



Research into
Malignant and Non-
malignant
Respiratory Disease
Prescriptions: Report
for Silica and COPD

Damien McElvenny, Anne Sleuwenhoek, Helena Copsey,
Will Mueller, Ken Dixon, David Fishwick (HSE), Hilary Cowie

Our Impact on the Environment

At IOM we seek to minimise our environmental impact. We produce thousands of reports every year and these consume a large quantity of paper. To minimise our impact on the environment, we prefer to only provide an electronic copy of reports, although we can provide a paper copy on request. If you have any additional requirements please let us know.

Contents

1 Introduction	4
2 Methods	5
3 Results	6
3.1 Cohort studies	6
3.2 Case-control studies	8
4 Synthesis and Discussion	10
5 Conclusions	12
6 References	13
Appendix 1 – Data Extraction Spreadsheet and List of All the References Considered	15
Appendix 2 – Current prescription for COPD	16

1 Introduction

Silica and chronic obstructive pulmonary disease is the first of 6 high-priority occupational exposure-disease combinations that were identified with IIAC as being of highest priority for more detailed investigation (see Report of Phase 1 of this project).

This document contains a commentary on the extracted data for relevant occupational epidemiological studies and is meant to be read in conjunction with the associated spreadsheets containing the data extraction from these studies (See Appendix 1).

2 Methods

Searches of Web of Science and NLM PubMed databases were undertaken in September/November 2022 using the following search string: exposure AND silica AND (COPD OR “chronic obstructive pulmonary disease”). The searches were run in PubMed in the title/abstract field (no date restrictions) in ‘Advanced Search’ and in Web of Science Core Collection in the topic field (title, abstract, keywords) from 1996 to present. Note that we did not explicitly search for non-malignant respiratory disease studies (a large proportion of these conditions will be COPD).

Bibliographies of the studies included in the silica and COPD reviews found in our earlier literature searches (Phase 1) were searched to identify any additional individual studies that should be screened for inclusion in the tables of evidence.

A full risk of bias assessment was not carried out. A crude quality assessment was carried out on the basis of whether the study adjusted for smoking and/or other known/suspected occupational causes of COPD.

3 Results

Overall, 108 relevant papers were identified from the literature searches and screened using title and abstract (see Appendix 1).

After screening on title and abstract 37 were identified for full paper screening. plus one other identified at the data extraction stage (Dement et al., 2015) giving a total of 38 to proceed to full text screening. Twenty two papers were subsequently excluded:

- No silica exposure assessment or not silica exposed – 7
- Duplicate paper – 1
- Cohort of silicotics – 1
- Review – 12
- Not COPD – 1

Data extraction was therefore completed for 16 included papers, of which 3 were cross-sectional studies and so not considered further in this report.

No additional papers, not already identified in the searches, were identified from bibliographies of recent systematic reviews (Hoet et al., 2017; Möhner et al., 2013; Sen et al., 2016) and the recent WHEC paper (HSE Workplace Health Expert Committee (WHEC), 2019).

The full extracted data are contained in Appendix 1.

3.1 Cohort studies

Eight relevant cohort studies were identified and had their data extracted (Cherry et al., 2013; Dement et al., 2010; Graber et al., 2014; Grahn et al., 2021; Hochgatterer et al., 2013; Lenander-Ramirez et al., 2022; Mazitova et al., 2012; Moshhammer & Neuberger, 2004; Reynolds et al., 2017) (see accompanying spreadsheet). Note studies of uranium miners were excluded due to lack of relevance for the UK.

Over 40,000 subjects from the Stockholm Public Health Survey were followed up from 2007 through to 2014. COPD cases were identified via prescription of COPD-associated medical treatment and/or self-reported physician's diagnosis of COPD (Grahn et al., 2021). Exposure to silica was assessed using the FINJEM job-exposure matrix. Analysis was via proportional hazards regression modelling, separately for males and females, and adjusted for smoking (ever versus never, and pack-years) and age (and sometimes education). The Hazard ratios adjusted for age and smoking were 1.46 (95% CI: 1.13 to 1.90) for men and 1.28 (0.86 to 1.90) for high (≥ 0.048 mg/m³) vs low (0.048 mg/m³) exposure. Aside from smoking, there were potential co-exposures to a number of occupational exposures such as inorganic particles and fibres and organic particles. There was no suggestion of an increased risk above or even approaching 2, a finding consistent with the exposure levels from this study being too low.

