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Unemployment and smoking: Causation, selection, or common cause? Evidence from longitudinal data

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Unemployment and Smoking: Causation, Selection, or Common Cause? Evidence from Longitudinal Data.

Reinhard Schunck¹, Benedikt G. Rogge²

ABSTRACT

Background: This study investigates possible mechanisms that can explain the association between unemployment and smoking, that is a) unemployment increases smoking probability (causation), b) smoking increases the probability to become unemployed (selection), and c) differences in both smoking and unemployment probabilities trace back to differences in socio-economic position (common cause).

Methods: Longitudinal data from the German Socio-Economic Panel (SOEP) from the years 1998, 1999, 2001, 2002, 2004, 2006, and 2008 were used to examine the effect of unemployment on smoking probability and vice versa (65,823 observations from 18,735 respondents, aged 18-60 years). Effects were estimated by using random and fixed effects logistic panel regressions.

Results: Results from the random effects logistic regression models suggest that unemployed have a higher probability to smoke and that smokers have a higher probability to become unemployed. However, the fixed effects models indicate that the observed associations are driven by unobserved factors. Results indicate that both smoking and unemployment probability co-vary systematically with (childhood) socio-economic position.

Conclusion: In contrast to previous studies, the present investigation suggests that there is neither a direct causal effect of unemployment on smoking behaviour nor a direct effect of smoking on unemployment probability. Rather, smoking and unemployment seem to be related through a common cause, with people from low socio-economic backgrounds being more likely to smoke as well as to become unemployed. These findings are interpreted in the frame of a life course perspective on the development of socially unequal health behaviours.

JEL: I12, J64

Keywords: Health behaviour, smoking, unemployment, longitudinal analysis, life course, health inequality, fixed effects, random effects

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BACKGROUND

Unemployment constitutes a serious global public health problem, in the current economic crisis more than ever. While health consequences of unemployment are well documented (Bartley, Ferrie and Montgomery 2006; Burgard, Brand and House 2007; Hammarstrom and Janlert 2005; Kroll and Lampert 2011; Thomas, Benzeval and Stansfeld 2005), the relationship between health-related behaviours and unemployment remains under-researched. Smoking is of particular importance, as it is associated with numerous forms of cancer and cardiovascular disease and is by far the strongest preventable risk factor for premature mortality (Ezzati and Lopez 2003; WHO 2009).

Cross-sectional studies reveal that the unemployed are significantly more likely to smoke than the employed and the average population (De Vogli and Santinello 2005; Kasl and Jones 2000; Schunck and Rogge 2010). This association has been interpreted as a selection mechanism from smoking into unemployment (Bartley 1994; Bartley 1996; Elkeles and Seifert 1993; Jusot et al. 2008) as well as a causal effect of unemployment on smoking behaviour (Bartley 1994; Bartley 1996; Elkeles and Seifert 1993; Hammarstrom and Janlert 2003). In contrast to this, what we term here the “common cause hypothesis” assumes a third variable, i.e. socio-economic position, to produce both the increased risk of unemployment and smoking (see figure 1). In unemployment research, by now, the common cause hypothesis has received comparatively little attention (Gilman, Abrams and Buka 2002; Montgomery et al. 1996). Particularly, there is a conspicuous lack of studies simultaneously scrutinising the three hypotheses with adequate data.

