

## RESEARCH ARTICLE

# Smoking, occupational exposures, and idiopathic pulmonary fibrosis among Swedish construction workers

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## Abstract

**Background:** Cigarette smoking and occupational exposures each have been reported to increase the risk of idiopathic pulmonary fibrosis (IPF), a disease previously considered of unknown origin. We investigated the risk of IPF mortality associated with combined smoking and occupational exposures.

**Methods:** A registry study of Swedish construction workers ( $N = 389,132$ ), linked baseline smoking and occupational data with registry data on cause of death and hospital care diagnoses. Occupation was classified by the likelihood of exposure to vapors, gases, dusts, or fumes using a job-exposure matrix. Those likely exposed to asbestos or silica were excluded from the analysis. Age-adjusted relative risks [RRs] were calculated using Poisson regression. Follow-up observation began at age 40 and ended at age 89.

**Results:** Heavy smokers at baseline who were exposed to inorganic dusts during their working life had an increased risk of IPF mortality (RR 1.70; 95% confidence interval [CI] 1.11–2.60), while there was no statistically increased risk in the other exposure groups. There were dose–response relationships between smoking at baseline and IPF mortality among both unexposed and dust exposed workers, with similar risk for dust exposed and unexposed, except among baseline heavy smokers, where workers exposed to inorganic dust manifested the highest risk (RR 4.22; 95% CI 2.69–6.60). Excluding workers with chronic obstructive pulmonary disease or emphysema did not affect the results substantively.

**Conclusion:** A clear dose–response relationship was seen between smoking at baseline and IPF, supporting a causal relationship. Occupational exposure to inorganic dusts, excluding silica and asbestos, was associated with increased risk of IPF in baseline heavy current smokers.

## KEYWORDS

construction, inorganic dust, IPF, occupation, smoking

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## 1 | INTRODUCTION

There are multiple causes of lung fibrosis, including occupational dusts, radiation, medications, and systemic diseases. Interstitial pulmonary fibrosis without any of these known causes is classified as idiopathic pulmonary fibrosis (IPF),<sup>1-3</sup> but a number of potential occupational risk factors for IPF have been identified on epidemiologic grounds, including exposure to wood dust, metal dust, and combined exposures (vapors, gases, dusts and fumes [VGDF]).<sup>4,8</sup> Lung fibroses caused by exposure to certain specific dusts, (e.g., asbestos, coal dust, or silica), known as pneumoconioses, are well-established occupational lung diseases.<sup>9</sup> Epidemiological studies of mortality in silica exposed workers have not been able to establish any threshold dose below which there is no increased mortality.<sup>10,11</sup> While silicosis may have distinguishing pathological signs such as discrete silicotic nodules, asbestosis has no specific pathological feature except for findings of deposited material, that is, asbestos bodies, in the lungs. The cellular mechanisms are still not fully understood,<sup>9,12</sup> and there may be a risk of silicosis and asbestosis being misdiagnosed as IPF.<sup>13</sup> Also of note, a few studies have found that cigarette smoking is a risk factor for IPF,<sup>4,6,14-16</sup> which may be an indicator of a “generic” particulate and fume effect, and smokers with occupational exposure have been suggested to be a high-risk group for IPF.<sup>4</sup>

The identification of occupational risk factors for IPF is important given that an occupational disease may be prevented, and that prevention is often the only effective measure to reduce such morbidity. Construction is a relatively large industrial sector in many countries, and includes various trades involving high dust exposure. A recent official American Thoracic Society and European Respiratory Society statement on the occupational burden across a range of lung disease highlighted that one in four cases of IPF may be attributable to VGDF and thus preventable with better control of workplace exposures.<sup>7</sup> We aimed to investigate possible occupational contributors to the etiology of IPF among a cohort of Swedish construction workers. We also considered concomitant cigarette smoking, given that this also is a potential risk factor for IPF. Importantly, to avoid misclassification bias, we excluded workers with likely exposure to asbestos or silica.

