 95% CI 1.49−1.63) and between smoking and depression (OR 1.48, 1.40−1.57) were very similar. However when pure and comorbid disorders

Table 1
Sample characteristics (reported separately for categorical and dimensional variables as used in the following analyses)

Variable	N	%	Mean	SD
Age			48.8	16.58
Gender				
Males	28,794	47.3		
Females	32,020	52.7		
Somatic symptoms (No. of organ systems affected)			1.62	1.22
Educational level				
Primary school only	22,676	37.3		
Secondary	26,056	42.8		
High school/university level	12,082	19.9		
Living with a spouse/partner				
No	21,109	34.7		
Yes	39,705	65.3		
Alcohol problems, CAGE				
0 points	32,483	53.4		
1 point	4136	6.8		
2 points	1900	3.1		
3 points	1076	1.8		
4 points	389	0.6		
Missing data	20,830	34.3		
Physical activity				
No activity	9266	15.2		
Less than 1 h/week	9120	15.0		
1−2 h/week	21,110	34.7		
3 or more hours/week	21,318	35.1		
Body mass index (BMI)			26.3	4.06
Diastolic blood pressure			80.0	12.1
Systolic blood pressure			137.0	21.3
Total cholesterol level			5.86	1.25
Number of somatic diagnoses			0.49	0.83

were distinguished (with no disorder as the reference group), associations were strongest for comorbid anxiety and depression (OR 1.82, 1.69−1.95), followed by pure anxiety (1.43, 1.35−1.52), while the association with pure depression was relatively weak (OR 1.16, 1.07−1.26). The stronger association in comorbid anxiety depression is an additive effect (Table 2).

We reached the same conclusion employing z-scored scale-scores for symptom load of anxiety and depression in a secondary analysis: adjusted for age and gender, the association between smoking and anxiety ($B = 0.175$) was similar, though slightly stronger than that of depression ($B = 0.153$). The association between symptom load of anxiety and smoking was more robust for adjustment for symptom load of depression ($B = 0.084$) than the opposite ($B = 0.052$) (Table 3).

There were no differences in the outcome of interest between those smoking cigarettes only ($N = 16,686$) versus those smoking cigars or pipe only ($N = 569$) or combinations ($N = 432$) (all $p > 0.17$ adjusted for age and gender).

Analyses of confounding factors are presented in Table 2, ordered by effect of a given factor on the association between comorbid anxiety/depression and smoking. Somatic symptom score was the strongest factor accounting for the association between anxiety/depression and smoking, followed by socio-demographic factors, alcohol problems and physical activity.

Table 2
Anxiety and depression in relation to smoking, adjusted for confounders (OR with 95% confidence intervals)

Separate adjustments beyond age and gender	Anxiety alone	Depression alone	Both[a]
Age and gender only	1.43 (1.35−1.52)	1.16 (1.07−1.26)	1.82 (1.69−1.95)
Somatic symptoms	1.32 (1.24−1.40)	1.10 (1.01−1.20)	1.63 (1.51−1.75)
Socio-demographics	1.39 (1.32−1.48)	1.13 (1.04−1.24)	1.69 (1.58−1.82)
Alcohol problems	1.38 (1.30−1.46)	1.15 (1.06−1.25)	1.75 (1.63−1.88)
Physical activity	1.42 (1.34−1.50)	1.12 (1.03−1.22)	1.76 (1.64−1.88)
BMI, BP, total cholesterol level	1.42 (1.34−1.50)	1.18 (1.08−1.28)	1.81 (1.68−1.94)
Number of somatic diagnoses	1.44 (1.36−1.52)	1.16 (1.07−1.27)	1.82 (1.70−1.96)
Adjustment for all above	1.19 (1.12−1.27)	1.04 (0.95−1.14)	1.40 (1.30−1.52)

[a] There was no anxiety by depression interaction in the association to smoking (all $p > 0.05$ across all levels of adjustment) by inclusion of the interaction term on top of a model already including anxiety, depression and covariates as mentioned under adjustments.

