

Work-related stress, inability to relax after work and risk of adult asthma: a population-based cohort study

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Abstract

Background: There is an extensive literature linking stressful work conditions to adverse health outcomes. Notwithstanding, the relationship with asthma has not been examined, although various other measures of psychological stress have been associated with asthma. Therefore, we aimed to investigate the relation between work stress and asthma prevalence and incidence.

Methods: We used data from a population-based cohort study ($n = 5114$ at baseline in 1992–1995 and $n = 4010$ at follow-up in 2002/2003). Asthma was measured by self-reports. Two scales that assessed psychologically adverse work conditions were extracted from a list of work-condition items by factor analysis (these scales were termed 'work stress' and 'inability to relax after work'). For each scale, the derived score was employed both as continuous z -score and as categorized variable in analyses. Associations with asthma were estimated by prevalence ratios (PRs) and risk ratios (RRs) using Poisson regression with a log-link function adjusting for demographics, health-related lifestyles, body mass index and family history of asthma. Analyses were restricted to those in employment ($n = 3341$).

Results: Work stress and inability to relax z -scores were positively associated with asthma prevalence (PR = 1.15, 95%CI = 0.97, 1.36 and PR = 1.43, 95%CI = 1.12, 1.83, respectively). Prospective analyses using z -scores showed that for each 1 standard deviation increase in work stress and inability to relax, the risk of asthma increased by approximately 40% (RR for work stress = 1.46, 95%CI = 1.06, 2.00; RR for inability to relax = 1.39, 95%CI = 1.01, 1.91). Similar patterns of associations were observed in analyses of categorized exposures.

Conclusions: This is the first study to show a cross-sectional and longitudinal association of work stress with asthma.

Psychosocial stress has been linked to the prognosis and incidence of atopic diseases (1). Examples of psychosocial factors that have been related to adult asthma include depression, anxiety, stressful life events, and stress-related personality traits (2–6). It would therefore seem plausible that work-related psychological stress may be associated with asthma as well.

Work stress is widespread in European and US workforces. For instance, about one out of four European workers reports having to work at very high speed all or almost all the time (7). Likewise, every fourth worker in the

United States reports being often or very often burned out or stressed by his or her job (8). Stressful work conditions have been linked to a range of adverse health outcomes including the metabolic syndrome (9), cardiovascular disease (10), diabetes (11), depression (12) and anxiety disorders (13).

Although the link between certain types of occupations (e.g., nurses) or occupational exposures (e.g., various chemicals) and asthma is well established (14), to the best of our knowledge the association between psychological work conditions and asthma has not yet been investigated. We therefore aimed to explore associations between work stress and

asthma prevalence as well as asthma incidence in a population-based cohort study of adults.

Methods

Study population

Between 1992 and 1995, a total of 5114 women and men aged 40–65 from Heidelberg and surroundings, filled out a questionnaire collecting medical and psychological information. In 2002/2003, after a median of 8.5 years, this population-based sample was followed up using a similar questionnaire (15). Among those alive at follow-up ($n = 4857$), 83% ($n = 4010$) completed and returned the follow-up questionnaire. Study protocols were approved by the ethics committee of the Medical faculty of the University of Heidelberg. For the current investigation, we restricted our study population to those reporting to be in full-time employment ($n = 2396$) or part-time employment ($n = 945$) at baseline.

Work stress

The baseline questionnaire included 22 items aiming to assess work conditions. The introductory question was: 'Which of the following work conditions places, or has placed, a strain on you during your current or last job?'. The subsequent items focused on various aspects that included hazardous exposures (e.g., radioactivity, heat), demanding work schedules (e.g., shift work) and social interactions at the work place (e.g., competition with colleagues). Participants responded to each item using a 4-point Likert scale ('not applicable'; 'puts absolutely no strain on me'; 'puts a strain on me to some degree'; 'puts a strong strain on me'). Four additional items assessed how people usually feel at the end of their workday. Respondents were asked to indicate on a 4-point Likert scale ('never', 'rarely', 'sometimes', 'often') how often they experienced work-related rumination, felt emotionally unbalanced, felt exhausted, and felt unable to cope with their work demands.