A cohort of 1225 miners (726 slate miners; 529 unexposed non-miners) was followed up for COPD (Reynolds et al., 2017). COPD was defined as FEV₁/FVC < 0.7, measured at the time of the survey. The OR for COPD was 1.38 (1.06 to 1.81), and was adjusted for age. Analysis by duration of work as a miner, stratified by age gave the following ORs: Age 40-54, 1-8 years 1.32 (0.66 to 2.65), 9-24 years 1.13 (0.60 to 2.11), >25 years 2.05 (1.03 to 4.09); age 55+, 1-8 years 1.07 (0.46 to 2.46), 9-24 years 1.26 (0.68 to 2.34), >25 years 1.55 (0.95 to 2.52). Slate mining was used as a surrogate for silica exposure and there is some suggestion that prolonged working (>25 years before the age of 55) is associated with a doubled risk of COPD.

A Russian cohort of 1375 workers aged over 30 who worked for an industrial enterprise for at least 5 years from 2 foundries, 1 aircraft plant and 2 oil extracting companies was compared with healthy workers with no signs of COPD over 2005 to 2008 (Mazitova et al., 2012). A common definition for COPD was based on the last GOLD revision (Minette, 1989). A significantly elevated OR of COPD was found for exposure to silica dust containing 10% or more silica of 6.2 (3.6 to 10.7), probably unadjusted for any covariates. There was potential co-exposure to other vapours, gases, dusts and fumes. There was evidence of a more than doubled OR in non-smoking workers in dusty trades. The authors concluded that there was evidence in support of dust containing more than 10% silica doubling the risk of COPD.

A cohort of pottery workers or workers from sandstone industries employed during 1931 to 1992 in Stoke on Trent was followed to 2008 for comparison to mortality rates for England and Wales (Cherry et al., 2013). COPD was defined as the underlying cause of death for the external SMR analyses and for the internal analysis, COPD mentioned anywhere on the death certificate. The SMR and 95% CI for COPD for follow-up from 1985 to 2008 was 1.46 (1.24 to 1.71). After adjusting for smoking status, compared to a duration of exposure of < 2 years, the hazard ratios for 2.5<10 years was 1.03 (0.65 to 1.63), for 10<25 years was 1.51 (0.97 to 2.37) and for > 25 years was 1.49 (0.95 to 2.36). Analysis by mean exposure concentration (respirable silica), adjusted for smoking among smokers only, compared to exposures < 100 $\mu\text{g}/\text{m}^3$ was 1.58 (0.88 to 2.87) for exposure 100<150 $\mu\text{g}/\text{m}^3$, was 0.98 (0.57 to 1.68) for 150<200 $\mu\text{g}/\text{m}^3$ and was 0.92 (0.55 to 1.56) for > 200 $\mu\text{g}/\text{m}^3$. This study supported there being a raised RR of COPD following silica exposure and that may be an exposure response for duration of employment in years, but not for mean exposure concentration. However, the evidence for a doubling of the RR was not present in this study.

A US cohort study of 9,033 male underground coal miners was followed up for 37 years to 2007 (Graber et al., 2014). COPD was defined on the basis of death certificates. There was a lack of information on the proportion of dust due to silica exposure. The SMR for COPD was 1.11 (95% CI: 0.99 to 1.24). A proportional hazards modelling of log of exposure as a continuous variable adjusted for smoking category and coal dust exposure suggested a marginally statistically significantly increased mortality from COPD associated with silica exposure HR 1.04 (0.96 to 1.52), which in turn suggests that there may be an exposure level at which the RR of COPD mortality from silica exposure is doubled. Examination of exposures in a categorical model, adjusted for the same variables, suggested significantly increased RR compared to the lowest exposure category, but an unclear pattern of how RR varied with higher exposure categories. There is a suggestion from this study that there might be a doubling of the RR in underground miners, but probably only for with very high exposures (which may not be possible in practice).

