**The longitudinal relationship of work stress with peak expiratory flow: a cohort study**

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**Acknowledgements:** This paper uses data from SHARE waves 2 and 4 (DOIs: 10.6103/SHARE.w2.500, and 10.6103/SHARE.w4.500). The SHARE data collection has been primarily funded by the European Commission through FP5 (QLK6-CT-2001-00360), FP6 (SHARE-I3: RII-CT-2006-062193, COMPARE: CIT5-CT-2005-028857, SHARELIFE: CIT4-CT-2006-028812) and FP7 (SHARE-PREP: N°211909, SHARE-LEAP: N°227822, SHARE M4: N°261982). Additional funding from the German Ministry of Education and Research, the U.S. National Institute on Aging (U01\_AG09740-13S2, P01\_AG005842, P01\_AG08291, P30\_AG12815, R21\_AG025169, Y1-AG-4553-01, IAG\_BSR06-11, OGHA\_04-064) and from various national funding sources is gratefully acknowledged (see www.share-project.org). The funders had no involvement in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the article for publication.

**ABSTRACT**

**Purpose:** Research has suggested that psychological stress is associated with reduced lung function and with the development of respiratory disease. Among the major potential sources of stress in adulthood are working conditions. We aimed to examine the relationship of work stress with lung function.

**Methods**: We drew on four-year prospective data from the Survey of Health, Ageing and Retirement in Europe. The analyzed sample comprised 2,627 workers aged 50 years or older who were anamnestically free of respiratory disease. Work stress at baseline was operationalized by abbreviated instruments measuring the well-established effort-reward imbalance model (seven items) and the control component of the job-demand control (two items). Peak expiratory flow (PEF) was determined at baseline and at follow-up. Continuous and categorized (i.e. by the tertile) work stress variables were employed in multivariable linear regression models to predict PEF change.

**Results**: Work stress did not show statistically significant associations with PEF change. For instance, the unstandardized regression coefficient for PEF decline according to high versus low effort-reward imbalance was -1.41 (95% confidence interval= -3.75, 0.94).

**Conclusions:** Our study is the first to examine prospective relationships between work stress and PEF. Overall, we did not observe meaninful associations. Future studies should consider a broader spectrum of spirometric parameters and should expand research to younger and possibly less selected working populations (i.e. aged < 50 years).

**Keywords:** Cohort study, Effort-reward-imbalance, Job strain, Lung function, Peak flow, The Survey of Health, Ageing and Retirement in Europe

**INTRODUCTION**

Psychosocial factors, such as stress and poor mental health, have been associated with impaired lung function ([Ochs-Balcom et al. 2013](#_ENREF_17); [Ritz et al. 2015](#_ENREF_25)) and respiratory disease, including asthma ([Douwes et al. 2011](#_ENREF_6)) and chronic-obstructive pulmonary disease (COPD) ([Clark et al. 2015](#_ENREF_5); [Ng et al. 2009](#_ENREF_16)). A recent observational study among university students found, for instance, that the forced expiratory volume in one second (FEV1) decreased throughout academic examination periods ([Ritz et al. 2015](#_ENREF_25)). The psychobiological explanations put forward for such relationships mainly highlight autonomic and ventilatory changes, such as hyperpnea ([Ritz 2012](#_ENREF_24)).

While, among students, academic exams represent a major potential source of stress, working conditions may be similar and likewise common stressors among populations in employment ([American Psychological Association 2015](#_ENREF_1)). Accordingly, a handful of prior studies have examined whether work stress is predictive of respiratory disease. While, overall, the number of studies remains limited, some prospective epidemiological investigations, including work from our group, have confirmed positive associations of work stressors with asthma ([Lietzen et al. 2011](#_ENREF_12); [Loerbroks et al. 2014](#_ENREF_13); [Loerbroks et al. 2010](#_ENREF_15)) and COPD ([Clark et al. 2015](#_ENREF_5)). Yet, other studies contributed inconclusive evidence ([Heikkila et al. 2014a](#_ENREF_9); [Heikkila et al. 2014b](#_ENREF_10); [Renzaho et al. 2014](#_ENREF_23)).