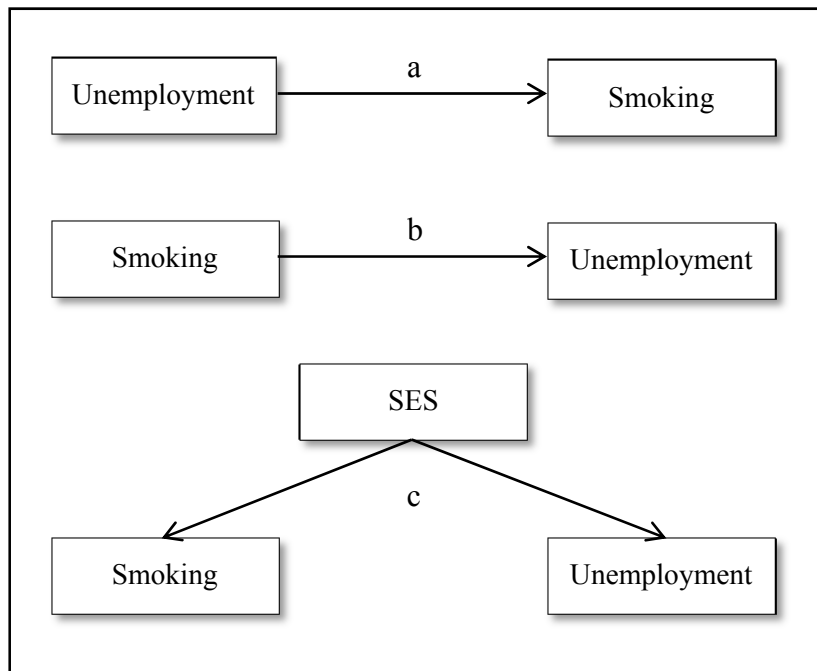


Figure 1: Possible Mechanisms Relating Unemployment and Smoking

Note: a denotes a direct impact of unemployment on smoking (“causation hypothesis”); b denotes a direct impact of smoking on unemployment (“selection hypothesis”); c denotes a situation without any direct association between unemployment and smoking (“common cause hypothesis”).

Cross-sectional studies are unapt to deal with our research issue as they cannot identify the mechanisms which underlie the observed association. The few available longitudinal studies, however, delivered mixed evidence. For Great-Britain (Montgomery et al. 1996), Finland (Leino-Arjas et al. 1999), France (Jusot et al. 2008), and the U.S. (Ryan, Zwerling and Jones 1996) there is evidence for the selection of smokers into unemployment. At the same time, there is evidence that unemployment increases smoking probability for Sweden (Hammarstrom and Janlert 2003), with mixed evidence from the U.S. (Bolton and Rodriguez 2009; Falba et al. 2005; Gallo et al. 2005; Ryan, Zwerling and Jones 1996), Britain (Montgomery et al. 1998; Morris, Cook and Shaper 1992), and Germany (Marcus 2012; Schunck and Rogge 2012) and no evidence for such a relation in Finland (Virtanen et al. 2008) and Denmark (Osler 1995).

Objectives

The above reported differences could stand for country specific effects (Bambra and Eikemo 2009). However, they might also go back to differences in methodology and data use. In our view, any study on the relationship between unemployment and smoking should consider the following aspects: First, it should simultaneously test the three existing hypotheses that are causation hypothesis, selection hypothesis and common cause hypothesis. Second, it needs accurate longitudinal data on unemployment duration and, third, has to distinguish continuous unemployment from discontinuous work biographies. Fourth, longitudinal data call for the application of statistical methods which take full advantage of the data (Baltagi 2005; Rabe-Hesketh and Skrondal 2008; Wooldridge 2010).

Proponents of the causation hypothesis (see figure 1) have predominantly made reference to stress theory to account for health changes after job loss (Pearlin 1989; Thoits 1995; Umberson, Lui and Reczek 2008). They assume that unemployment increases psychological distress and individuals, in order to reduce this distress, draw on potentially health-damaging behaviours (De Vogli and Santinello 2005; Siegrist and Rodel 2006). This lets us put forth the following causation hypothesis: (1) Becoming unemployed increases the likelihood of smoking.

In contrast, the selection hypothesis postulates the reverse causality. It contends that employees who smoke are more likely to become unemployed, because they are (perceived as) on average less productive than non-smokers (Bartley 1994; Bartley 1996; Elkeles and Seifert 1993; Hammarstrom and Janlert 2003; Montgomery et al. 1996). Their lower productivity is suggested to trace back either directly to the negative health effects of smoking or indirectly to employer's assumption that smoking signals personal characteristics that are associated with lower productivity (Heckman, Stixrud and Urzua 2006; Jusot et al. 2008;

Mastekaasa 1996). From this, we derive the selection hypothesis: (2) Smoking increases the probability of becoming unemployed.