Factor analyses were conducted to further validate the grouping of these items in distinct subscales. We randomly split the sample into two equally sized subsamples and ran separate factor analyses. As these factor analyses revealed identical factor structures in both subsamples, we here present the results from factor analysis ran in the entire sample. We conducted factor analysis (in the entire sample and in the two subsamples) of the 26 items from both scales using varimax rotation. Based on the Kaiser's criterion (16), a six-factor solution was extracted. However, as the eigenvalue of the sixth factor only marginally exceeded 1 (< 1.06 in all samples), we repeated the factor analyses requesting a five-factor solution, which was also supported by the screeplot. To interpret the extracted factors, we defined variables with factor loadings > 0.5 , which is considered a substantial correlation (17), as markers for the specific factor. Two of these factors primarily comprised items on shift and piecework or on physical job demands (see Table 1). The three other factors focused on psychosocial features of the

working environment, including perceived psychophysiological effects of a strenuous workday. As we were interested in work conditions closely related to psychological stress, we focused only on those latter three factors. The Cronbach's α for these three factors was acceptable for two factors, which we labeled as 'work stress' (factor III in Table 1) and 'inability to relax after work' (factor V in Table 1) (Cronbach's α was 0.70 and 0.66, respectively). The third psychosocial factor (factor IV in Table 1) exhibited low internal consistence (Cronbach's $\alpha = 0.47$) and was therefore not used in further analyses. Items comprising each factor were combined into an unweighted mean score. Both summary scores had a potential range between 0 and 3, with higher scores indicating higher work stress or greater inability to relax. Only summary scores of participants without missing item values were included (the work stress score and the inability to relax score could be calculated for 94.3% and 98.7% of the sample, respectively). In alternative analysis of the work stress measure, we merged the response categories 'not applicable' and 'puts absolutely no strain on me' when calculating the work stress summary score (both were scored as 0). This alternative analysis did however not reveal different results for the work stress-asthma association.

Asthma

At baseline, participants were asked whether they currently have or ever had asthma. Response categories were: 'I currently have asthma', 'I do not have asthma anymore', 'I do not know whether I still have it', and 'No, I do not have asthma/I have never had asthma'. Prevalent asthma at baseline was defined as a participant responding 'I currently have asthma', 'I do not have asthma anymore' or 'I do not know whether I still have it'. If the answer 'No, I do not have asthma/I have never had asthma' was given, the individual was considered to be free of asthma. At follow-up, respondents reported whether they had ever been diagnosed with asthma by a physician (yes/no). The format of the asthma item changed between baseline and follow-up to harmonize questions on many incident diseases as assessed at follow-up. We defined the cumulative incidence of asthma over the follow-up period as no asthma at baseline and reporting asthma at follow-up.

Statistical analyses

Work stress scores and inability to relax scores were employed both as continuous and as categorized variables in analyses. For analyses with continuous measures, we used z -transformed scores. Such analyses provide more statistical power than analyses with categorized exposures. For analyses of categorized variables, participants were grouped into three (cross-sectional analyses) or two (prospective analyses) equally sized groups. In cross-sectional analyses, the significance of a potential trend of asthma prevalence across the three exposure categories was assessed by entering the tertiles as an ordinal variable in statistical models. The decision to employ two rather than three categories in prospective analyses was because of the low incidence of asthma leading to

Table 1 Factor analysis of items characterizing working conditions (rotated component matrix)

Items	Extracted factors				
	I	II	Factors related to psychological work conditions		
			III	IV	V
Over hours, long working hours					
Exclusively working at night	0.76				
Shift work without night shift	0.76				
Shift work with night shift	0.76				
Piece work	0.69				
Radioactivity	0.58				
Working at assembly line	0.73				
Noise		0.59			
Chemical contaminates		0.59			
Heat, cold, moisture		0.75			
Physically heavy work		0.69			
Unpleasant and monotonous physical strain and posture		0.60			
High work pace, time pressure					
Contradictory requirements					
Frequent interruptions			0.54		
Forced to make fast decisions			0.73		
High responsibility for machines					
High responsibility for people			0.73		
Strong competition with colleagues			0.57		
Working alone, talks with colleagues are not possible				0.57	
Boring, monotonous work				0.69	
My performance is closely monitored					
Feels dissatisfied and underemployed at the end of the work day					0.77
Feels tired and exhausted at the end of the work day					0.60
Cannot stop thinking about work at the end of the work day					0.77
Feels at the end of the work day overchallenged by work					

Only factor loadings of >0.5 are shown as these items were included in the corresponding factor.

reduced statistical power. Associations between work stress, inability to relax and prevalent or incident asthma were estimated by prevalence ratios (PRs) and risk ratios (RRs) together with their 95% confidence intervals (95% CIs). These estimates were modeled based on Poisson regression with a log-link function and the empirical (robust) variance (18). We decided *a priori* to control our estimates for age, sex, education, smoking status, alcohol consumption, body mass index (BMI), physical exercise and family history of asthma.