A Swedish study of 1750 male foundry workers born between 1945 and 1958 was followed up from 2001 to 2017 (Lenander-Ramirez et al., 2022). Cases of COPD were obtained from the Swedish Outpatient Register and from death certificates. Cumulative exposure to silica was assessed using historical measurements. The SIR for COPD for total respirable dust < 2.76 $\text{mg}/\text{m}^3\cdot\text{years}$ was 1.81 (1.25 to 2.54), for exposure group 2.77-9.82 $\text{mg}/\text{m}^3\cdot\text{years}$ was 2.04 (1.42 to 2.83) and for exposure 9.83+ $\text{mg}/\text{m}^3\cdot\text{years}$ the SIR was 1.97 (1.36 to 2.77). When the analysis was restricted to respirable silica the SIR for <0.14 $\text{mg}/\text{m}^3\cdot\text{years}$ was 2.35 (1.56 to 3.40), for exposure 0.15-0.38 $\text{mg}/\text{m}^3\cdot\text{years}$ was 2.07 (1.43 to 2.91) and for exposure 0.39+ $\text{mg}/\text{m}^3\cdot\text{years}$ the SIR was 1.64 (1.17 to 2.24). This suggests a possible threshold effect, rather than exposure-response, but could be due to lack of adjustment for smoking and other potential confounders. There is a suggestion that the RR for silica exposure in foundry workers may be as high as doubled, but it's not clear if this is due to the silica exposure or other co-exposures that may be present (due to the lack of a trend with silica exposure).

A cohort study in Austria examined the influence of work-associated dust on spirometric results in a cohort of 3,229 mostly male workers from 195 companies during 2002 to 2010 (Hochgatterer et al., 2013). Data on occupational exposure was provided according to the most prominent type of mineral dust. Lung function (not necessarily COPD) was compared with the general population in a multiple linear regression model. Statistically significant associations were found for quartz dust after adjusting for smoking, age, height, gender and weight for FEV1, FVC and MEF50. Duration of exposure to quartz (measured in working years) was a predictor for reduced annual respiratory capacity. An earlier report examined follow-up to 2000 and found a significantly elevated HR of 1.82 (Moshhammer & Neuberger, 2004). Because this study examined lung function, rather than COPD, it was limited in its informativeness.

A cohort of 7,579 US workers participating in a medical screening programme at Department of Energy (DOE) nuclear weapons facilities were followed up for airways obstruction to 2008 (Dement et al., 2010). A variety of definitions for COPD were used to indicate different levels of severity: mild ($FEV_1/FVC < 0.70$ but $FEV_1 \geq 80\%$ of predicted; moderate $FEV_1/FVC < 0.7$ and $50\% \leq FEV_1 \leq 80\%$ of predicted; severe $FEV_1/FVC < 0.7$ and $30\% \leq FEV_1 \leq 50\%$; and very severe $FEV_1/FVC < 0.7$ and $FEV_1 < 30\%$ of predicted. Workers classified as security, scientific/technical or administrative acted as a reference group. Various analyses of FEV_1/FVC were carried out adjusting for age, sex, race, years of DOE work and hobbies outside work. Age standardised prevalence ratios (with NHANES III data as the comparator, adjusted for race, sex, smoking status and years of DOE work, were statistically significantly raised but only elevated between 10 and 20%. There was also evidence of a significant exposure response relation of increasing risk with increased silica exposure score, after adjustment for the same factors as above. This study provides some evidence of an increased risk of COPD and some evidence that there may be a level of exposure at which the RR may be doubled.

3.2 Case-control studies

Four relevant case-control studies were identified and had their data extracted (Calvert et al., 2003; J. Dement et al., 2015; Polatli et al., 2008; Ulvestad et al., 2020) (see accompanying spreadsheet).