The common cause hypothesis draws attention to the role of socio-economic characteristics and childhood experiences (Bartley 1994; Bartley 1996; Kuh and Hardy 2002; McKenzie et al. 2011). The strong social gradient in smoking behaviour is mirrored by a social gradient in the probability of becoming unemployed, as people from lower socio-economic positions smoke more often and also become unemployed more often (Chan and Goldthorpe 2007; Conrad, Flay and Hill 1992; Gilman, Abrams and Buka 2002; Link and Phelan 1996; Montgomery et al. 1996; Phelan, Link and Tehranifar 2010; Umberson, Lui and Reczek 2008). This mechanism is captured by the following common cause hypothesis: (3) Smoking and unemployment are not directly causally connected. Their association traces back to a common cause that is present and past socio-economic position.

We do not argue that the three mechanisms are mutually exclusive. They may operate at the same time. The goal of our study is to test their assumption against each other so as to evaluate the respective explanatory potential of each hypothesis.

METHODS

Data

This study is based on data from the German Socio-Economic Panel (SOEP) (Wagner, Frick and Schupp 2007). The SOEP is an on-going, representative longitudinal survey of German households with currently more than 19,000 individual respondents. It has been conducted annually since first administered in 1984. It covers a wide range of topics, including detailed information on labour market integration, socio-economic position, as well as household and family composition. Respondents remain in the survey until they drop out (Kroh 2010). To ensure continuous representativeness, the SOEP is regularly enlarged with new samples.

The SOEP provides self-reported information on smoking status in the years 1998, 1999, 2001, 2002, 2004, 2006, and 2008. From this information we created a binary variable, indicating whether a person smokes or not. This variable is used both as dependent variable, to test the effect of unemployment (hypothesis (1)), and as independent variable, to test the effect of smoking status on unemployment probability (hypotheses (2)). When unemployment is the main independent variable (hypothesis (1)), we relied on the SOEP's detailed information on employment status on a monthly basis, which is collected annually with regard to the last year. This allowed reconstructing the exact duration of unemployment spells. In order to allow for differential effects of different unemployment durations, we created four binary variables. Short-term unemployment is measured in stages of 1-6 months of unemployment and 7-12 months; long-term unemployment as 1-2 years and 2 years or more.

When unemployment is the dependent variable (hypothesis (2)), we used a lead variable, to ensure the correct temporal ordering between possible cause and effect. Thus, smoking at time t is used to predict unemployment probability at $t + 1$. To test if selection operates on health rather than on smoking, a measure of subjective health (bad, poor, satisfactory, good, very good) and days of sickness absence were included in the models as proxy-variables.

Socio-economic position is assessed through education, coded similarly to the International Standard Classification of Education (OECD 1999), as well as income quintiles, the latter computed annually on the basis of the annual net household income of the whole sample. Additionally, when testing the common cause hypothesis, parental education (highest degree) is included as a proxy measure of the respondent's socio-economic position during childhood.

Further controls are respondents' gender, age, age-squared, employment status (full time, part time and marginal employment, which describes an atypical employment form in

Germany usually with fixed term contracts and low wages (up to 400/800 € per months)), marital status, migration status, the number of persons in the household, number of children in the household, whether or not a respondent lives in East-Germany, as well as year dummies. The analysis sample was restricted to employed and unemployed respondents of working age (18-60 years). All observations with missing values were excluded from the analyses. The data for the analyses are unbalanced within an average of 3.50 observations per respondent.

