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Occupational exposures and COPD: an ecological analysis of international data

P.D. Blanc*, A.M.B. Menezes[#], E. Plana[¶,+,§], D.M. Mannino[ƒ], P.C. Hallal[#], K. Toren, M.D. Eisner* and J-P. Zock[¶,+,§]**

ABSTRACT: The occupational contribution to chronic obstructive pulmonary disease (COPD) has yet to be put in a global perspective. In the present study, an ecological approach to this question was used, analysing group-level data from 90 sex-specific strata from 45 sites of the Burden of Obstructive Lung Disease study, the Latin American Project for the Investigation of Obstructive Lung Disease and the European Community Respiratory Health Survey follow-up. These data were used to study the association between occupational exposures and COPD Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage II or above.

Regression analysis of the sex-specific group-level prevalence rates of COPD at each site against the prevalence of occupational exposure and ever-smoking was performed, taking into account mean smoking pack-yrs and mean age by site, sex, study cohort and sample size.

For the entire data set, the prevalence of exposures predicted COPD prevalence (0.8% increase in COPD prevalence per 10% increase in exposure prevalence). By comparison, for every 10% increase in the proportion of the ever-smoking population, the prevalence of COPD GOLD stage II or above increased by 1.3%. Given the observed median population COPD prevalence of 3.4%, the model predicted that a 20% relative reduction in the disease burden (*i.e.* to a COPD prevalence of 2.7%) could be achieved by a 5.4% reduction in overall smoking rates or an 8.8% reduction in the prevalence of occupational exposures. When the data set was analysed by sex-specific site data, among males, the occupational effect was a 0.8% COPD prevalence increase per 10% change in exposure prevalence; among females, a 1.0% increase in COPD per 10% change in exposure prevalence was observed.

Within the limitations of an ecological analysis, these findings support a worldwide association between dusty trades and chronic obstructive pulmonary disease for both females and males, placing this within the context of the dominant role of cigarette smoking in chronic obstructive pulmonary disease causation.

KEYWORDS: Chronic obstructive pulmonary disease, ecological, occupation, work-related

Multiple studies of occupation and chronic obstructive pulmonary disease (COPD) risk at the individual level have addressed the association between work-related exposure and disease. These investigations have consistently observed a smoking-adjusted excess risk of COPD associated with occupational exposures (*e.g.* vapours, gas, dust or fumes) [1, 2]. As critical as such individual-level studies are, they do not capture population-level effects that may be important contributors to exposure prevalence and disease risk. For example, a high prevalence of individually reported workplace exposures may indicate generally poor employment conditions even for those who may not report it, leading to imprecision of personal-level estimates of the workplace contribution to COPD and more accurate measures in grouped data. Moreover, since any intervention to improve working conditions is likely occur at the group level (*i.e.* through regulation), examining exposure and disease across groups can provide key insights for public health policy makers.

For these reasons, the current authors intentionally undertook an ''ecological'' approach to this question, analysing morbidity at the population level in order to gain a better understanding of the role that workplace factors play in the burden of COPD internationally. Two recent population-based studies have addressed the prevalence of COPD on an international scale, focusing on the primary role of cigarette smoking as the dominant individual-level risk factor for disease, although occupational exposure was considered as a potential covariate [3, 4]. A third international

AFFILIATIONS
*Division of Occupational and Environmental Medicine, University of California San Francisco, San Francisco, CA,
[ƒ]University of Kentucky, Lexington, KY, USA,
[#]Federal University of Pelotas, Pelotas, Brazil.
[¶]Centre for Research in Environmental Epidemiology (CREAL),
[+]Municipal Institute of Medical Research (IMIM-Hospital del Mar),
[§]CIBER Epidemiología y Salud Pública (CIBERESP), Barcelona, Spain.
**Dept of Occupational and Environmental Medicine, SahIgrenska University Hospital, Gothenburg, Sweden.

CORRESPONDENCE
P.D. Blanc
Division of Occupational and Environmental Medicine
University of California San Francisco
350 Parnassus Ave.
Suite 609
San Francisco
CA 94117
USA
Fax: 1 4154766426
E-mail: paul.blanc@ucsf.edu

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study has been principally concerned with asthma risk in adults, including the individual-level effects of occupational exposures [5, 6]. Although a relatively young cohort, follow-up has extended into an age range where COPD end-points could also be studied [7, 8].