Adjustment for physical measures (BMI, blood pressure and total cholesterol level) and number of somatic diagnoses did not substantially explain the associations of interest. No single factor explained more than 25% of the association, and with all factors included in the model, about half the associations between smoking and anxiety and between smoking and comorbid anxiety and depression remained unexplained. The association between smoking and depression alone was almost entirely explained by inclusion of all confounders in the model.

In Table 4, anxiety and depression in both former and current smokers are compared to never-smokers, adjusted for age and gender, and then for all confounders reported in the previous model. For both anxiety and depression, associations were stronger for current, compared to former smokers. Associations were again strongest for comorbid anxiety/depression, followed by anxiety alone, and weak for depression alone. The stronger association in comorbid anxiety depression was again an additive effect. All odds ratios were statistically significant (also in the fully adjusted model), the exception being for depression alone which was not significant in the fully adjusted model. Adjustment for all confounders explained about two thirds of the associations for depression alone, slightly less for anxiety alone, and least (about half) for comorbid anxiety depression. Variables accounting for the differences between never, former, and current smokers (Table 4) were mainly the same as for the comparisons of smokers and non-smokers (Table 2).

When excluding both never-smokers and current smokers from the analyses, we were not able to find any association between time since cessation and odds of anxiety or depression (Table 5). Although there was a trend towards lower prevalence of depression alone with time since cessation (comparing individuals having quit smoking 15−25 and >25 years ago with 0−2 years ago), but across all five categories for time since cessation, this comparison was not statistically significant ($p = .085$). This conclusion was the same when restricting our sample to those old enough to have quit smoking long time ago (Table 5).

Smoking's associations to anxiety and comorbid anxiety/depression were stronger in females and younger participants (Table 6).

4. Discussion

4.1. Main findings

Our study has four main findings: (a) Anxiety was more strongly associated with smoking than was depression and the association between smoking and depression was marginal except in the presence of comorbid anxiety. Strongest associations were found in comorbid anxiety and depression. The associations were stronger in females and young participants. (b) Multiple factors confounded the associations, the most important being somatic symptoms (though not reported physical disorders, BMI, cholesterol level, or blood pressure), socio-demographics, and alcohol problems. (c) Anxiety and comorbid anxiety/depression were associated most strongly with current compared to former smokers, highest in smokers, followed by former smokers, and then never-smokers; all differences being statistically significant also having adjusted for available confounding factors. There were no associations between anxiety or depression and time since cessation in former smokers.

4.2. Strengths and limitations

The present study has several strengths: the sample-size is large, giving sufficient power to detect even small associations, and the participation rate was reasonable considering the size of the population targeted. The broad range of measured health, health-related, and socio-demographic factors allowed thorough examination of confounding factors. Finally,

Table 3
Symptom load of anxiety and depression in relation to smoking (continuous *z*-scored scores, obtained from linear regression analyses with 95% confidence intervals)

	Anxiety symptom load		Depression symptom load	
	B	95% CI	B	95% CI
Adjusted for age and gender	0.175	0.15−0.19	0.153	0.14−0.17
Further adjusted for other sub-scale[a]	0.084	0.07−0.10	0.052	0.04−0.07

[a] The association between smoking and anxiety adjusted for depression, and the association between smoking and depression adjusted for anxiety, all as *z*-scored continuous variables.