In order to operationalize incident respiratory outcomes, prior studies relied mainly on two types of approaches: first, participants’ reports of manifest disease ([Loerbroks et al. 2014](#_ENREF_13); [Loerbroks et al. 2010](#_ENREF_15); [Renzaho et al. 2014](#_ENREF_23)) and, second, on registry data ([Clark et al. 2015](#_ENREF_5); [Heikkila et al. 2014a](#_ENREF_9); [Heikkila et al. 2014b](#_ENREF_10); [Lietzen et al. 2011](#_ENREF_12)) which mostly covered hospitalizations for or death due to respiratory disease. One may assume that self-reports of respiratory conditions have limited validity. By contrast, registries of respiratory events provide more objective data. The significance of such data is limited however, as hospital admission are rare and considered to be largely avoidable by appropriate primary care ([OECD 2015](#_ENREF_18)).

In light of these limitations, utilization of lung function data may make novel contributions to research on work stress and respiratory outcomes: Lung function, as indicated by spirometric markers such as the peak expiratory flow (PEF), is an objectively assessed outcome and provides more detailed data on respiratory health than hospital admissions or death due to respiratory disease. Yet, spirometric measures have not yet been considered in relation to work stress, despite calls for such analyses ([Hartmann et al. 2017](#_ENREF_8); [Heikkila et al. 2014a](#_ENREF_9)). We therefore aimed to address this knowledge gap.

**METHODS**

**Study population**

We used data from the Survey of Health, Ageing and Retirement in Europe (SHARE). SHARE is a multidisciplinary and multi-country cohort study which addresses health, social factors and economic conditions among Europeans aged 50 and above ([Börsch-Supan and Jürges 2005](#_ENREF_3)). Ethical approval for SHARE was obtained from the institutional review board at the University of Mannheim, Germany. The baseline assessment (wave 1) was carried out in 2004/2005 among representative samples in eleven European countries. At study onset, the overall response rate equalled 61% and ranged from 39% in Switzerland to 81% in France ([Börsch-Supan and Jürges 2005](#_ENREF_3)). Five follow-up assessments were performed during which the initial samples were replenished while additional national samples were included. At all SHARE waves, interviews served as primary tools for data collection. PEF was measured only at wave 2 (2005/2007) and at wave 4 (2010/2011) and we used the data from those two waves as the respective baseline and follow-up for our study. Overall, there were 36,277 study participants at wave 2 and PEF data was available for 31,810 of those (88%). The mean duration between both assessments was 4.26 years ([Borsch-Supan et al. 2013](#_ENREF_2)). Our longitudinal data set for analyses included a total of twelve countries (Austria, Belgium, Czech Republic, Denmark, France, Germany, Italy, the Netherlands, Poland, Spain, Sweden, and Switzerland). Among the SHARE wave 2 participants (n=36,277) a total of 18,405 also participated in wave 4. We restricted our analyses to participants who were in employment at both wave 2 and wave 4 (n=3,001), because retirement during follow-up would imply that the work stress exposures is removed. Accordingly, retirement has been associated with health-related improvements, in particular in those with prior work stress exposure ([Westerlund et al. 2009](#_ENREF_32)). In addition, to limit confounding due to manifest respiratory disease, we excluded individuals who reported physician-diagnosed asthma (n=92), COPD (n=47) or both conditions (n=11). Upon removal of those with missing values (n=224), a sample of 2,627 individuals was available for our analyses.

**Work stress**

The two dominant theoretical models in epidemiological work stress research are the effort-reward imbalance (ERI) model and the job-demand control (JDC) model. Briefly, the key assumption of the ERI model ([Siegrist 1996](#_ENREF_27)) is that work stress is experienced if efforts exceed the received rewards. By contrast, the JDC model hypothesizes that work stress is experienced when individuals are concurrently exposed to high demands and low job control ([Karasek and Theorell 1990](#_ENREF_11)). Abbreviated versions of the original ERI and JDC questionnaires were used in SHARE ([Siegrist et al. 2012](#_ENREF_28)). For the present study, we drew on the work stress data collected at wave 2. Effort was measured by two items capturing perceptions of time pressure and high physical demands. Rewards were assessed by five items addressing, amongst others, to what extent respondents perceived that they receive adequate support, recognition, and a fair salary. In SHARE, the assessment of the JDC model was limited to the control component, which was measured by two items inquiring after one’s freedom to decide how to do one’s work and the opportunity to acquire new skills. All items were presented as statements and participants indicated their level of (dis)agreement on a 4-point Likert scale (scored as 1-4). We added the scores across the respective items to construct scales for effort (potential range= 2-8 points), reward (potential range = 5-20), and control (potential range= 2-8). Also, as recommended and widely practiced ([Siegrist et al. 2012](#_ENREF_28)), we estimated the level of ERI by the ratio of effort and reward scores while correcting for the differing number of items. Higher values of the ERI ratio reflected higher levels of work stress.