In the present study, grouped data from all three of these studies combined together were used to assess the ecological associations among the prevalence rates for occupational exposures, cigarette smoking and COPD. The current authors wished to test whether the frequency of adverse working conditions predicted COPD prevalence rates at a population level, taking into account group-level smoking data, thus providing a global context for potential public health interventions.

METHODS

Overview

The current study is an ecological analysis using group-level data from three separate cohort studies of COPD involving 45 different sites, each yielding results stratified by sex. Thus, 90 separate sets of observations were available for analysis, each providing frequency data at the group level for the independent predictor variables of study interest (occupational and smoking data and mean age of each site by sex) and the prevalence of COPD (the dependent variable). The overall analytic strategy was designed to estimate the association between the population frequency of occupational exposures and prevalence of COPD, taking into account potential clustering by study cohort and the potential confounding effects of smoking and age at the group level, studying these relationships among males and females together and separately.

Study design

Data from three large studies were used, each with international, random population sample designs: the Burden of Obstructive Lung Disease (BOLD) study; the Latin American Project for the Investigation of Obstructive Lung Disease (PLATINO); and the European Community Respiratory Health Survey follow-up study (ECRHS II). Studying occupational factors in COPD was not a primary goal of any of these studies, but each did collect data relevant to this question. Representatives from each study group participated in this analysis.

For the BOLD study, study site-level, sex-stratified data were extracted as they appeared in published tables [3]. For that analysis, the numbers of participants with acceptable spirometry data (total n=8,775) were used as study site weights in lieu of the somewhat larger total study population (n=9,425). For the PLATINO study, study site-level data paralleling tabular data were used as previously published, but further stratified by sex and with additional data on years of occupational exposure [4]. Stratified data were provided by PLATINO study investigators who participated in the current analysis (A.M.B. Menezes and P.C. Hallal). Site-level, sex-stratified data from the ECRHS II were provided by authors of that study (E. Plana and J-P. Zock). Since the ECRHS II recruited a population with a younger age range at baseline, the data set was limited to persons from the population-based sampling frame aged $\geqslant$40 yrs who completed spirometric measurements at follow-up, in order to exclude younger subjects unlikely to have yet manifested COPD even if exposed to risk factors.

Definition of occupational exposure

Occupational exposure was defined using differing but comparable protocols among the studies. For BOLD and PLATINO, occupational exposure was based on self-report of exposure to ''dusty work'' for at least but not limited to 1 yr or more at any point in time [3, 4]. For the ECRHS II analysis, exposure was based on baseline interview (ECRHS I) self-report of ''vapours, gas, dust or fumes'' at the current or any previous job and/or (at ECRHS II follow-up) any interval of employment for at least but not limited to 3 months in an occupation with a high likelihood of exposure to dust (biological or inorganic), or gases or fumes based on a job exposure matrix [6, 8]. Site- and sex-specific durations of adverse working conditions were also analysed for each study, as another measure of occupational exposure.

Definition of COPD

COPD was defined consistently with the Global Initiative for Chronic Obstructive Lung Disease (GOLD) cut-off criteria of stage II or greater disease: a forced expiratory volume in one second (FEV_1) <80% of height-, age- and sex-predicted, and a ratio of FEV_1 to forced vital capacity of <70% [9]. Use of the GOLD stage II cut-off is consistent with recent analysis of international COPD prevalence in the BOLD study [3].

Although all spirometry was performed in a manner consistent with American Thoracic Society criteria, there were differences in how data were obtained in each study. BOLD measured lung function post-bronchodilator using an EasyOne spirometer (ndd Medical Technologies, Andover, MA, USA) and calculated predicted FEV_1 values using equations for white males and females from the third US National Health and Nutrition Survey [3]. PLATINO also measured lung function post-bronchodilator using an EasyOne spirometer, but calculated predicted FEV_1 values using internally generated equations [4, 10]. ECRHS II measured pre-bronchodilator lung function at most, but not all, centres using a Biomedin water-sealed bell spirometer (Biomedin, Padova, Italy) and the formulae of QUANJER *et al.* [11] were used to calculate predicted FEV_1 [12].