A case-control study was carried out in the USA to investigate the association between potential occupational crystalline silica exposure and mortality from a variety of health outcomes including COPD (Calvert et al., 2003). Mortality data were obtained from the US National Occupational Mortality system and contained 95,000 cases and just under half a million controls. Around $\frac{3}{4}$ of cases were male. Controls were matched to cases on the basis of sex, race, state of residence and age group. Cases were ascertained from 1982 to 1985. Each occupation/industry combination was assigned a category of super high, high, medium, low/no silica exposure by an expert panel of 3 NIOSH hygienists. Conditional logistic regression analyses revealed a significantly raised relative risk for ever exposed versus low/no exposure OR 1.12 (1.10 to 1.14) and an increasing risk with increased exposure category – OR for medium versus low/no exposure 1.02 (0.99 to 1.04); OR for high versus low/no exposure 1.29 (1.25 to 1.33); and OR for super high versus low/no exposure 1.47 (1.30 to 1.66). The exposure response trend was positive and $p < 0.001$. There was a lack of information on potential confounding factors such as smoking and co-exposure to other agents causing lung impairment. This study provides support for silica being a cause of COPD, but not for a doubling of the RR.

A small case-control study was carried out in Turkey to investigate pulmonary function changes in silica-exposed workers and silicosis among employers workers exposed to quartz and feldspar (Polatli et al., 2008). Although various pulmonary measurements were assessed, COPD was not defined. Two of 18 office workers and 4 of 27 workers exposed to silica had COPD, giving an unadjusted odds ratio of 1.39 (95% CI: 0.23 to 8.53). This study was too small to contribute much to the evidence base for silica and COPD.

A case-control study examined the risk of COPD attributable to occupational exposures among construction workers (Dement et al., 2015). There were 834 cases and 1243 controls. COPD was used to describe airways obstruction based on an epidemiologic, rather than clinical case definition (i.e. spirometry without bronchodilation). Qualitative exposure indices were developed based on lifetime work and exposure histories. The increase in COPD OR was examined for an exposure at each proportion of the maximum for the exposure index compared to those unexposed. OR (0.25) = 1.21 (1.11 to 1.32); OR (0.50) = 1.46 (1.23 to 1.74); OR (0.75) = 1.77 (1.36 to 2.30); and OR (1.00) = 2.13 (1.50 to 3.03). Analyses were adjusted for smoking status, pack-years of smoking, age, sex, race/ethnicity, blood relative with COPD and BMI. Therefore there was some evidence of a doubling of the RR for those with the highest silica exposure. This study provided evidence in support of high exposure to silica in the construction industry being associated with a double relative risk of COPD.

The final case-control study was carried out in three heavy construction (rock drilling) companies in Norway to determine the effects of exposure to crystalline silica on pulmonary function (Ulvestad et al., 2020). Personal samples were taken between 2015 and 2018 to measure respirable crystalline silica. Occupational histories were obtained from in-depth interviews. Multiple regression was carried out with $FEV_1/FVC * 100$ as the outcome of interest (so not

necessarily COPD). COPD is characterised by airflow limitation that is not fully reversible, with or without symptoms. In this study, the authors reported that they did not test reversibility and could therefore not clearly distinguish between COPD and asthma. Analyses were adjusted for smoking, age, BMI and asthma. Potential co-exposure exist for oil mist and vapours. The study concluded that outdoor rock drillers exposed to crystalline silica had significantly lower pulmonary function than controls, and signs of airflow obstruction. This study provides some evidence for silica being a cause of COPD, but not necessarily at levels that double the RR.

4 Synthesis and Discussion

The current prescription for COPD is in Appendix 2. It is noteworthy that the only occupation prescribed for COPD currently is work as a underground coalminer (for 20 years or more), surface coal miners (for 40 years or more before 1983) or as both (with 2 years underground being equivalent to one year above ground).

Command paper 7253 (COPD – Chronic Bronchitis and Emphysema) was published in 2007¹. The report makes reference to a review of COPD and silica that was commissioned by IAC (Rushton, 2007), as this was one of the exposures deemed by IAC to have sufficiently suggestive evidence such that further investigation was needed. The review by Rushton concluded that:

“Longitudinal studies suggest that loss of lung function occurs with exposure to silica dust at concentrations of between 0.1 and 0.2 mg.m³, and that the effect of cumulative silica dust exposure on airflow obstruction is independent of silicosis. Nevertheless, a disabling loss of lung function in the absence of silicosis would not occur until between 30 and 40 years exposure.”