**Peak expiratory flow**

In SHARE, a short physical assessment battery was used which was completed during face-to-face interviews. This battery included measurement of PEF by a Mini-Wright™ Standard Range Peak Flow Meter with a disposable mouthpiece. Interviewers provided standardized instructions to ensure that expiratory manoeuvers were performed with maximum effort. PEF was determined as the highest value of two expiratory manoeuvres. For all statistical analyses (i.e. the PEF difference and PEF ratio outcome measures, see below), the measured PEF was divided by values based on the European Respiratory Society (ERS) reference equations ([Quanjer et al. 1993](#_ENREF_20)) to obtain the percentage of the predicted PEF (% predicted PEF).

**Statistical analysis**

Linear regression analyses were performed to examine the associations between the work stress components at wave 2 (independent variables) and change of % predicted PEF between wave 2 and wave 4 (dependent variable). Estimates were expressed as unstandardized regression coefficients (b) with corresponding 95% confidence intervals (CIs). Work stress components were used both as continuous variables (z-transformed) and as dichotomized variables. Dichotomization was based on the respective upper tertile of each components, which is a common approach ([Siegrist et al. 2012](#_ENREF_28)) and seems to represent a reasonable trade-off between the intentions to choose a meaningful cut-off versus a cut-off which is statistically efficient. To test the robustness of our findings to different outcome measure operationalizations, we created two different % predicted PEF outcome measures analyses: The first variable was based on the difference between % predicted PEF values of wave 4 and wave 2 (% predicted PEF wave 4 – % predicted PEF wave 2). The second variable considered the ratio of the % predicted PEF values at wave 4 and wave 2, which was multiplied by 100 to express estimates as percentages (% predicted [PEF wave 4 / % predicted PEF wave 2] \* 100) ([Redlich et al. 2014](#_ENREF_22)). We first ran unadjusted model (Model 1), which were subsequently corrected for age and sex (Model 2) and then additionally adjusted for, education, smoking, and physical exercise (Model 3). A final model (Model 4) made additional adjustments for chronic diseases and respiratory symptoms (i.e. self-reported stroke, cardiovascular disease, hypertension, diabetes, breathlessness and coughing). Analyses were carried out in the entire sample, but also stratified by sex.

We conducted various sensitivity analyses to test the robustness of our findings. First, one may suspect that correction of our estimates for age and sex induces statistical overadjustment since the % predicted PEF is derived from reference equations which consider age and sex. We however decided to control for these factors to account for their potential to affect the *association* between work stress and % predicted PEF (rather than affecting merely PEF). We nevertheless carried out additional analyses that omitted age and sex from the multivariate models (i.e., Model 3 and Model 4). Second, we re-ran our primary analyses using outcome measures that built on raw PEF data (i.e., in ml). Third, PEF assessments were preferably carried out in standing position in SHARE (as opposed to sitting or lying position). As the examination position may affect PEF performance, we repeated our primary analyses restricted to those tested in standing position (n=1,943).

**RESULTS**

Table 1 shows baseline characteristics of the study participants. The distribution of sexes was fairly balanced and, on average, participants were aged in their mid-50s. The majority of participants reported to have intermediate or high educational levels. More than half were former or current smokers and three quarters of the participants engaged into physical exercise. Respiratory symptoms, stroke, cardiovascular disease and diabetes were rare, but hypertension was common. In light of the potential score ranges, work-related efforts were at intermediate levels while rewards and control were rather low. Peak flow showed little change throughout the follow-up.

We observed no consistent pattern of associations between work stress and the decline of % predicted PEF (Table 2). The magnitude of associations was weak and the corresponding estimates were not statistically significant. For instance, the % predicted PEF showed a non-significant decline (i.e. difference) in those with high ERI as compared to low ERI (b= -1.41, 95% CI= -3.75, 0.94). Similar observations were made for low versus high control (b= -0.55, 95% CI= -3.23, 2.13). We observed no sex-specific patterns (data not shown).