Statistical analysis

Differences in the site–sex stratum prevalence of occupational exposure, ever-smoking and COPD stage II or greater among the three study cohorts were tested using the Kruskall–Wallis test for nonparametric data; differences in duration of occupational exposure and pack-yrs of smoking were tested by ANOVA. Differences by sex were tested by the Wilcoxon rank sum test or the t-test. The associations among occupational exposure, smoking and COPD prevalence by sex–site stratum were estimated using the Spearman rank correlation.

The ecological associations among occupation, smoking and COPD were tested in mixed-effects multiple linear regression models. Regression diagnostics supported the validity of linear regression modelling. The prevalence of COPD GOLD stage II or greater was the dependent variable. The proportion of the population with occupational exposure and the prevalence of ever-smoking were independent variables. The independent covariates included were sex of the sex-stratified site, mean age

and mean pack-yrs of smoking (for ever-smokers) for each site stratum, and the study cohort (BOLD, ECHRS II, or PLATINO), which was included as a random effect. Parameter estimates were weighted based on the sample size of each stratum. In order to test the potential effect of clustering at the study-site level, the model was also re-estimated adding an additional level for study site as a random effect. These analyses were repeated with stratification by sex. To assess the effect of exposure duration, additional analyses were carried out, adding mean duration of occupational exposure among those exposed (by study site and by sex) to the models previously tested.

In order to evaluate potential cigarette-smoking–occupational interactions at the group level, the feasibility of including a simple cross-product term of smoking and occupational exposure in the models was evaluated. This interaction term was highly collinear with both cigarette smoking (r=0.72) and occupational exposure (r=0.93). Therefore, an alternative approach was taken, dividing the study sites into four categories: above the sex-specific median values for both smoking and occupational exposure (n=18; 20% of 90 observations); above the median for smoking only (n=26; 29%); above the median for occupational exposure (n=24; 27%) and below the median for both (n=22; 24%). A mixed model including the first three categories as indicator variables was tested (with lower occupational exposure and lower smoking being the referent), while including all of the other covariates as previously described.

RESULTS

For the three studies included in the analysis, data were available representing 19,094 study participants (8,627 males and 10,467 females) from 27 countries on six continents. Table 1 presents, for each of the three cohorts and for all subjects combined, study site-specific data for sample size and the mean age of participants, stratified by sex. Overall, subject numbers per site were smaller and participants younger within the ECRHS II cohort (limited in the present analysis to persons aged $\geqslant$40 yrs at follow-up), compared with the BOLD and PLATINO cohorts.

Table 2 provides the distributions of site–sex stratum data for occupational exposure as a percent prevalence, the mean years of exposure among those exposed and ever-smoking (current or past cigarette use) as a percent prevalence, and the mean pack-yrs of smoking among ever-smokers. Among the 90 sex–site strata overall, the median (interquartile range (IQR)) prevalence of occupational exposures in dusty or related adverse job conditions was 41 (29–62)%, with an absolute range of 7–85% (the latter not shown in table). The proportions of exposures by site–sex strata did not differ statistically by cohort (Kruskal–Wallis p=0.23). The mean$\pm$SD duration of occupational exposure among those exposed was 13.0$\pm$4.8 yrs. Duration of exposure did differ overall by cohort (ANOVA p<0.0001); the only statistically significant pairwise comparison was between the BOLD and ECRHS cohorts (difference 4.6%, 95% confidence interval (CI) 2.1–7.1%). Exposure duration was significantly greater at the group level among males than females (mean difference 4.1 yrs, 95% CI 2.3–5.9 yrs).

The median (IQR) prevalence of ever-smokers overall was 61 (50–70)%, with an absolute range of 6–83% among all the site–sex strata. As with occupational exposures, the cigarette smoking prevalence distribution did not differ significantly among the three study cohorts (Kruskal–Wallis p=0.60). Smoking intensity duration among ever-smokers was highest in the BOLD and lowest in the PLATINO cohorts and, in each cohort, lower among females than males. Mean pack-yrs per site–sex stratum did not differ significantly by cohort (Kruskal–Wallis p=0.11).

Table 2 also includes the distribution for proportions of COPD of severity GOLD II or higher for each site–sex stratum. For all 90 site–sex strata, the median (IQR) proportion was 3.4 (1.4–6.8)%. The proportions for COPD were highest in the BOLD study sites and lowest in the ECRHS II data set. There were 15 sex–site strata in the ECRHS II with no COPD GOLD II or above. The differences in COPD proportions differed significantly among the three study cohorts (Kuskal–Wallis p<0.001).