Table 4
Anxiety and depression in smokers and quitters compared to never-smokers (OR with 95% confidence intervals)

Groups	N (%)	Anxiety alone	Depression alone	Both
Adjusted for age and gender				
Never-smokers	25,504 (41.9)	1.00 (ref)	1.00 (ref)	1.00 (ref)
All quitters	17,623 (29.0)	1.22 (1.14−1.31)	1.11 (1.01−1.21)	1.34 (1.23−1.47)
Current smokers	17,687 (29.1)	1.55[a] (1.46−1.65)	1.24[b] (1.13−1.37)	2.09[c] (1.92−2.26)
Fully adjusted model				
Never-smokers	25,504 (41.9)	1.00 (ref)	1.00 (ref)	1.00 (ref)
All quitters	17,623 (29.0)	1.08 (1.00−1.62)	1.03 (0.93−1.13)	1.13 (1.03−1.24)
Current smokers	17,687 (29.1)	1.22[a] (1.13−1.30)	1.10[b] (0.99−1.21)	1.52[c] (1.39−1.66)

[a] For quitters compared to current smokers: age and gender adjusted OR = 1.23 (95% CI 1.15−1.33), fully adjusted OR = 1.13 (95% CI 1.05−1.22).
[b] For quitters compared to current smokers: age and gender adjusted OR = 1.10 (95% CI 0.99−1.21), fully adjusted OR = 1.03 (95% CI 0.93−1.13).
[c] For quitters compared to current smokers: age and gender adjusted OR = 1.51 (95% CI 1.38−1.64), fully adjusted OR = 1.32 (95% CI 1.21−1.45).

the population-based survey reduced the likelihood of selection bias which may be problematic in clinical samples.

The principal limitation was the cross-sectional nature of the study which limits our possibilities of empirically examining the extent and direction of causality between mental disorders and smoking behaviour. Higher psychiatric morbidity among non-responders in health surveys [15], and elevated consumption of tobacco among people with mental health problems [33,22], may have led to an underestimation of the association between smoking and mental health through selective participation.

Smoking and smoking cessation were self-reported and may be subjected to recall bias leaning towards desirable health behaviour, e.g. under-reporting smoking and over-reporting cessation which could weaken the associations.

Further, our reliance of this uni-method assessment approach is known to be associated with shared method variance, an issue that can affect observed relations. Consequently, future work in this area could benefit by using cross-method approach.

The data for this study is collected in one region of the world only, which obviously precludes generalizing to other and different populations and contexts.

The cross-sectional design precludes disentangling confounding from mediating effects in examination of possible mechanisms underlying the association of interest (Table 2). The main strength of this analysis is to identify the extent to which the candidate variables account for the association of interest. We include in empirical analyses a wider range of candidate mechanisms than any previous study in this field. Still, there are candidate explanations not available in our dataset, including for example shared genetic factors [30], coping styles or life events.

Finally, substance use was not covered in the survey, and might represent residual confounding. This is, however, a limited problem, as substance use (e.g. marijuana use) is relatively rare in the population studied, and as we know individuals with substance use to be under-represented in public health surveys.

Former smokers are also likely to be a self-selected group. Absence of mental disorders may make cessation more likely and more successful, and may offset to some extent adverse effects of withdrawal on mental health status, although a recent meta-analysis found that a history of major depression does not have an impact on future abstinence success rate [24].

Table 5
Time since cessation (quitters only) in relation to anxiety and depression, adjusted for age and gender (OR with 95% confidence intervals)

Time since cessation	N (%)	Anxiety alone	Depression alone	Both
All quitters included, age 20 to 89, N = 15,132[c]				
0−2 years	2325 (15.4)	1.00 (ref)	1.00 (ref)[a]	1.00 (ref)
3−7 years	2873 (19.0)	1.11 (0.93−1.33)	0.89 (0.69−1.15)	1.21 (0.95−1.53)
8−14 years	2701 (17.8)	0.90 (0.74−1.10)	0.88 (0.69−1.14)	1.12 (0.88−1.43)
15−25 years	4967 (23.8)	0.95 (0.79−1.14)	0.77 (0.62−0.97)	1.06 (0.85−1.33)
>25 years	2266 (15.0)	0.87 (0.68−1.10)	0.73 (0.56−0.94)	0.87 (0.66−1.14)
Restricting to quitters 50 years and older at health survey, N = 8717[c]				
0−2 years	790 (9.1)	1.00 (ref)	1.00 (ref)[b]	1.00 (ref)
3−7 years	1211 (13.9)	1.24 (0.88−1.74)	0.78 (0.57−1.08)	1.22 (0.85−1.75)
8−14 years	1253 (14.4)	0.90 (0.63−1.29)	0.83 (0.61−1.14)	1.19 (0.83−1.71)
15−25 years	3309 (38.0)	1.02 (0.76−1.39)	0.75 (0.57−0.98)	1.08 (0.78−1.49)
>25 years	2148 (24.7)	0.93 (0.67−1.29)	0.70 (0.53−0.94)	0.92 (0.65−1.30)