Various sensitivity analyses confirmed the robustness of our observations (data not shown): The multivariate models (i.e., Models 3 and 4) suggested virtually identical associations when age and sex were omitted. Likewise, when we re-ran our primary analyses using PEF outcome measures that were constructed based on raw PEF data (ml), comparable patterns of associations were observed. Finally, analyses restricted to those with PEF assessments in standing position yielded similar findings..

**DISCUSSION**

The associations between work stress and PEF observed in the present study were not statistically significant and not clinically relevant. Overall, this observation emerged with reasonable robustness from our data, that is, regardless of the level of statistical adjustment and the approaches to operationalize work stress or PEF change. Patterns of associations were similar in women and men.

Our original rationale to examine links between work stress and PEF built on findings from different lines of research: There had been evidence that psychological stress is associated with poorer lung function ([Ochs-Balcom et al. 2013](#_ENREF_17); [Ritz et al. 2015](#_ENREF_25)) and with respiratory disease ([Clark et al. 2015](#_ENREF_5); [Douwes et al. 2011](#_ENREF_6); [Ng et al. 2009](#_ENREF_16)). Further, some prospective epidemiological studies ([Clark et al. 2015](#_ENREF_5); [Lietzen et al. 2011](#_ENREF_12); [Loerbroks et al. 2014](#_ENREF_13); [Loerbroks et al. 2010](#_ENREF_15)) had specifically confirmed links between work stress and respiratory disease and the biological plausibility of those findings had received empirical support ([Ritz 2012](#_ENREF_24)). To our knowledge, the present study is the first longitudinal investigation of work stress and lung function. Overall, we did not observe pronounced associations, which is in agreement with findings from several prospective epidemiological studies on work stress and respiratory disease ([Heikkila et al. 2014a](#_ENREF_9); [Heikkila et al. 2014b](#_ENREF_10); [Renzaho et al. 2014](#_ENREF_23)). It may be suspected that the observation of links between work stress and respiratory outcomes primarily emerges from studies that draw on participant-reported outcomes (e.g. self-reported asthma) due to common method variance, but not from studies using more objective outcomes (e.g. registry data). This notion does not receive consistent support though as there have been studies with objective outcomes suggesting positive associations ([Clark et al. 2015](#_ENREF_5); [Lietzen et al. 2011](#_ENREF_12)) as well as prospective studies entirely based on participant-reported data and that showed nil findings ([Renzaho et al. 2014](#_ENREF_23)).

The conclusiveness of our study may be hampered by the fact that spirometric evaluations were solely based on PEF. Compared to other spirometric indices (e.g. FEV1), PEF assessment is usually subject to higher variability ([Global Initiative for Asthma (GINA) 2016](#_ENREF_7)), which may somewhat have limited our study’s ability to detect statistically significant associations. More advanced pulmonary function tests, such as body plethysmography or impulse oscillometry, had possibly been more sensitive and better been able to reveal possible effects of work stress on respiratory function. However, in line with recommendations ([Global Initiative for Asthma (GINA) 2016](#_ENREF_7)) the same type of measurement instrument for PEF determination has been used in SHARE for all respondents at baseline and at follow-up examinations thereby minimizing information bias. Also, it needs to be acknowledged that PEF represents an important health indicator in its own right: PEF predicts poor health in general population samples, (e.g. mortality ([Smith et al. 2013](#_ENREF_29))) and is a particularly useful marker of the health status and physical functioning in older adults with and without respiratory disease ([Roberts and Mapel 2012](#_ENREF_26)). Another drawback of our study may stem from the fact that we used abbreviated work stress questionnaires. While those instruments have repeatedly been used in publications from the SHARE data set ([Siegrist et al. 2012](#_ENREF_28)), we cannot rule out that their validity is lower than that of the full-length instruments. Thirdly, it would be of interest to examine links between work stress and PEF in subsamples with respiratory disease. This holds particularly true for asthma as PEF serves a key role in the management of this condition. Yet, the subsample of workers with asthma seemed too small (n=92) to run adequately powered analyses. Fourthly, we might have made different observations (i.e. of a significant association) if we had been able consider short-term (and possibly transient) effects of acute stress exposures on lung function ([Ritz et al. 2015](#_ENREF_25)). Regrettably, such data (i.e. stress experienced at the time of PEF measurements) was lacking. Finally, we need to acknowledge that we addressed only one single important source of stress in adult life (i.e. working life). There may be other major domains that affect respiratory health independently or in combination with work stress, such as familial stress ([Loerbroks et al. 2017](#_ENREF_14)).