Figure 1 presents the scatter plots and correlations between occupational exposure prevalence and the prevalence of COPD GOLD II or higher for each study cohort separately. Occupational exposure and COPD prevalence were correlated within the BOLD cohort (Spearman r=0.48, p=0.02), the ECRHS II (r=0.26, p=0.06) and the PLATINO study (r=0.63, p=0.05).

TABLE 1 International studies included in analysis: geographic distribution and study size

Study cohort	Subjects n	Nations included n	Study sites n	Subjects per site		Mean age by site yrs	
				M	F	M	F
BOLD	8775	12	12	334 (206–685)	343 (237–435)	56.4 (52.1–58.6)	57.5 (53.4–60.1)
ECRHS II	4648	14	28	70.5 (30–179)	78.5 (35–79)	47.5 (44.9–57.2)	47.2 (45.2–50.2)
PLATINO	5671	5	5	442 (380–474)	632 (558–983)	56.0 (54.9–59.3)	55.5 (55.0–61.0)
All	19094[#]	31[¶]	45[+]	111 (65–324)	108 (72 –334)	48.9 (44.9–59.3)	48.3 (45.2–61.0)

Data are presented as median (range), unless otherwise stated. M: male; F: female; BOLD: Burden of Obstructive Lung Disease; ECRHS II: European Community Respiratory Health Survey follow-up survey; PLATINO: Latin American Project for the Investigation of Obstructive Lung Disease. [#]: data for 8,627 males and 10,467 females are included; [¶]: taking into account duplicated countries between BOLD and ECRHS II (Iceland, Norway, USA and Australia), 27 different countries are included among the three cohorts; [+]: Reykjavik (Iceland) and Bergen (Norway) served as study sites in both BOLD and ECHRS II, so 43 distinct sites are represented.

TABLE 2 Occupational exposure, cigarette smoking and chronic obstructive pulmonary disease (COPD) prevalence by study cohort and sex

Study cohort and stratum	Sites by sex n	Dusty/dirty jobs		Cigarette smoking		COPD ⩾GOLD II
		Prevalence of dirty jobs	Exposure yrs#	Prevalence of ever-smoking	Pack-yrs in smokers	
BOLD						
M	12	48 (39–75)	16.8±3.6	69 (60–80)	27.8±6.8	10.7 (9.3–14.4)
F	12	28.5 (20–35.5)	15.2±4.7	46.5 (36.5–55.5)	19.0±6.1	8.0 (6.0–11.6)
Total	24	38 (28.5–48)	16.0± 4.2	58.5 (46.5–69)	23.4±7.8	9.4 (7.8–13.0)
ECRHS II						
M	28	64.5 (51–78.5)	13.9±2.6	68 (62–74.5)	24.1±6.4	2.5 (0.5–4.1)
F	28	27 (22–37.5)	8.8±4.7	56 (49–62.5)	16.0±4.7	1.6 (0–2.8)
Total	56	44 (27–64.5)	11.4±4.6	62.5 (55–69.5)	20.0±6.9	2.0 (0–3.4)
PLATINO						
M	5	63 (61–64)	17.0±2.3	74 (73–77)	22.5±8.3	5.4 (4.9–6.6)
F	5	40 (33–45)	12.9±2.1	44 (44–50)	14.4±4.2	4.9 (4.5–5.3)
Total	10	46 (40–63)	14.9±3.0	65 (44–74)	18.5±7.5	5.1 (4.5–5.4)
All						
M	45	62 (49–75)	15.0±3.1	70 (63–77)	24.9±6.8	4.1 (2.2–9.1)
F	45	30 (22–39)	11.0±5.3	53 (44–60)	16.6±5.2	2.9 (1.3–5.4)
Total	90	41 (29–62)	13.0±4.8	61 (50–70)	22.8±7.3	3.4 (1.4–6.8)

Data are presented as median (interquartile range) %, or mean±SD, unless otherwise stated. GOLD: Global Initiative for Chronic Obstructive Lung Disease; ⩾ GOLD II: GOLD stage II or greater (forced expiratory volume in one second (FEV_1) <80% of height-, age- and sex-predicted, and a ratio of FEV_1 to forced vital capacity of <70%); BOLD: Burden of Obstructive Lung Disease; M: male; F: female; ECRHS II: European Community Respiratory Health Survey follow-up survey; PLATINO: Latin American Project for the Investigation of Obstructive Lung Disease. #: among those with any exposure.