## 2 | MATERIALS AND METHODS

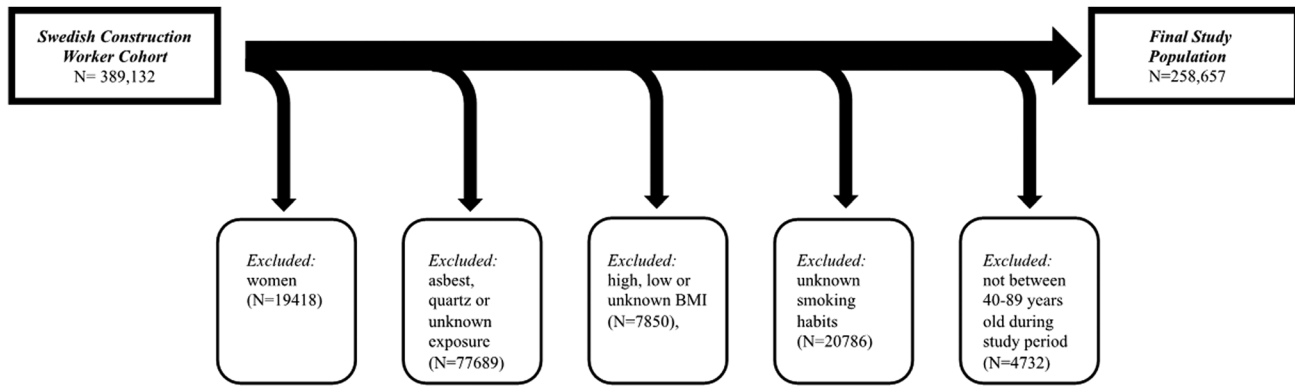
From the mid-1960s until the beginning of 1993, Swedish construction workers participated in a dedicated, national occupational health service. Under this scheme, all construction industry workers were offered free regular health examinations, typically every 3–5 years, but with highly variable participation. Worker participation in the health examinations was estimated to be approximately 80%.<sup>17</sup> From 1971 onward, the results from the periodic examinations were incorporated into a data registry including information on job, year of birth, weight, height, and tobacco smoking status. Up until 1985, there were 215 distinct job titles for workers included. In 1985, the classification was consolidated into 90 job titles. In the mid-1970s, likely exposures linked to different jobs were classified on a 5 grade

Likert scale (none-highest). This classification was reviewed by occupational hygienists in the early 2000s and the exposure to cement dust, concrete dust, man-made mineral fibers (MMMf), asbestos, silica, wood dust, fumes from asphalt, welding, diesel exhaust, vapors from epoxy, isocyanates (although few workers in the Swedish construction industry have been exposed to isocyanates) or organic solvents were incorporated into a job-exposure matrix (JEM). There were no specific data on talc exposure. We selected five categories of exposure: wood dust, inorganic dust (cement dust, concrete dust, MMMf), vapors, fumes, and gases, dichotomized as none versus any level (1–5). We also combined these categories (VGDF). The subcategories of VGDF were not mutually exclusive (an employee could be assigned one or more JEM exposure subcategory). We defined referents as those without any of these exposures. Through linkage, using the unique Swedish personal number, with the Swedish Death Registry, the Registry of Hospital Care, and the National Population Registry, we added information about cause and year of death, diagnoses during hospital care, and emigration.

The cohort includes 389,132 individuals employed in the construction industry, who participated in at least one health examination between 1971 and January 1993. For the purposes of this study, we excluded persons with unknown smoking habits, unknown BMI, very high or low BMI ( $BMI \geq 35$  or  $<18.5 \text{ kg/m}^2$ ) at the first examination. Further, all workers in jobs that were classified as likely exposed to any level of asbestos or silica were excluded. We also excluded women because there were few cases of IPF among women and few who were exposed to VGDF (Figure 1).