In cross-sectional analyses, we used conventional methods to adjust for confounding. In contrast, in longitudinal analyses we applied propensity scores to control for confounding (19). This method was chosen because of the limited number of incident cases in our study and because of the relatively high proportion of exposed participants (because of the use of two exposure groups of similar size). Propensity score adjustment was done by first estimating the propensity for each study participant to be exposed to each dichotomized exposure. We estimated the individual probability of exposure by logistic regression employing the dichotomized work stress variable (or inability to relax after work variable) as dependent variable and the possible confounders as independent variables. In a second step, participants who had a

propensity score outside of the range common to exposed and unexposed individuals were excluded from the analyses. Third, the continuous propensity scores were divided into quintiles. These quintiles were then used as dummy variables in the multivariable outcome models for asthma incidence.

Results

Out of the 3341 participants employed at baseline, 3206 provided complete information on asthma of whom 179 (5.6%) reported prevalent asthma. Those reporting to be free of asthma ($n = 3027$) represent the cohort for our longitudinal analyses. Of these, 87 (2.9%) deceased between baseline and follow-up (mortality follow-up was 100% complete). Of those alive at follow-up ($n = 2940$), 2475 (84.2%) participated in the follow-up assessments, of whom 2463 (99.5%) provided information on asthma. Among this group, 47 participants (1.9%) reported asthma at follow-up (the cumulative incidence over a median follow-up of 8.5 years).

The mean work stress score was 1.17 (SD = 0.76, range = 0.00–3.00). The mean inability to relax after work score was 1.69 (SD = 0.62, range = 0.00–3.00). As Table 2 shows, men reported higher levels of work stress than

Table 2 Work stress and inability to relax after work by baseline characteristics

Characteristic	Work stress*			Inability to relax after work†		
	Low	Medium	High	Low	Medium	High
<i>n</i>	1124 <i>n</i> (%)	1078 <i>n</i> (%)	950 <i>n</i> (%)	1308 <i>n</i> (%)	1235 <i>n</i> (%)	754 <i>n</i> (%)
Age (years)						
<50	509 (45.28)	448 (41.56)	430 (45.26)	584 (44.65)	526 (42.59)	317 (42.04)
50 to <60	534 (47.51)	550 (51.02)	455 (47.89)	624 (47.71)	615 (49.80)	390 (51.72)
≥60	81 (7.21)	80 (7.42)	65 (6.84)	100 (7.65)	94 (7.61)	47 (6.23)
Sex						
Men	421 (37.46)	635 (58.91)	632 (66.53)	760 (58.10)	657 (53.20)	356 (47.21)
Women	703 (62.54)	443 (41.09)	318 (33.47)	548 (41.90)	578 (46.80)	398 (52.79)
Education (years)						
<10	527 (48.30)	442 (42.46)	356 (38.57)	582 (45.47)	494 (41.41)	342 (47.17)
10	262 (24.01)	220 (21.13)	163 (17.66)	262 (20.47)	259 (21.71)	136 (18.76)
>10	302 (27.68)	379 (36.41)	404 (43.77)	436 (34.06)	440 (36.88)	247 (34.07)
Smoking status						
Never	482 (43.15)	421 (39.16)	327 (34.68)	487 (37.40)	505 (41.12)	295 (39.39)
Former	375 (33.57)	380 (35.35)	385 (40.83)	495 (38.02)	421 (34.28)	277 (36.98)
Current	260 (23.28)	274 (25.49)	231 (24.50)	320 (24.58)	302 (24.59)	177 (23.63)
Alcohol consumption (g/day)						
None	202 (18.26)	144 (13.45)	111 (11.80)	179 (13.82)	178 (14.55)	120 (16.19)
0.1–15.0	495 (44.76)	409 (38.19)	361 (38.36)	520 (40.15)	496 (40.56)	318 (42.91)
15.1–30.0	235 (21.25)	287 (26.80)	239 (25.40)	317 (24.48)	296 (24.20)	175 (23.62)
>30.0	174 (15.73)	231 (21.57)	230 (24.44)	279 (21.54)	253 (20.69)	128 (17.27)
Physical exercise, (h/week)						
None	303 (27.03)	279 (26.05)	216 (22.81)	291 (22.35)	322 (26.16)	228 (30.36)
>0 to 2	583 (52.01)	542 (50.61)	541 (57.13)	659 (50.61)	681 (55.32)	400 (53.26)
>2	235 (20.96)	250 (23.34)	190 (20.06)	352 (27.04)	228 (18.52)	123 (16.38)
Body mass index (kg/m ²)						
<25	633 (57.29)	553 (52.02)	492 (52.23)	715 (55.43)	655 (53.82)	381 (50.94)
25 to <30	378 (34.21)	416 (39.13)	369 (39.17)	476 (36.90)	445 (36.57)	292 (39.04)
≥30	94 (8.51)	94 (8.84)	81 (8.60)	99 (7.67)	117 (9.61)	75 (10.03)
Family history of asthma						
No	991 (88.96)	955 (90.26)	832 (88.42)	1153 (89.31)	1088 (89.40)	660 (88.59)
Yes	123 (11.04)	103 (9.74)	109 (11.58)	138 (10.69)	129 (10.60)	85 (11.41)