The correlations between the prevalence of ever-smoking and COPD by strata (not shown in figure) were r=0.58 (p=0.003), r=0.27 (p=0.04) and r=0.55 (p=0.10), respectively. The population prevalence of occupational exposure and cigarette smoking by sex–site stratum proportion was intercorrelated overall (Spearman r=0.49, p<0.001). The intercorrelations were highest in two of the cohorts (BOLD r=0.65, p<0.001; PLATINO r=0.86, p=0.001) and less so in the ECRHS II (r=0.37, p=0.005).

The results of mixed-effects multiple linear regression modelling are presented in table 3. The model including all strata took into account study cohort as a random effect variable and adjusted for the sex, mean age and mean pack-yrs of smoking among ever-smokers in each stratum. Both the proportions of dusty/dirty jobs and of ever-smoking were independently associated with the prevalence of COPD. Based on this ecological estimate, for every 10% increase in the frequency of dusty employment and related exposures, the prevalence of COPD was increased by 0.8%. By comparison, for every 10% increase in the proportion of the ever-smoking population, the prevalence of COPD GOLD stage II or above was increased by 1.3%. Based on the observed IQR for occupational exposure prevalence (33%), the model predicts a 2.6% shift in prevalence of COPD stage II and above, over this range; based on the IQR in smoking prevalence (25%), the model predicts a 3.3% shift in COPD. Put in terms of public health actions, given the observed median population COPD prevalence of 3.4%, the model predicts that a 20% relative reduction in the disease burden (*i.e.* to a COPD prevalence of 2.7%) could be achieved by a 5.4% reduction in overall smoking rates or an 8.8% reduction in the prevalence of occupational exposures.

The mixed multivariate model, as noted, also included sex, age, and pack-yrs of smoking as independent predictors. In this model, compared with males, females had a 3.2% higher predicted prevalence of COPD (p=0.005), while mean age and pack-yrs among smokers were not statistically significant predictors of COPD prevalence. Re-estimating the effect, adding a random term for study site, yielded similar findings for smoking (1.2% increase in COPD per 10% change) and occupation (0.7% increase per 10% change; p<0.05 for both). Owing to the aforementioned intercorrelation between the prevalence of smoking and occupational exposure, the potential impact on the parameter estimate and standard error term for smoking was also examined when the model excluded the occupational exposure variable, comparing this to the final model. Inclusion of occupational exposure had little substantive impact on the smoking term, indicating that collinearity between these two variables was not impacting the performance of the model. An additional model that included mean years of occupational exposure was also tested, and yielded similar results.

Stratifying by sex yielded similar estimates for the sex-specific ecological associations between the occupational exposures and smoking and COPD (table 3). The ecological association between dusty trades and COPD among males was consistent, with a 0.8% increase per 10% change in prevalence; among females the estimate was a 1.0% increase in COPD for every