Strengths of the current study are its prospective design, the sample in terms of its size and representativeness, and the ability to account for several important confounders. Also, good participation rates were attained at the follow-up, which generally limits potential selection bias. Nevertheless, as SHARE includes only adults aged 50+, some selection processes may have operated prior to that age, especially among workers with respiratory disease. Research suggests that older workers with the poorest lung function may have been less likely to participate at baseline and/or at follow-up ([Radon et al. 2002](#_ENREF_21); [Spierenburg et al. 2015](#_ENREF_30)). To some extent this may also hold true for our data: among those with good PEF performance (i.e. individuals with a PEF >80%predicted at baseline), 35.4% did not participate at follow-up as compared to 39.0% in those with a PEF ≤80%predicted (age-and-sex-adjusted odds ratio for non-participation according to poor versus good PEF= 1.18, 95% CI=1.06, 1.31). Similar selection processes may have occurred in workers experiencing high work stress ([Paterniti et al. 2002](#_ENREF_19)). As a consequence, potential associations between work stress and decline of lung function may have been underestimated. Finally, as our sample was restricted to workers aged 50+ our findings cannot be generalized to younger workers. We cannot rule out that work stress and PEF show stronger associations in younger and less selected occupational samples. Such observations would be consistent with evidence from studies that suggest stronger links between work stress and cardiovascular health in younger workers than in older workers ([Chandola et al. 2008](#_ENREF_4); [Theorell et al. 1998](#_ENREF_31)).

In conclusion, the present study was the first to examine potential prospective relationships between work stress and PEF. Overall, we did not observe meaningful associations though. Future prospective studies should consider a broader spectrum of spirometric parameters and should expand research efforts to younger workers (i.e. aged < 50 years).

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**Table 1.** Baselin**e** characteristics of study participants (n=2,627 )

|  |  |  |
| --- | --- | --- |
| Characteristics |  |  |
| Sex, n (%) | Male | 1304  | (49.64) |
|  | Female | 1323  | (50.36) |
| Age, mean (SDa) |  | 54.87  | (3.38) |
| Education, n (%) | Low | 622  | (23.68) |
|  | Intermediate | 1078 | (41.04) |
|  | High | 927 | (35.29) |
| Daily smoking, n (%) | Never  | 1195 | (45.5) |
|  | Former  | 815 | (31.02) |
|  | Current  | 617 | (23.49) |
| Physical exercicea, n (%) | Yes | 2022 | (77.0) |
|  | No | 605 | (23.0) |
| Stroke, n (%) | Yes | 12 | (0.46) |
|  | No | 2615 | (99.54) |
| Cardiovascular disease, n (%) | Yes | 82 | (3.12) |
|  | No | 2545 | (96.88) |
| Hypertension, n (%) | Yes | 525 | (19.98) |
|  | No | 2102 | (80.02) |
| Diabetes, n (%) | Yes | 93 | (3.54) |
|  | No | 2534 | (96.46) |
| Breathlessness, n (%) | Yes | 82 | (3.12) |
|  | No | 2545 | (96.88) |
| Coughing, n (%) | Yes | 78 | (2.97) |
|  | No | 2549 | (97.03) |
| Effort, mean (SDb) |  | 4.92  | (1.43) |
| Reward, mean (SD) |  | 11.22  | (2.42) |
| ERIc, mean (SD) |  | 0.94  | (0.38) |
| Control, mean (SD) |  | 4.00  | (1.32) |
| Peak Flow difference, mean (SD) |  | 0.18  | (26.48) |
| Peak Flow ratio, mean (SD) |  | 103.05  | (48.04) |

All variables expect for peak flow were assessed based on participants’ self-reports.

a Physicial exercise was defined based on participants’ self-reports to carry out vigorous sports or activities at least one to three times a month.