Statistical Analysis

Since both dependent variables are binary, we computed logistic regression models for each outcome. First, we computed random effect panel models. This model is given by $(Pr = 1|X_{it}) = \text{logit}(\beta_0 + \beta_1 x_{it} + u_i)$. It has considerable advantages over pooled cross-sectional models, as it accounts for clustering of observations within respondents and (time-constant) differences in the dependent variable by adding a person-specific random intercept (u_i). However, the model hinges on the assumption that u_i is a random variable that follows a normal distribution ($u_i \sim N(0, \sigma^2)$) and is uncorrelated with the predictors ($\text{Cov}(x_{it}, u_i) = 0$). If this is not the case, the estimated coefficients are inconsistent, because u_i contains omitted, albeit relevant independent variables (i.e. unobserved heterogeneity or omitted variable bias). Alternatively, u_i can be treated as a fixed quantity, which leads to a fixed effects logistic regression model (also known as conditional logit model or change score model). In the two period case it is given by $\text{Pr}(y_{i1} = 0, y_{i2} = 1 | y_{i1} + y_{i2} = 1) = \text{logit}(\beta_1(x_{i2} - x_{i1}))$. This model uses only variation within respondents that is it estimates how a within-person change in the independent variable is associated with a within-person change in the dependent variable. Hence, it uses only individual observations which experience a change both in the dependent variable and in the independent variable, which reduces sample size and efficiency. However, the advantage of these models is that their estimates are not susceptible to bias due

to omitted time-constant confounders (Baltagi 2005; Giesselmann and Windzio 2012; Rabe-Hesketh and Skrondal 2008; Wooldridge 2010). From the perspective of fixed effects models, the independent variable can be thought of as resembling a treatment effect, with each case serving as its own control.

Hausman tests were computed to investigate if possible differences in effect estimates from random and fixed effects models stem from the fixed effects models' lower efficiency or if they indicate a possible bias in the random effects models' estimates.

Since change in an independent variable is necessary to estimate a variable's effect, time-constant predictors cannot be included in the analysis. Fixed effects models are therefore only sensible if the predictors are time-varying. While this is the case with smoking and unemployment, it is not the case with (childhood) socio-economic position. Thus, when it comes to estimate the effect of socio-economic position on smoking and unemployment probability, we had to rely on random effect models.

The logistic regression models that predict unemployment probability can also be interpreted from the perspective of a discrete time survival analysis (Box-Steffensmeier and Jones 2004: 58; Edelen, Tucker and Ellickson 2007). We control for time-dependency of the process by including job tenure (in years) as well as the number of times a person partakes in the SOEP. The analyses were carried out using Stata 12.1.

RESULTS

Table 1 displays the descriptive statistics for the variables investigated in this study. Column 2 presents descriptive statistics for all observations (person-years: $N \times t$). 53.40% of the 65,823 observations come from men. The overall average age is 41.43 years. During the observation period 7.87% of the sample members experienced unemployment and 36.68% report to smoke.

Table 1: Descriptive statistics on smoking and demographic characteristics of multivariate sample

	Observations (N x t) = 65,823		Observations, employed (N x t) = 60,645		Observations, unemployed (N x t) = 5,178	
	Mean / %	SD	Mean / %	SD	Mean / %	SD
Gender	53.40%		53.57%		51.37%	
Age	41.43	10.29	41.30	10.09	42.94	12.25
Smoking	36.68%		35.47%		50.95%	
Subjective health	3.57	0.86	3.60	0.83	3.21	1.01
Sickness absence (days)	8.39	22.89	8.10	21.91		
Income						
1. quintile	14.04%		11.47%		44.11%	
2. quintile	17.91%		17.38%		24.10%	
3. quintile	21.43%		21.94%		15.49%	
4. quintile	23.26%		24.41%		9.73%	
5. quintile	23.36%		24.80%		6.57%	
Education						
Inadequately completed / general elementary	12.21%		11.22%		23.74%	
Lower secondary	49.74%		48.92%		59.33%	
Upper secondary	15.09%		15.68%		8.13%	
Higher education	22.97%		24.18%		8.81%	
Employment						
Full-time	70.72%		76.76%			
Part-time	16.50%		17.91%			
Marginal	4.91%		5.33%			
Unemployed	7.87%					
Average length of unemployment spell(months)					19.86	21.73

Source: SOEP 1998-2008, own computations, unweighted.