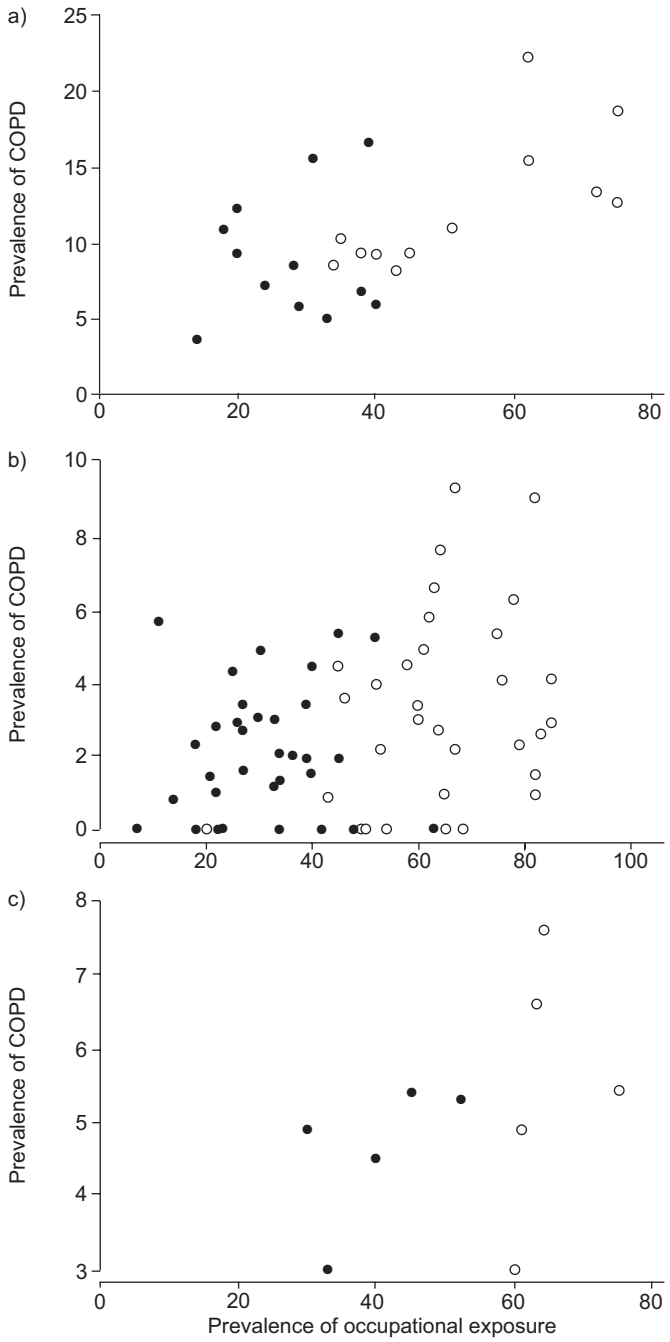

FIGURE 1. Prevalence of occupational exposure against the prevalence of chronic obstructive pulmonary disease (COPD) of Global Initiative for Chronic Obstructive Lung Disease stage II or higher, by study cohort and for all the site–sex strata combined. a) Burden of Obstructive Lung Disease cohort: Spearman correlation r=0.48 (p=0.02). b) European Community Respiratory Health Survey follow-up survey cohort: Spearman correlation r=0.26 (p=0.06). c) Latin American Project for the Investigation of Obstructive Lung Disease: Spearman correlation r=0.63 (p=0.05). ●: females; ○: males.

10% increase in prevalence of job exposures. Among females, pack-yrs among smokers was positively associated with COPD prevalence (p=0.02), but mean age was not. Among males in the stratified analysis, both pack-yrs among smokers (p=0.006) and mean age (p=0.04) were statistically associated with COPD, but in a negative direction, that is, taking ever-smoking and occupational exposure into account, sites with younger mean age and less pack-yrs of smoking intensity manifested a higher prevalence of disease.

The potential for a smoking–occupational exposure interaction was assessed by analysing the effect of conjoint prevalence of risk at the group level. Sites with a higher combined prevalence of smoking and occupational exposure were associated with a 2.8% higher COPD prevalence relative to sites with both lower smoking and exposure (p=0.003). There was no significant difference associated with higher smoking or occupational exposure alone, relative to lower smoking and exposure. The same pattern was seen when the sites were stratified by sex, with a higher COPD prevalence associated with combined higher smoking and occupational exposure of 3.3% among females (p=0.01) and 3.0% among males (p=0.008).

DISCUSSION

The individual-level association between occupational exposure and COPD has been documented in a rapidly accumulating literature on this subject [1, 2, 13–18]. The findings of the current ecological analysis are consistent with those studies, but also add another important dimension by assessing this question from a population perspective. Moreover, the association between adverse working conditions and COPD found in the present study carries significance as a global finding, applicable to both males and females. This observation places the occupational burden in COPD squarely in the public health context, alongside the well-appreciated and critical contribution of cigarette smoking to disease prevalence.

It is paramount to keep in view the inherent limitations, as well as the advantages, of an ecological group-level analytical approach [19, 20]. For example, the overall prevalence of smoking, included in the current model, is an imprecise surrogate of individual-level, direct cigarette exposure. However, conversely, this group-level smoking measure has the advantage of also subsuming the likelihood of second-hand smoke exposure in the population in addition to direct personal smoking. Indeed, data from the ECRHS has suggested an ecological association between personal and workplace second-hand smoking [21]. Lifetime second-hand cigarette smoke exposure has been implicated as a risk factor for COPD and can be difficult to measure accurately at the individual level using standard survey methods [22, 23]. Similarly, the prevalence of *in utero* cigarette smoking exposure, which may be a risk factor for COPD through low birthweight or other mechanisms, is also likely to correlate with female population smoking rates and is difficult to ascertain at the individual level [24]. Smoking intensity duration at the population level, which was also included in the current models, may reflect second-hand smoke exposure intensity as well. This measure, however, is complicated by potential inter-relationships with the distributions of population age and age at smoking initiation and cessation. This may account for the paradoxical association observed between COPD and age and pack-yrs in the male stratum. On balance, although ecological measures of smoking imperfectly estimate individual personal smoking, such measures do have the