b SD = standard deviation

c ERI = effort reward imbalance

**Table 2**. Longitudinal associations between work stress and peak flow (i.e., % predicted) in individuals free of respiratory disease at baseline (n=2,627, linear regression)

|  |  |  |
| --- | --- | --- |
|  |  | Peak flow difference |
|  |  | Model 1a | Model 2b | Model 3c | Model 4d |
| Continuous z-scores | High Effort  | -0.59 (-1.61, 0.43) | -0.58 (-1.60,0.44) | -0.71 (-1.76, 0.33) | -0.69 (-1.74, 0.35) |
|  | Low Reward  | -0.48 (-1.50, 0.53) | -0.50 (-1.52,0.52) | -0.50 (-1.53, 0.53) | -0.55 (-1.58, 0.48) |
|  | High ERIe | -0.72 (-1.73, 0.30) | -0.72 (-1.74,0.30) | -0.82 (-1.86, 0.23) | -0.83 (-1.88, 0.21) |
|  | Low control | -0.44 (-1.46, 0.58) | -0.46 (-1.49,0.57) | -0.51 (-1.58, 0.55) | -0.51 (-1.58, 0.55) |
| Dichotomized by the tertile |  |  |  |  |  |
|  | Effort (high vs low)  | 0.64 (-1.75, 3.02) | 0.61 (-1.77, 3.00) | 0.48 (-1.95, 2.90) | 0.46 (-1.97, 2.88) |
|  | Reward (low vs high) | -0.33 (-2.63, 1.97) | -0.36 (-2.66, 1.94) | -0.32 (-2.64, 2.00) | -0.43 (-2.75, 1.89) |
|  | ERI (high vs low) | -1.08 (-3.36, 1.21) | -1.10 (-3.40, 1.19) | -1.26 (-3.60, 1.08) | -1.41 (-3.75, 0.94) |
|  | Control (low vs high) | -0.31 (-2.95, 2.32) | -0.31 (-2.95, 2.33) | -0.37 (-3.05, 2.31) | -0.55 (-3.23, 2.13) |
|  |  |  | Peak flow ratio (\*100) |  |
|  |  | Model 1a | Model 2b | Model 3c | Model 4d |
| Continuous z-scores | High Effort  | -0.17 (-2.07, 1.72) | -0.10 (-2.00, 1.79) | -0.36 (-2.30, 1.57) | -0.33 (-2.26, 1.61) |
|  | Low Reward  | -0.53 (-2.42, 1.35) | -0.49 (-2.38, 1.40) | -0.67 (-2.57, 1.23) | -0.69 (-2.60, 1.21) |
|  | High ERIe | -0.34 (-2.23, 1.55) | -0.27 (-2.16, 1.63) | -0.54 (-2.47, 1.39) | -0.54 (-2.47, 1.40) |
|  | Low control | 1.08 (-0.82, 2.97) | 1.21 (-0.70, 3.12) | 0.65 (-1.33, 2.62) | 0.68 (-1.29, 2.66) |
|  |  |  |  |  |  |
| Dichotomized by the tertile | Effort (high vs low)  | 0.06 (-4.37, 4.48) | 0.08 (-4.35, 4.50) | -0.13 (-4.62, 4.36) | -0.14 (-4.64, 4.36) |
|  | Reward (low vs high) | 0.31 (-3.95, 4.57) | 0.41 (-3.85, 4.68) | 0.16 (-4.13, 4.45) | 0.07 (-4.24, 4.37) |
|  | ERI (high vs low) | -0.64 (-4.89, 3.60) | -0.50 (-4.76, 3.75) | -1.11 (-5.44, 3.22) | -1.23 (-5.59, 3.12) |
|  | Control (low vs high) | 3.12 (-1.77, 8.00) | 3.30 (-1.60, 8.19) | 2.41 (-2.55, 7.37) | 2.23 (-2.74, 7.20) |

This table shows unstandardized regression coefficients with 95% confidence intervals in brackets.

a Unadjusted

b Adjusted for age and sex

c Adjusted for age, sex education, smoking and physical exercise

dAdjusted for age, sex, education, smoking and physical exercise, stroke, coronary heart disease, hypertension, diabetes, breathlessness, cough

eERI = effort reward imbalance

**Compliance with Ethical Standards**

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflict of Interest: The authors declare that they have no conflict of interest.

**Informed consent:** Informed consent was obtained from all individual participants included in the study.