COPD may occur earlier than this in susceptible individuals or individuals with high exposure. Three reviews were included in our phase 1 report which were published in the last 10 years or so and which considered silica and COPD (Hoet et al., 2017; Möhner & Nowak, 2020; Sen et al., 2016).

The 2016 review by Sen et al concluded that (Sen et al., 2016):

“Silica induces progressive emphysematous changes in the lungs, which is a common phenotype of COPD and study has shown similar evidence among a group of construction workers exposed to various silica-derivatives. A series of investigations have established that silica could be a risk factor for COPD.”

The 2017 review considered whether the current Occupational Exposure Limits for silica were protective for COPD via a review of the epidemiological literature (Hoet et al., 2017). The review concluded that:

“However, no well-founded quantitative estimate can be drawn from these investigations; the available relevant literature does not allow defining a RCS exposure threshold associated with an increased risk of obstructive lung dysfunction, as defined by spirometry, in workers without silicosis. Further research is needed, but, as highlighted in this review, conducting epidemiological studies with both valid exposure and outcome measurements is a real challenge.”

Epidemiological and pathological studies suggest that silica dust exposure can lead to COPD, even in the absence of radiological signs of silicosis, and that the association between cumulative silica dust exposure and airflow obstruction is independent of silicosis (Hnizdo and Vallyathan, 2003).

Heterogeneity in the definitions of COPD add to the complications of interpretation and robustness of the epidemiological evidence presented in this report. There was also a general lack of information on the proportion of dust being silica in the various studies.

¹ https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/243152/7253.pdf

Our search of the epidemiological literature for relevant epidemiological evidence for silica and COPD yielded eight cohort and four case-control studies. The evidence for a causal association between silica and COPD seems quite strong, especially as there seems quite robust evidence for the existence of an exposure-response relationship. However, the studies remain quite heterogeneous in terms of occupations and industries covered, as do the approaches taken for exposure assessment.

There was evidence of a doubled relative risk for manufacturing plants with high silica exposure (foundries, aircraft manufacturing, oil extraction), however it's not clear if the risk was spread across all these facilities or concentration in or two. There was some evidence of a doubling of the RR in another cohort study of foundry workers, suggesting that these may be the workers in the previous study who were most at risk of COPD. There was a suggestion of an increased RR at a nuclear weapons facility which suggested there may be a silica exposure level at which the RR was doubled. A case-control study in construction showed evidence of a doubled RR of COPD.

5 Conclusions

We have summarised the relevant occupational epidemiological evidence for silica and, and based on this review alone it is not possible to recommend prescription in relation to silica and COPD in occupational circumstances covered by this review. However, there are two sets of occupational circumstances that seem worthy of further consideration by searching for studies in relevant occupations and industries, not just studies informative about silica exposure. These are foundry workers and construction workers, and it may be appropriate for IAC to consider doing this as a matter of priority, noting that there are potentially other risk factors relevant for COPD in these industrial settings. It is possible that there are other occupational circumstances where silica exposures are relatively high (e.g. manufacture or installation of kitchen worktops) and we would suggest that IAC also considers these.