TABLE 3 Population prevalence of occupational exposure and cigarette smoking as independent predictors of chronic obstructive pulmonary disease (COPD) prevalence: multiple linear regression analysis

Independent variables	Increase in COPD prevalence per 10% increase in exposure	p-value
All observations[#]		
Dusty/dirty jobs	0.8 (0.3–1.3)	0.003
Cigarette ever-smokers	1.3 (0.7–1.8)	<0.0001
Males only[¶]		
Dusty/dirty jobs	0.8 (0.3–1.3)	0.004
Cigarette ever-smokers	0.9 (0.1–1.8)	0.04
Females only[¶]		
Dusty/dirty jobs	1.0 (0.1–11.9)	0.03
Cigarette ever-smokers	1.1 (0.4–1.8)	0.005

Data are presented as estimated percentage (95% confidence interval), unless otherwise stated. [#]: n=90; mixed model also included sex, mean age per stratum and mean pack-yrs among ever-smokers per stratum as fixed effect variables and study cohort as a random variable; analysis weighted for study number per sex–stratum per site. [¶]: n=45; mixed model included mean age per stratum and mean pack-yrs among ever-smokers per stratum as fixed effect variables and study cohort as a random variable; analysis weighted for study number per site.

advantage of capturing elements of second-hand smoke exposure that may otherwise not be assessed.

As with cigarette smoking, population-level rates of adverse occupational exposures provide a measure that is more than simply a surrogate of individually assessed employment conditions and is thus at risk of ecological bias [25]. Although the ECRHS measure of occupational risk included assigned exposure likelihood using a job exposure matrix, a key measure in all three cohorts is self-reported exposure. Such self reports can theoretically introduce bias in exposure assessment, but the actual impact of this remains open to question, with heterogeneous estimates of such impact in different studies internationally [26–28]. In areas with generally poorer workplace conditions, some subjects may be more likely to under-report exposure because, relative to other sites, they minimise their individual on-the-job condition; in situations of overall excellent hygiene, even trivial exposures may lead to report of vapours, gas, dust or fumes. Both tendencies would lead to measurement ''noise'' that is dampened in group-level analysis. The confounding effect of recall bias systematically linking exposure to disease can also be mitigated by analysis at the population level. Finally, to the extent that workplaces are indeed widely contaminated, there is increased likelihood of local neighbourhood and wider ambient air pollution exposure from site sources that can be captured with a group-level analysis.

Several individual-level analyses suggest that there is smoking–occupational exposure interaction that is a more than additive for increased chronic obstructive pulmonary disease risk [29–31]. In the present analysis, sites with smoking and occupational exposure above the sex-specific median had a significantly higher prevalence of chronic obstructive pulmonary disease, an observation that could be consistent with such an interaction. More fully teasing out the nuanced inter-relationships between occupation and smoking in chronic obstructive pulmonary disease, including the potential associations between second-hand smoke in youth and later exposure-ridden employment, may require multi-level analyses that simultaneously take into account both individual-level and population-level exposures, thus balancing potential biases present in individual-level and ecological approaches [18]. Going forward, it will also be important undertake analysis through longitudinal study associations with specific occupations or occupational groups with chronic obstructive pulmonary disease, while taking into account temporal changes in the occupational contribution to chronic obstructive pulmonary disease that might come from improved or deteriorating working conditions. Such changes may modify the role that vapours, gas, dust and fumes play in the burden of disease. By the same token, it is important to recognise as well that reductions in direct and second-hand cigarette smoke exposure, resulting from successful public health efforts, may increase the proportion of disease attributable to occupational factors. Smoking remains critical in chronic obstructive pulmonary disease causation, but the contribution of occupational exposures must not be ignored. This is true not only at the population level, but also in the prevention of disease initiation and progression at the one-on-one level of clinical practice.

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