We identified all persons (cases) in the cohort who, according to the Death Registry from 1971 to 2015, had a diagnosis of IPF as an underlying or contributing cause of death. We used the following codes to define IPF: ICD10 J84.1 (1997–2015), ICD9 515 or 516.3 (1987–1996), and ICD8 517 (1971–1986). Cases who had a diagnosis of pneumoconiosis, hypersensitivity pneumonitis, or lung fibrosis due to medications as an underlying or contributing diagnosis noted in either the Death Registry or the Registry of Hospital Care were excluded. We calculated person-years from the year after the calendar year of the first health examination until death, emigration, or 2015. For smoking status, information from the first health examination was used, but if that was missing, any information from the second or third examination was used to classify the person as baseline nonsmoker, ex-smoker, moderate current smoker (1–14 cigarettes/day) or heavy current smoker ( $\geq 15$  cigarettes per day). There were not enough workers with more than one health examination for reliable analysis of tobacco use longitudinally.

We analyzed each dichotomous JEM exposure category separately (VGDF and each subcategory, respectively), adjusting for age (10-year classes) and baseline smoking habits (classified as four categories as above). Follow-up began from age 40 years, since there were only three IPF cases below that age. The workers were followed until 89 years old, since diagnostics may be less intensive in persons above that age. In these analyses, we calculated relative risk (RR) and 95% confidence interval (CI) using Poisson regression models with a log link, using maximum likelihood estimates for RR and Wald estimates for CI using



**FIGURE 1** Flow-chart of exclusion procedure for the present study within the Swedish Construction Workers Cohort

procedure GENMOD<sup>®</sup> (SAS institute). In exploratory modeling, we included BMI (three classes: 18.5–24.9, 25.0–29.9, and 30.0–34.9 kg/m<sup>2</sup>) as a possible confounder. In the test of trend (Table 1), baseline smoking was modeled as an ordinal integer variable (0 = nonsmokers, 1 = ex-smokers, 2 = moderate current smokers, 3 = heavy current smokers), while in other analyses it was analyzed as a categorical variable.

Ethical permission was granted by the Swedish Ethics Authority, no 2019–05992.

### 3 | RESULTS

The number of workers (*N*) within each group, age at first examination, BMI, and baseline smoking habits are presented in Table 2. Within the cohort as a whole and for each exposure category, there was no statistically significant occupationally related increased risk of IPF mortality,

while the 33 cases among workers exposed to wood dust represented a significantly reduced risk (Table 3).

Table 4 shows an analysis of the occupational risk of IPF restricted to baseline current smokers, stratified by smoking intensity. Among baseline moderate smokers (<15 cigarettes per day) there was no statistically increased risk associated with VGDF overall or any of its components. Among the stratum of baseline heavy smoking ( $\geq 15$  cigarettes per day), however, occupations with likely exposure to inorganic dusts (cement dust, concrete dust, MMMf) carried significant increased risk of IPF: RR, 1.70 (95% CI, 1.11–2.60).

We further explored a potential step-up in smoking effects as shown in Table 5. Former smoking and current moderate and heavy smoking at baseline were each associated with statistically significant increased risk of IPF, and the trend of increasing risks by smoking was significant regardless of occupational exposure. Of note, in the stratum with occupational inorganic dust exposure, baseline heavy smoking manifested the

**TABLE 1** Estimates of RR of IPF mortality among 40–89 years old VGDF exposed workers, inorganic dust exposed workers, and referents respectively, by baseline smoking habits and adjusted for age and BMI

Baseline smoking habits	RR (95% CI)		
	Referents	VGDF-exposed	Inorganic dust-exposed
Nonsmokers	1.0	1.0	1.0
Ex-smokers	1.41	1.48	1.54
Moderate current smokers	1.98	2.18	2.36
Heavy current smokers	2.78	3.22	3.63
Coefficient for trend(B) according to smoking (SE)	0.34 (0.052)	0.39 (0.058)	0.43 (0.071)
<i>p</i> value for coefficient deviation from 0	<.001	<.001	<.001