*Three groups were constructed approximating similar size as closely as possible. Work stress score ranges were low 0 to <1.0; medium ≥1.0 to <1.75; high >1.75.

†Three groups were constructed approximating similar size as closely as possible. Inability to relax after work score ranges were low 0 to ≤1.34; medium >1.34 to ≤2.0; high >2.0.

women. Further, educational levels, former smoking (but not current smoking), alcohol consumption, and overweight (but not obesity) appeared to be positively associated with work stress. Women indicated higher inability to relax after work than men. Physical exercise was inversely related with inability to relax after work. The prevalence of obesity tended to increase with increasing inability to relax.

Cross-sectional analyses of the work stress *z*-score (Table 3) showed that asthma prevalence increased by 15% with every 1 SD work stress increase (PR = 1.15, 95%CI = 0.97, 1.36). Using categories of work stress, the prevalence of asthma was nonsignificantly elevated in those

with high (PR = 1.36, 95%CI = 0.92, 2.03), but not in those with medium levels of work stress (PR = 1.03, 95%CI = 0.69, 1.54), both compared with low work stress. Regarding inability to relax after work, the corresponding *z*-score showed a positive association with asthma prevalence (PR = 1.43, 95%CI = 1.12, 1.83). Further, contrasted with those in the lowest tertile, a higher prevalence of asthma was observed in those with medium and high scores (indicating a lower ability to relax after work) (PR = 1.45, 95%CI = 1.00, 2.09, and PR = 1.65, 95%CI = 1.11, 2.44), along with a statistically significant trend across tertiles ($P < 0.01$).

Table 3 Prevalence ratios (PRs) and 95% confidence intervals (95%CI) for asthma prevalence according to work stress and inability to relax after work

	<i>n</i> (% with prevalent asthma)	Age-and-sex adjusted*		Multivariable†	
		PR	95%CI	PR	95%CI
Work stress					
Low	1082 (4.7)	1.00	Ref	1.00	Ref
Medium	1037 (4.7)	0.98	0.66, 1.45	1.03	0.69, 1.54
High	918 (7.0)	1.45	0.98, 2.12	1.36	0.92, 2.03
		<i>P</i> trend = 0.06		<i>P</i> trend = 0.13	
Z-score	–	1.17	0.99, 1.37	1.15	0.97, 1.36
Inability to relax after work					
Low	1256 (4.1)	1.00	Ref	1.00	Ref
Medium	1185 (5.9)	1.46	1.03, 2.08	1.45	1.00‡, 2.09
High	727 (7.3)	1.84	1.27, 2.67	1.65	1.11, 2.44
		<i>P</i> trend = <0.01		<i>P</i> trend = <0.01	
Z-score	–	1.52	1.19, 1.93	1.43	1.12, 1.83

*Analyses adjusted for age (continuous and squared) and sex.

†Analyses adjusted for age (continuous and squared), sex, education (<10, 10, >10 years), smoking status (never, former, current), alcohol consumption (0, 0.1–15.0, 15.1–30.0, >30.0 g/day), physical exercise (0, >0–2, >2 h/week), body mass index (<25, 25 to <30, ≥30 kg/m²), family history of asthma (yes, no).

‡*P* = 0.0495

Table 4 Risk ratios (RRs) and 95% confidence intervals (95%CI) for asthma incidence according to work stress and inability to relax after work

	<i>n</i> (% with incident asthma)	Age-and-sex adjusted*		Multivariable†	
		RR	95%CI	RR	95%CI
Work stress					
Low	1118 (1.3)	1.00	Ref	1.00	Ref
High	1220 (2.4)	2.02	1.10, 3.69	2.30	1.16, 4.54
Z-score	–	1.36	1.04, 1.80	1.46	1.06, 2.00
Inability to relax after work					
Low	1488 (1.5)	1.00	Ref	1.00	Ref
High	946 (2.6)	1.77	1.00‡, 3.10	2.11	1.15, 3.85
Z-score	–	1.32	0.99, 1.75	1.39	1.01, 1.91

*Analyses adjusted for age (continuous and squared) and sex.