[a] Wald 8.200, df = 4, p = .085.
[b] Wald 6.616, df = 4, p = .158.
[c] About 2491 of the 17,623 quitters were excluded due to missing responses on question on time of cessation. In the secondary analysis of quitters > = 50 years of age, 1366 were excluded.

Table 6
Current smoking (yes/no) in relation to anxiety and depression

	Total N/N smokers (%)	Anxiety only	Depression only	Both
Sex, significance of interactions by sex		$\chi^2 = 5.48$, df = 1, $p = 0.019$	$\chi^2 = 0.1$, df = 1, $p = 0.796$	$\chi^2 = 5.2$, df = 1, $p = 0.023$
Male	28,794/2814 (28.5)	1.16 (1.14–1.39)	1.09 (0.98–1.22)	1.46 (1.31–1.64)
Female	32,020/9473 (29.6)	1.47 (1.36–1.58)	1.13 (0.99–1.30)	1.79 1.62–1.97)
Age, significance of interactions by age		$\chi^2 = 22.4$, df = 5, $p < 0.001$	$\chi^2 = 9.6$, df = 5, $p = .088$	$\chi^2 = 847.0$, df = 5, $p < 0.001$
20–29	8523/2286 (26.8)	1.59 (1.23–1.67)	1.08 (0.70–1.67)	2.26 (1.73–2.95)
30–39	11,355/3622 (31.9)	1.46 (1.28–1.65)	1.41 (1.08–1.85)	2.04 (1.70–2.44)
40–49	13,283/4885 (36.8)	1.34 (1.19–1.51)	1.23 (1.02–1.50)	1.53 (1.32–1.77)
50–59	10,774/3341 (31.0)	1.31 (1.13–1.50)	0.94 (0.78–1.13)	1.61 (1.39–1.87)
60–69	8301/2206 (26.6)	1.35 (1.12–1.61)	1.24 (1.03–1.49)	1.48 (1.23–1.78)
70–89	8578/1347 (15.7)	1.07 (0.83–1.38)	0.94 (0.78–1.14)	1.32 (1.06–1.66)

Analyses stratified for age and gender. Odds ratios from logistic regression models with adjustment for demographics.

4.3. The association between smoking and common mental disorder partly depends on the presence of anxiety and is confounded by somatic symptoms

Research on smoking in relation to mental health has predominantly focused on depression and severe psychopathology. More recently, increased attention has been given to anxiety disorders [6]. In contrast to the findings of Glassman [18], we found the association between smoking and depression almost entirely explained by anxiety, and there was little evidence of an association between smoking and depressive symptoms except in the presence of comorbid anxiety symptoms. Previously reported associations between smoking and depression may have been overestimated through not taking the possible impact of comorbid anxiety into account [8,19]. The results also imply that somatic symptoms are the most important confounder in the smoking versus mental health association although it has largely remained out of scope in the existing literature [6,12,17,33]. An alternative, but in our view less likely interpretation of the results, may follow from the choice of measurement: HADS was designed to avoid false positive cases in contexts where somatic symptoms are prevalent by omitting the vegetative symptoms of depression, a feature separating the HADS from other commonly used screening instruments for depression. Applying an instrument including vegetative symptoms of depression in addition to the aspects of cognition and anhedonia covered in HADS might reveal stronger effects for depression and could leave the association between smoking and depression less attenuated when adjusting for anxiety and somatic symptoms.