Note: income quintiles are not evenly distributed, as their computation is based on the whole SOEP sample.

Columns 3 and 4 report the well-known cross-sectional association between unemployment and smoking, as smoking is reported more often (50.95%) amongst unemployed than amongst employed respondents (35.47%). Looking at education and the

income distribution, the data also show the familiar association between unemployment and socio-economic position.

Table 2 presents the results of the logistic regression models on smoking behaviour. The random effects model shows that there is a strong and highly significant association between unemployment and smoking probability (model 1: OR_{1-6months} 1.42, 95% CI 1.06-1.88; OR_{7-12months} 1.50, 95% CI 1.04-2.17; OR_{1-2years} 1.53, 95% CI 1.08-2.18; OR_{2years and more} 1.62, 95% CI 1.12-2.36). The differences between the estimated effects of different unemployment durations are significant, indicating that longer unemployment spells are associated with an increased smoking probability.

Table 2: Logistic regression for smoking probability, random and fixed effects

	Model 1: random effects	Model 2: fixed effects
Employed (full-time)	ref.	ref.
Unemployed: 1 to 6 months	1.42* [1.06,1.88]	0.88 [0.65,1.20]
Unemployed: 7 to 12 months	1.50* [1.04,2.17]	0.86 [0.59,1.26]
Unemployed: 1 to 2 years	1.53* [1.08,2.18]	0.83 [0.56,1.21]
Unemployed: 2 years and longer	1.62* [1.12,2.36]	0.73 [0.49,1.10]
Persons	18,735	2,727
Observations	65,823	12,952

OR; 95% confidence intervals in brackets

Source: SOEP 1998-2008, own computations.

Models also control for education (ISCED), income (quintiles), employment status (part-time/marginal), gender, marital status, number of persons in household, number of children in household, migration background, living in East-Germany, period (year), and number of observations per person.

Hausman test (Chi²(1)=347.76) indicates that estimates from random effects model are inconsistent.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

However, a Hausman test, used to check for consistency of random versus fixed effects (see table 2), indicates that the random effects model's estimates may be biased by unobserved heterogeneity. The fixed effects model suggests that the relation between unemployment and smoking probability is not causal (model 2: $OR_{1-6months}$ 0.88, 95% CI 0.65-1.20; $OR_{7-12months}$ 0.86, 95% CI 0.59-1.26; $OR_{1-2years}$ 0.83, 95% CI 0.56-1.21; $OR_{2years\ and\ more}$ 0.73, 95% CI 0.49-1.10). Apparently, the observable differences between unemployed and employed respondents with regard to smoking probability appear to trace back to systematic yet unobserved time-constant differences between these groups. Thus, the results do not support hypothesis (1).

The selection hypothesis (2) suggests that smokers have a higher probability of being selected into unemployment than non-smokers. Effect estimates from the random effects models (table 3) indicate that respondents who smoke indeed have a higher probability of becoming unemployed (model 1: $OR_{smoking}$ 1.67, 95% CI 1.46-1.91). The association remains significant (model 2: $OR_{smoking}$ 1.57, 95% CI 1.37-1.80) even after controlling for subjective health and days absent from work due to ill health – both of which are significant predictors of becoming unemployed. It seems as if respondents who smoke have a higher probability of becoming unemployed, regardless of potential differences in health. A Hausman test (see table 3), however, suggests that the random effects models' estimates are inconsistent. The fixed effects models indicate that this association, again, is not causal (model 3: $OR_{smoking}$ 1.14, 95% CI 0.76-1.71; model 4: $OR_{smoking}$ 1.11, 95% CI 0.74-1.68). Thus, while there are significant differences between smokers and non-smokers in their probability of becoming unemployed, a change in individual smoking behaviour is not associated with a change in unemployment probability.