†Analyses adjusted for age (continuous and squared), sex, education (<10, 10, >10 years), smoking status (never, former, current), alcohol consumption (0, 0.1–15.0, 15.1–30.0, >30.0 g/day), physical exercise (0, >0–2, >2 h/week), body mass index (<25, 25 to <30, ≥30 kg/m²), family history of asthma (yes, no). For categorized variables, propensity score adjustment was employed.

‡*P* = 0.0465

Prospective analyses (Table 4) showed that work stress and inability to relax after work were both associated with an increased risk of asthma. In both exposures, a 1 SD increase was related to an approximately 40% increased risk of asthma (RR for work stress = 1.46, 95%CI = 1.06, 2.00 and RR for inability to relax = 1.39, 95%CI = 1.01, 1.91). Using dichotomized variables, we found that risk of asthma was approximately doubled in those indicating high work stress as contrasted with those reporting low work stress (RR = 2.30, 95%CI = 1.16, 4.54). A similar association was

found for high vs low inability to relax after work (RR = 2.11, 95%CI = 1.15, 3.85).

To further explore associations, multivariable models estimating RRs for z-scores were submitted to three additional analyses. First, one may hypothesize that the association between work stress and asthma is mediated by lifestyle-related factors. When we omitted BMI, physical activity, smoking, and alcohol consumption from the multivariable models, we observed slightly attenuated estimates (RR for the work stress z-score = 1.33, 95%CI = 0.99, 1.79, RR

for inability to relax z -score = 1.30, 95%CI = 0.96, 1.76). The direction of changes observed in RRs induced by models without lifestyle variables seems to suggest that lifestyle-related factors are confounders (which need to be controlled for) rather than mediating factors.

Secondly, we examined to what extent physical exposures encountered in the work environment are able to account for the observed associations. Therefore, multivariable models estimating RRs for z -scores were additionally adjusted for such physical work characteristics. We used a variable which we had employed previously (2) combining information on exposure to chemicals or heat, cold, or moisture and/or working in an asthma-risk profession. Additional correction for this variable altered the estimates only marginally (resulting RR for the work stress z -score = 1.45, 95%CI = 1.05, 2.00; RR for the inability to relax z -score 1.39, 95%CI = 1.01, 1.90).

A final sensitivity analysis focused on depression. Depression could, at least in part, account for the observed associations. Additionally including depressive symptoms (which were measured by a validated scale (20) and included as a z -score) in multivariable models marginally attenuated both the association between the work stress z -score and incident asthma (RR = 1.39, 95%CI = 1.00, 1.91 vs RR = 1.46, 95%CI = 1.06, 2.00) and the association between the inability to relax after work z -score and asthma incidence (RR = 1.30, 95%CI = 0.92, 1.82 vs RR = 1.39, 95%CI = 1.01, 1.91).

Discussion

The present study investigated the cross-sectional and longitudinal association between psychosocial work conditions (work stress and inability to relax after work) and asthma. Cross-sectional analyses showed that work stress and inability to relax after work were associated with a weakly elevated prevalence of asthma. Longitudinal analyses showed stronger associations: for 1 SD increase in each of these parameters, roughly a 40% increased risk of asthma was observed. Our finding of positive associations between psychologically adverse work conditions and adult asthma is consistent with other reports (1, 3, 5, 6), including findings from our group (2, 4), linking various aspects of stress-exposure (e.g., stress-related personality traits, life events, depressive symptoms, and social support) to asthma incidence.

The major strengths of our study are the use of a large population-based sample, the use of a cohort design, and the high response rate at follow-up. These features give confidence with regard to the reasonable control of selection and information bias as well as the generalizability of our findings. The generalizability may, however, strictly be limited to the age range of the working population included in this study (40–65 years at baseline). Workers of younger age (<40 years) may be less selected for good health yet, which could lead to stronger associations between worker stress, inability to relax after work and asthma in such samples. We could not draw on a previously validated scale to measure psychologically adverse work conditions. We used factor

analysis to cluster items into largely independent factors. The items that were used to measure 'work stress' resemble items contained in other validated work stress questionnaires (21–23). Given the acceptable internal consistency of the scale, we conclude that our work stress measure combines related facets of psychological work stress. The three-item measure of 'inability to relax' likewise had adequate internal consistency. Again, items or constructs similar to our individual items contained in this scale have been employed in other questionnaires (21, 24–26).