The stronger association between smoking and comorbid anxiety depression than anxiety or depression alone is probably a result of more pathology in the comorbid group, reflected in e.g. increased help-seeking [43] and disability [37]. It might therefore be regarded an indication of a dose–response association in the association between common mental disorder and smoking.

Comprehensive assessment of confounding factors is particularly important for the association between smoking and mental disorder as variables in both domains are associated with several adverse social and health-related factors [23].

Surprisingly in our analysis, the effects of reported physical disorders known to be strongly linked with smoking had hardly any effect on the association between smoking and depression and anxiety; neither did measures of BMI, cholesterol level or blood pressure. Adjusting for socio-economic factors explained some of the association. This is not surprising, since smoking as well as common mental disorders are known to be more prevalent among low-status segments of the Norwegian population.

Our results suggest that alcohol problems may be an important confounding factor for the association between smoking and anxiety, consistent with previous findings, and possibly indicating a shared dependence mechanism [4]. The confounding effect of alcohol problems is likely to have been underestimated in the present study through selective non-participation or under-reporting, as well as by limitations in the measurement used.

4.4. There is more psychopathology in current than former smokers

Our findings of more mental health problems in current than former smokers are in line with previous reports suggesting a mental health gain from cessation [6,9,27]. Alternatively, psychopathology might preclude cessation, a hypothesis supported by the reported dose–response relationship with psychiatric severity [33]. For instance, depressed individuals have stronger withdrawal symptoms during the days after cessation [42] and it has been argued that nicotine could act as an antidepressant agent which could be a major obstacle to effective cessation among people with depression [14]. For anxiety, a popular belief among smokers is that smoking has a calming effect and acts as a stress-relieving agent [40], a notion that may stem from withdrawal symptoms resembling core symptoms of anxiety disorders.

4.5. There is no association between mental health and time since cessation

A short-term decline in mental health risk after cessation has been reported. Our study, however, attempts to examine

effects of cessation on psychiatric illness in a lifetime perspective. As there are differences in prevalence of anxiety and depression between former and current smokers, we anticipated a decline in anxiety and depression with time since cessation. Despite the large sample size and high level of statistical power in our study, we were not able to identify any such association. This may indicate that the mental health gain from cessation is apparent over a short time frame (possibly within the first 6 months after cessation), and not over a longer period, as is the case for many other health-related consequences of smoking. This conclusion must, however, be drawn with reservations of our cross-sectional design, as well as biases relating memory and cessation.

4.6. Former smokers have a higher risk of mental disorders than never-smokers

The literature on differences in mental health between former smokers and never-smokers is scarce. In this study we found increased prevalence of anxiety and comorbid anxiety and depression in former smokers compared to those who have never smoked, even after adjustment for multiple potential confounders. This finding can be explained in several ways: (i) Increased likelihood of smoking initiation is reported in individuals with mental illness [7,33]. (ii) Further, individuals prone to mental illness might run a higher risk of developing nicotine dependence having experimented with or tried tobacco [7]. (iii) Cessation is reported to impose a short-term risk of development or remission of mental illness [10,19], but this is an unlikely explanation for our finding as most former smokers in our study quit smoking several years ago. (iv) Long-term adverse effects of smoking cannot be ruled out, though we have no empirical indication of this.

4.7. Age and gender

We found smoking's association to anxiety and comorbid anxiety depression to be stronger in young participants and females. There is some inconclusive evidence that common mental disorder precludes cessation [35]. Following this argument, we speculate that the recent increased public champagnes against smoking might be more effective in prevention or cessation of smoking by individuals without particular risk factors like anxiety and depression.

5. Conclusion

Previous studies of associations between depression and smoking might have overestimated the association when ignoring comorbid anxiety and the whole range of relevant confounding factors. The smoking anxiety association is stronger and more robust than that of depression. Our study inspires the hypothesis that anxiety and depression is increased already before onset of smoking, which needs to be tested in a future longitudinal study of a child or adolescent population.

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