Note: Smoking was modeled on an ordinal scale (0 = nonsmokers, 1 = ex-smokers, 2 = moderate current smokers, 3 = heavy current smokers) by Poisson regression analysis using log-link ( $\log(\text{incidence rate}) = A + B \times \text{smoke} + C_2 \times \text{BMICL}_2 + C_3 \times \text{BMICL}_3 + E \times \text{age}$ ), where A, B, C, D, E are coefficients estimated by maximum likelihood statistics using the SAS procedure GENMOD). Normal BMI (18.5–24.9 kg/m<sup>3</sup> (BMICL<sub>1</sub>)) is used as reference. In Table 5, the relative risks according to smoking habits are estimated by the log-link function ( $\log(\text{incidence rate}) = A + B_1 \times \text{exsmoke} + B_2 \times \text{moderatesmoke} + B_3 \times \text{heavysmoke} + C_2 \times \text{BMICL}_2 + C_3 \times \text{BMICL}_3 + E \times \text{age}$ ) where exsmoke, moderatesmoke, and heavysmoke are 0/1 variables and the relative risk depending on smoking is estimated by the coefficients B<sub>1</sub>–B<sub>3</sub> using nonsmokers as reference.

Abbreviations: BMI, body mass index; CI, confidence interval; RR, relative risk; VGDF, vapors, gases, dusts, or fumes.

**TABLE 2** Age at first examination, BMI, and smoking by exposure category among workers 40–89 years of age

Exposure	N	Age at first examination		BMI at baseline (%)			Cigarette smoking at baseline (%)			
		Mean		18.5–24.9 kg/m <sup>2</sup>	25.0–29.9 kg/m <sup>2</sup>	30–34.9 kg/m <sup>2</sup>	Nonsmokers	Ex-smokers	Current moderate	Current heavy ≥15/day
VGDF <sup>a</sup>	117,802	34.4	30.4	65.5	30.4	4.0	42.6	15.8	27.6	13.8
Fumes	23,710	35.7	35.9	58.4	35.9	5.7	36.7	16.9	28.5	17.8
Gases	46,554	33.0	27.6	68.9	27.6	3.4	45.4	14.7	25.6	14.2
Wood dust	19,328	33.4	28.5	68.5	28.5	3.0	48.7	15.0	24.3	12.0
Inorganic dusts <sup>b</sup>	76,267	34.4	29.7	66.3	29.7	3.9	43.0	16.0	28.2	12.9
Referents	140,315	33.6	29.5	66.6	29.5	3.9	45.5	16.3	24.3	13.9

Abbreviations: BMI, body mass index; MMMf, man-made mineral fibers; VGDF, vapors, gases, dusts, or fumes.

<sup>a</sup>VGDF subsumes all of the subcategories specified below. These subcategories are not mutually exclusive, with overlap possible. Silica and asbestos-exposed workers excluded from analysis (see Methods).

<sup>b</sup>Cement dust, concrete dust, and MMMf.

**TABLE 3** RR of IPF mortality, number of cases, and pyr in relation to different occupational exposures among workers 40–89 years of age, adjusted for age, BMI, and baseline smoking

Exposure	Cases	pyr	RR	95% CI
VGDF	304	2,635,654	0.99	0.85–1.16
Fumes	59	557,540	0.95	0.72–1.26
Gases	110	982,790	1.05	0.85–1.30
Wood dust	33	412,569	0.69	0.49–0.99
Inorganic dusts	214	1,701,697	1.05	0.88–1.25
Referents	343	3,262 891	1.0	NA

Abbreviations: BMI, body mass index; CI, confidence interval; IPF, idiopathic pulmonary fibrosis; pyr, person years; RR, relative risk.

highest observed point estimate of IPF risk: RR 4.22 (2.69–6.60). As shown in Table 5, the RR of baseline heavy smoking (compared to never smokers) in the absence of occupational exposure was 2.55 (1.78–3.65).

In an alternative analysis, using non-VGDF exposed never smokers as reference group, the RR of IPF for never smokers exposed to inorganic dusts was 0.99 (95% CI, 0.68–1.45); for baseline heavy smokers without inorganic dust exposure 2.62 (1.83–3.74); and for baseline heavy smokers exposed also to inorganic dusts 4.46 (3.00–6.63), suggesting an interactive effect on IPF mortality by combined heavy baseline current smoking and inorganic dust exposure (data not in tables). A trend analysis showed a statistically significant increased risk by smoking intensity both in referents and exposed workers ( $p < .001$ ; Table 1).