Measurement of asthma was based on self-reports. However, we may add that there is no gold standard for diagnosing asthma (27) and even in clinical settings there is frequent disagreement (28). We used slightly differently worded questions to assess asthma at baseline and follow-up (self-reported asthma vs physician-diagnosed asthma). Among the different questions which can be used to assess asthma in surveys, these two items are among those with the highest specificity (94% and 99%, respectively when using bronchial challenges tests as the comparative asthma definition) (27). A specificity close to 1.0 is sufficient to minimize bias in measures of relative risk in epidemiological studies (29). Because of its lower specificity, the baseline question used to assess asthma in our study has probably identified a higher proportion of false-positives than the follow-up question. However, all baseline asthma cases were excluded from prospective analyses of incident asthma. Thus, it seems unlikely that the somewhat lower specificity of the baseline asthma question could have biased our longitudinal analyses.

We were able to take into account many important confounders in our analyses. Nevertheless, residual confounding as well as unmeasured or unknown confounding can never be ruled out as an alternative explanation.

What underlying mechanisms might account for our findings? We hypothesized that adverse psychosocial work conditions may lead to unhealthy lifestyles, which may, in turn, increase the risk of asthma. However, our analyses did not support mediation by health lifestyle-related factors. This inability to demonstrate a mediating effect might be explained by the rather weak associations between psychosocial work conditions and lifestyles in our study (see Table 1), which is in line with observations from other studies (30). Further sensitivity analyses suggested that the detected associations are also not explained by characteristics of the physical work environment. Additional analyses addressing the role of depression indicated that a limited part of the association between work stress, inability to relax after work and asthma incidence may be attributable to depressive symptoms. These concepts are probably slightly overlapping. The marginal attenuation of the multivariable estimates by additional control for depressive symptoms illustrates the robustness of our findings.

Another set of explanations for our findings pertains to direct immunological effects of work stress. There is an extensive literature showing that stressors can dysregulate neuro-endocrine and immunological processes relevant to the inflammatory responses in asthma (31, 32). Examples are enhanced release of inflammation-modulating neuropeptides

such as substance P, enhanced secretion of the allergy-promoting type-2 cytokines (e.g., IL-4, IL-5), greater delayed-type hypersensitivity responses and increased airway inflammation after allergen exposure (31, 32). Significantly, markers of job stress, such as effort–reward imbalance and overcommitment, have been linked to patterns of dysregulated neuroendocrine and inflammatory activity that resembles observations made in patients with asthma and other atopic diseases. Examples are a blunted cortisol response to acute stressors (33, 34), lowered vagal/parasympathetic tone (35) and elevated inflammatory activity (31, 32, 36). Research into possible psychobiological mechanisms linking job strain and asthma incidence appears warranted therefore.

These immunological mechanisms may explain how psychosocial work conditions impact asthma morbidity in patients with asthma, although it remains unclear how such processes may affect asthma onset in initially asthma-free individuals. Possibly, psychologically adverse work conditions promote the expression of asthma in individuals that have an increased preposition to develop asthma, but showed no clinical symptoms at baseline. For example, in many people immunological indicators of atopic activity can be observed (e.g., elevated immunoglobulin E levels or positive skin prick tests), which is indicative of an increased allergic sensitivity, without concomitantly expression of symptoms that warrant a clinical diagnosis (37).

When interpreting our findings from prospective analyses, one needs to bear in mind the difference between relative risks and absolute risks. The absolute risk of developing asthma is low in adults (e.g., only 1.9% across 8.5 years in our study population). Therefore, our findings of elevated relative risks of asthma among people with high work stress and pronounced inability to relax after work do not have major public health implications. Even assuming causality, if levels of work stress were reduced, the absolute number of asthma cases, which would be prevented, remains low. However, our findings do add to our understanding of the etiology of asthma and highlight the relevance of psychological stress.

In conclusion, presenting the first epidemiological study to date on the relation between psychologically adverse working conditions and the development of asthma, our investigation suggests that work stress and the inability to relax after work are associated with an increased risk of asthma. Additional large prospective studies on psychosocial work conditions in relation to risk of asthma are needed to replicate our findings and to explore the potential role of inflammation.

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