Table 3: Logistic regression for unemployment probability (t+1) random and fixed effects models

	Model 1: random effects	Model 2: random effects (incl. health)	Model 3: fixed effects	Model 4: fixed effects (incl. health)
Smoking: yes	1.67*** [1.46,1.91]	1.57*** [1.37,1.80]	1.14 [0.76,1.71]	1.11 [0.74,1.68]
Subjective health		0.81*** [0.75,0.88]		0.81** [0.70,0.93]
Number of sick days		1.01*** [1.01,1.01]		1.01*** [1.00,1.01]
Persons	16,031	16,031	900	900
Observations	52,507	52,507	3,262	3,262

OR; 95% confidence intervals in brackets

Source: SOEP 1998-2008, own computations.

Models also control for education (ISCED), income (quintiles), economic sector, tenure, gender, marital status, employment status (part-time/marginal), number of persons in household, number of children in household, migration background, living in East-Germany, period (year), and number of observations per person.

Hausman test ($\text{Chi}^2(1)=347.76$) indicates that estimates from random effects model (1) are inconsistent.

Hausman test ($\text{Chi}^2(1)=369.64$) indicates that estimates from random effects model (2) are inconsistent.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

This indicates that smoking itself does not increase unemployment probability, but that both smoking and unemployment trace back to unobserved, time-constant common causes that differentiate smokers from non-smokers. Thus, the results do not seem to support hypothesis (2).

Table 4: Logistic regression for smoking and unemployment probability (t+1) random effects models

	Model 1: Smoking, random effects	Model 2: Unemployment, random effects
Smoking: yes		1.56 ^{***} [1.37,1.79]
Subjective health		0.81 ^{***} [0.75,0.88]
Number of sick days		1.01 ^{***} [1.01,1.01]
Employed (full-time)	ref.	
Unemployed: 1 to 6 months	1.41 [*] [1.06,1.87]	
Unemployed: 7 to 12 months	1.49 [*] [1.03,2.14]	
Unemployed: 1 to 2 years	1.52 [*] [1.07,2.15]	
Unemployed: 2 years and longer	1.60 [*] [1.10,2.32]	
Education:		
Inadequately / general elementary	ref.	ref.
Lower secondary	0.57 ^{***} [0.45,0.72]	0.76 ^{**} [0.63,0.92]
Upper secondary	0.23 ^{***} [0.17,0.31]	0.45 ^{***} [0.34,0.59]
Higher education	0.07 ^{***}	0.40 ^{***}

	[0.05,0.09]	[0.30,0.52]
Income:		
1. quintile	1.59 ^{***} [1.32,1.92]	2.53 ^{***} [2.04,3.14]
2. quintile	1.16 [*] [1.01,1.34]	1.40 ^{***} [1.17,1.68]
3. quintile	ref.	ref.
4. quintile	0.84 [*] [0.73,0.96]	0.64 ^{***} [0.53,0.78]
5. quintile	0.77 ^{**} [0.65,0.92]	0.44 ^{***} [0.35,0.55]
Highest education parents:		
Don't know	3.03 [*] [1.06,8.67]	1.63 [*] [1.05,2.53]
Inadequately / general elementary	ref.	ref.
Lower secondary	0.55 ^{***} [0.39,0.77]	0.98 [0.81,1.19]
Upper secondary	0.56 [*] [0.34,0.92]	1.06 [0.77,1.45]
Higher education	0.34 ^{***} [0.22,0.51]	1.03 [0.78,1.35]
Persons	18,735	16,031
Observations	65,823	52,507

OR; 95% confidence intervals in brackets

Source: SOEP 1998-2008, own computations.

Models also control for education (ISCED), income (quintiles), gender, marital status, employment status (part-time/marginal), number of persons in household, number of children in household, migration background, living in East-Germany, period (year), and number of observations per person. Model 2 additionally controls for tenure and economic sector.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

This brings us to the common cause hypothesis (3). The random effects models in table 4 show that both smoking and unemployment probability co-vary significantly with socio-economic position. The lower a respondent's socio-economic position, the higher the probability both to smoke (model 1) and to become unemployed (model 2). Moreover,

parental socio-economic position is a significant predictor of an individual's smoking probability (model 1). The higher the parents' education, the less likely is a respondent to smoke. What is more, respondents who do not know their parents' educational degree have elevated smoking as well as unemployment probabilities (models 1 and 2). Overall, the results thus lend support to the common cause hypothesis (3).