When all cases and referents with chronic obstructive pulmonary disease (COPD) or emphysema (ICD 10 J43 and J44, ICD 9 496 and 492, ICD 8 492 and 519) as underlying or contributing cause of death were

**TABLE 4** RR of IPF mortality among baseline moderate current and heavy current smokers respectively, number of cases and pyr in relation to different occupational exposures among workers 40–89 years of age, adjusted for age and BMI

Exposure	Cases	pyr	RR	95% CI
<i>Baseline moderate current smokers (1–14 cigarettes/day)</i>				
VGDF	124	796,423	0.90	0.71–1.15
Fumes	31	171,423	1.13	0.76–1.67
Gases	47	279,128	1.05	0.75–1.46
Wood dust	13	112,796	0.66	0.37–1.17
Inorganic dust	83	522,192	0.90	0.68–1.18
Referents	133	856,901	1.0	NA
<i>Baseline heavy current smokers (&gt;15 cigarettes/day)</i>				
VGDF	47	353,450	1.31	0.88–1.95
Fumes	8	95,646	0.88	0.42–1.86
Gases	16	137,455	1.25	0.71–2.20
Wood dust	3	49,382	0.59	0.19–1.90
Inorganic dusts	37	210,695	1.70	1.11–2.60
Referents	51	451,628	1.0	NA

Abbreviations: BMI, body mass index; IPF, idiopathic pulmonary fibrosis; pyr, person years; RR, relative risk.

**TABLE 5** RR of IPF mortality among 40–89 years old VGDF exposed workers, inorganic dusts exposed workers and referents respectively, by baseline smoking habits and adjusted for age and BMI

Baseline smoking habits	RR (95% CI)		
	Referents	VGDF-exposed	Inorganic dusts-exposed
Nonsmokers	1.0	1.0	1.0
Ex-smokers	1.76 (1.27–2.36)	1.87 (1.32–2.63)	1.86 (1.24–2.80)
Moderate current smokers	2.49 (1.87–3.31)	2.36 (1.72–3.23)	2.21 (1.52–3.22)
Heavy current smokers	2.55 (1.78–3.65)	3.39 (2.30–4.99)	4.22 (2.69–6.60)

Abbreviations: CI, confidence interval; IPF, idiopathic pulmonary fibrosis; RR, relative risk; VGDF, vapors, gases, dusts, or fumes.

excluded from the cohort, the RR of IPF mortality in relation to inorganic dust exposure was 1.06 (95% CI, 0.88–1.28) in the whole cohort but 1.88 (95% CI, 1.14–3.09) estimated among baseline heavy current smokers.

## 4 | DISCUSSION

Smoking has previously been suggested as a risk factor for IPF.<sup>4,6,14–16</sup> Occupational exposures also have been linked epidemiologically to IPF, an association supported in a recent systematic review.<sup>7</sup> In this study, we build on these observations by identifying an interactive risk between smoking, ascertained cross-sectionally at baseline, and occupational exposure to inorganic dust in construction workers.

Although analyses of occupational risk for IPF standardly adjust for smoking effects, the potential for a smoking-occupational interaction has been understudied. A previous Swedish study did identify male smokers with occupational exposure as a high-risk group for severe IPF, findings that are in line with our results.<sup>4</sup> Although smoking is not generally considered a major factor in classic pneumoconioses like asbestosis or silicosis, a recent Chinese study found a synergistic joint effect of smoking and silica exposure on the risk of silicosis.<sup>18</sup> The pathogenesis of IPF is not fully understood, and likely involves both genetic and environmental factors.<sup>19</sup> Both cigarette smoke and inhalant occupational agents may cause oxidative stress and damage the alveolar epithelium. Further, as inhalation of cigarette smoke may impair lung clearance mechanisms, it is reasonable to suppose that exposure to both smoking and dust may overload lung clearance capacity with a subsequent increased risk of particle retention and destructive lung damage.<sup>6</sup> We also considered other pulmonary fibrosis diagnoses than IPF or pneumoconioses, but these were very few compared with IPF and could not be analyzed reliably.

Occupational exposure to dust may include inhalation of silica dust and asbestos fibers, the causes of silicosis and asbestosis, respectively.<sup>20,21</sup> Like IPF, these diseases are lung fibroses. However, others have shown an association also between silica exposure and IPF.<sup>22</sup> Excluding those workers likely exposed to silica or asbestos is both a limitation and a strength. If silica or asbestos cause IPF by a mechanism that is wholly unrelated to that of pneumoconiosis, then we will be underestimating the

occupational risk of IPF. However, as silicosis and, especially, asbestosis may present similar radiological and pathological findings as in IPF, there may be a risk of misdiagnosing of silicosis or asbestosis as IPF which may confound the correlation of occupational dust exposure and IPF in an epidemiological study.<sup>13</sup> We, therefore, excluded workers with known exposure to silica and asbestos based on the JEM.

One limitation of our study is the lack of a detailed individual exposure assessment, which could lead to misclassification of exposure, especially among workers with less obvious exposure to silica or asbestos. Still, we believe that the possible effect of misclassified exposure after excluding workers with known exposure to asbestos and silica is probably small and unlikely to completely explain the association between occupational exposures and IPF in our study. Further, we cannot identify a specific agent within inorganic dust associated with IPF, and any invasive procedures to investigate lung burden of specific agents were not within the scope of our study. Nonetheless, within the exposure group of inorganic dusts there might be exposure to, for example, low levels of silica or asbestos unaccounted for in our JEM, and/or talc exposure. Talc may be a fibrotic agent by itself or contaminated with asbestos or silica.<sup>23</sup> However, to our knowledge, talc has not been used extensively in the Swedish construction industry. Another limitation of our study is that smoking history was obtained in a single assessment at the time of the health examination, while an individuals' smoking habits may change over time. Such misclassification would however most probably mean that the association between smoking and IPF seen in our study is underestimated.

It may be suspected that the increased IPF mortality among men who were smoking at the baseline exam, both with and without occupational exposure, could be explained by concomitant COPD or emphysema. COPD is a common disease among smokers, but may be aggravated or caused by occupational dust exposure,<sup>24</sup> findings reported earlier from the present cohort.<sup>25</sup> Emphysema is also linked to smoking, and a combination of IPF and emphysema has been described. A common pathogenic background is suspected, but it is still unclear whether IPF and emphysema sometimes constitute a single disease entity or if they develop in parallel because of common risk factors.<sup>26–28</sup> To study the possible confounding by COPD or emphysema on

IPF mortality, we made an additional analysis, excluding all workers where COPD or emphysema was mentioned on the death certificate as underlying or contributing cause of death. There was however no marked change in the risk estimates for IPF mortality in relation to dust exposure in that analysis, arguing that our findings are not explained by COPD or emphysema-related mortality.

The strengths of our study include the longitudinal study design, the very large population of construction workers with access to death registry data and information on job titles classified according to a JEM. We were able to adjust our analyses for baseline smoking habits and age. We also adjusted for BMI in a separate analysis, but BMI had no effect on the risk estimates. In conclusion, this study is a significant contribution to the literature on IPF risk factors. It supports previous findings suggesting smoking as a dose-dependent risk factor and indicates that occupational exposure to inorganic dust in combination with baseline heavy current smoking increase the risk. We also report findings suggesting that these associations are not explained by neither misclassification of silicosis or asbestosis as IPF nor confounded by COPD or emphysema mortality.

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#### CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

#### DISCLOSURE BY AJIM EDITOR OF RECORD

John Meyer declares that he has no conflict of interest in the review and publication decision regarding this article.

#### AUTHOR CONTRIBUTIONS

Martin Andersson drafted the manuscript and Bengt Järholm performed the analyses. Martin Andersson and Bengt Järholm are responsible for the Swedish Construction Workers Cohort and are accountable for all aspects of the present work in questions related to accuracy and integrity. All authors participated in the conception and design of the study, and in the analysis and interpretation of the data, as well as revising it critically for important intellectual content. All authors approved the final version.

#### DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

#### ETHICS APPROVAL AND INFORMED CONSENT

The Swedish Construction Workers Cohort is maintained at the Department of Public Health and Clinical Medicine, Sustainable Health, Umeå University, Umeå, Sweden. Verbal consent was obtained at the health examinations and ethical permission was granted by the Swedish Ethics Authority, no 2019-05992.

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