DISCUSSION

This study had two objectives: To deliver longitudinal evidence on the relationship between unemployment and smoking in Germany and to provide a test of different hypotheses which could explain the well-established cross-sectional association. Our study investigated three hypotheses, the causation hypothesis, the selection hypothesis, and the common cause hypothesis. As the analyses have shown – at least in the German context – unemployment and smoking do not appear to be linked through a direct causal relationship. First, the analyses did not provide evidence that becoming unemployed increases one's probability to smoke. Second, there is little evidence for the selection hypothesis. Once time-constant differences between respondents were controlled, smoking did not turn out as a significant predictor for becoming unemployed. However, if both smoking and unemployment probability is driven – to some extent – by unobserved (personality) differences that relate to employability and productivity, then the association between smoking and unemployment is spurious and it would indeed disappear once time-constant unobserved heterogeneity is accounted for. Accordingly, the lack of a causal effect of smoking on unemployment probability does not necessarily speak against a modified version of the selection hypothesis, which argues that smoking functions as a proxy measure for personality characteristics (Heckman, Stixrud and Urzua 2006; Jusot et al. 2008; Mastekaasa 1996).

There is, however, support for the common cause hypothesis (Bartley 1996; Gilman, Abrams and Buka 2002; Kuh and Hardy 2002; Montgomery et al. 1996; Umberson, Lui and Reczek 2008). Socio-economic position is significantly associated with both the probability to smoke and the probability to become unemployed. We also find lasting effects of childhood socio-economic position even though adult socio-economic position is controlled for, with parental education being a significant predictor for smoking. It is worth mentioning that respondents, who do not seem to know what educational degree their parents have, have a higher probability to smoke and to become unemployed. Not knowing about one's parents' education may be a (crude) proxy for adverse experiences during childhood, such as family or parental conflict, which is reported to predict smoking (Anda et al. 1999; Isohanni, Moilanen and Rantakallio 1991). How do unemployment and smoking then relate through a common cause? Low childhood socio-economic position goes along with a greater exposure to unfavourable circumstances over the life course, which may, then, lead to low educational attainment, a low socio-economic position, as well as (personality) characteristics, which independently influence both smoking probability and unemployment probability (Bartley, Blane and Montgomery 1997; Gilman et al. 2008; Heckman, Stixrud and Urzua 2006; Jusot et al. 2008; Kuh et al. 2003; McKenzie et al. 2011; Montgomery et al. 1996; Umberson, Lui and Reczek 2008; Virtanen et al. 2005). Considering that persons usually start to smoke in adolescence or early adulthood (Edelen, Tucker and Ellickson 2007), it is likely that social and economic circumstances in earlier life stages are particularly important, as they structure health behaviours, later life experiences, labour market opportunities and health (Cockerham 2007; Kuh et al. 2003; Kuh and Hardy 2002; McKenzie et al. 2011; Pollitt, Rose and Kaufman 2005).

However, one should note that the estimated associations between unemployment and smoking remain significant despite controlling for (childhood) socio-economic position. This

indicates that there is considerable heterogeneity within SES-groups regarding smoking behaviour and unemployment probability despite the strong differences in smoking behaviour and unemployment between SES-groups. Moreover, the estimated effects from the random effects models should be treated with care; they are likely biased by time-constant unobserved heterogeneity. Thus, there is need to better understand the concrete mechanisms, which generate the socio-economically structured link between smoking and unemployment and which can subsequently be tested directly.

Our study thus opens up some questions as the well documented cross-sectional association between smoking and unemployment disappears once adequate techniques of longitudinal data analysis are applied. Yet, it is at odds with the findings of various earlier studies (Hammarstrom and Janlert 2003; Jusot et al. 2008; Leino-Arjas et al. 1999; Montgomery et al. 1996; Montgomery et al. 1998; Ryan, Zwerling and Jones 1996). How is this divergence to be explained?

First, it might go back to methodological reasons as, to our knowledge, no other study has yet implemented fixed effects models to this research issue (for an exception see: Schunck and Rogge 2012). Second, unemployment and smoking could be linked in a more complex way than previously assumed, that is causal effects could be masked by unaccounted mediators (Kessler, Turner and House 1988; Kokko and Pulkkinen 1998) or might depend on past trajectories of health behaviours (Deb et al. 2011). Third, it is possible that the results we found are country specific, as different welfare state arrangements have been shown to produce diverging health outcomes in the unemployed (Bambra and Eikemo 2009).

These reflections are tentative. In the future more studies from other national contexts are needed to clarify if the common cause hypothesis that we have suggested to explain the association of smoking and unemployment in Germany also applies to other countries. Equally important, however, are studies which investigate the emergence of health behaviours

over the life course and their connection to other life outcomes, such as, for instance, educational attainment and labour market outcomes. Showing, as in this study, that (childhood) socio-economic position significantly co-varies with health behaviours and labour market outcomes is not sufficient. It is necessary to spell out and test the concrete mechanisms that are responsible for this association (Cherlin, Chase-Lansdale and McRae 1998; Hammarstrom, Stenlund and Janlert 2011; Thoits 2010; Wadsworth 1997). This means that we would have to put stronger emphasis on less proximate causes, as life course research promotes (Elder and Giele 2009; Kuh et al. 2003; Wadsworth 1997). A life course perspective, we hold, can integrate both the differential vulnerability and the differential exposure hypothesis of health research (Thoits 2010).

To avoid misinterpretations, we should stress that our findings do not challenge the fact that unemployment is a serious public health problem. Quite the reverse is true. Even if unemployment and smoking may not be causally related, unemployment may still be causally linked with negative health outcomes (Paul and Moser 2009). However, the analyses suggest a common cause between unemployment and smoking and thus point to what some scholars have denoted as a “fundamental cause” of health inequalities, that is the inequality of socio-economic positions (Link and Phelan 1996; Phelan, Link and Tehranifar 2010). The common cause explanation has important policy implications. It points to the prevalent need to reduce social inequalities in general to improve health in socially disadvantaged populations groups and calls into question the enduring effectiveness of intervention programs targeting unemployed individuals. Intervening earlier in the life course, before disadvantages have accumulated, may be most effective.

Limitations

The findings of our study have some limitations. Drawing causal inference from observational, even longitudinal, data is difficult. A word of caution also seems in place

regarding fixed effects models. Their estimation method relies exclusively on within-subject variation. As in our sample there are comparably few subjects who changed their status in both the dependent and independent variables, the observed lack of significant effects needs to be confirmed in studies with larger data. Moreover, fixed effects models only appear sensible if the dependent variable can be thought of as resembling a treatment. With regard to the experience of unemployment and smoking status, this seems unproblematic. However, as argued above, the application of fixed effects models is impossible if we are interested in estimating the effect of time-constant characteristics, such as childhood socio-economic position. Thus, until we are able to specify the concrete mechanisms, which produce the association between socio-economic position and smoking and which can be operationalized via time-varying constructs, the reported associations should be treated with great care. These associations should not be seen as causal, as long as the underlying mechanisms are not investigated. Furthermore, we had to rely on self-reports on smoking. Self-report data can be distorted due to item non-response and respondents' possible sensitivity to social desirability. We cannot rule out such impairments of data reliability. Yet, a potential underestimation of smoking behaviour would only bias our analyses in a relevant way if the item non-response or false reporting differed systematically between unemployed and employed respondents. However, there is no reason to assume this, considering that item non-response rates on smoking behaviour are exceptionally low and do not differ substantively between respondents who are full-time, part-time, marginally employed, or unemployed, ranging between 0.09% and 0.29%.

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