6 References

- Calvert, G. M., Rice, F. L., Boiano, J. M., Sheehy, J. W., & Sanderson, W. T. (2003). Occupational silica exposure and risk of various diseases: an analysis using death certificates from 27 states of the United States. *Occup Environ Med*, 60(2), 122-129. <https://doi.org/10.1136/oem.60.2.122>
- Carneiro, A. P. S., Teixeira, V., Silveira, A. M., Araujo, S. C. B. & Algranti, E. 2022. Non-malignant silica-related diseases in a specialized outpatient clinic. *Occup Med (Lond)*, 72, 394-402.
- Cherry, N., Harris, J., McDonald, C., Turner, S., Taylor, T. N., & Cullinan, P. (2013). Mortality in a cohort of Staffordshire pottery workers: follow-up to December 2008. *Occup Environ Med*, 70(3), 149-155. <https://doi.org/10.1136/oemed-2012-100782>
- Dement, J., Welch, L., Ringen, K., Quinn, P., Chen, A., & Haas, S. (2015). A case-control study of airways obstruction among construction workers. *Am J Ind Med*, 58(10), 1083-1097. <https://doi.org/10.1002/ajim.22495>
- Dement, J., Welch, L., Ringen, K., Quinn, P., Chen, A., & Haas, S. (2015). A case-control study of airways obstruction among construction workers. *American Journal of Industrial Medicine*, 58(10), 1083-1097. <https://doi.org/https://doi.org/10.1002/ajim.22495>
- Dement, J. M., Welch, L., Ringen, K., Bingham, E., & Quinn, P. (2010). Airways obstruction among older construction and trade workers at Department of Energy nuclear sites. *Am J Ind Med*, 53(3), 224-240. <https://doi.org/10.1002/ajim.20792>
- Dement, J., Welch, L., Ringen, K., Quinn, P., Chen, A. & Haas, S. 2015. A case-control study of airways obstruction among construction workers. *Am J Ind Med*, 58, 1083-97.
- Graber, J. M., Stayner, L. T., Cohen, R. A., Conroy, L. M., & Attfield, M. D. (2014). Respiratory disease mortality among US coal miners; results after 37 years of follow-up. *Occup Environ Med*, 71(1), 30-39. <https://doi.org/10.1136/oemed-2013-101597>
- Grahn, K., Gustavsson, P., Andersson, T., Lindén, A., Hemmingsson, T., Selander, J., & Wiebert, P. (2021). Occupational exposure to particles and increased risk of developing chronic obstructive pulmonary disease (COPD): A population-based cohort study in Stockholm, Sweden. *Environ Res*, 200, 111739. <https://doi.org/10.1016/j.envres.2021.111739>
- Hochgatterer, K., Moshhammer, H., & Haluza, D. (2013). Dust is in the air: effects of occupational exposure to mineral dust on lung function in a 9-year study. *Lung*, 191(3), 257-263. <https://doi.org/10.1007/s00408-013-9463-7>
- Hoet, P., Desvallées, L., & Lison, D. (2017). Do current OELs for silica protect from obstructive lung impairment? A critical review of epidemiological data. *Crit Rev Toxicol*, 47(8), 650-677. <https://doi.org/10.1080/10408444.2017.1315363>
- HSE Workplace Health Expert Committee (WHEC). (2019). Exposure to respirable crystalline silica and the requirement to carry out health surveillance (Evidence Review Paper, Issue).
- Hnizdo, E. & Vallyathan, V. 2003. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: a review of epidemiological and pathological evidence. *Occup Environ Med*, 60, 237-43.

- Lenander-Ramirez, A., Bryngelsson, I. L., Vihlborg, P., Westberg, H., & Andersson, L. (2022). Respirable Dust and Silica: Respiratory Diseases Among Swedish Iron Foundry Workers. *J Occup Environ Med*, 64(7), 593-598. <https://doi.org/10.1097/jom.0000000000002533>
- Mazitova, N. N., Saveliev, A. A., Berheeva, Z. M., & Amirov, N. (2012). COPD and occupation: a retrospective cohort study of industrial workers. *Arh Hig Rada Toksikol*, 63(3), 345-356. <https://doi.org/10.2478/10004-1254-63-2012-2178>
- Minette, A. 1989. Questionnaire of the European Community for Coal and Steel (ECSC) on respiratory symptoms. 1987--updating of the 1962 and 1967 questionnaires for studying chronic bronchitis and emphysema. *Eur Respir J*, 2, 165-77.
- Moshhammer, H., & Neuberger, M. (2004). Lung cancer and dust exposure: results of a prospective cohort study following 3260 workers for 50 years. *Occup Environ Med*, 61(2), 157-162. <https://doi.org/10.1136/oem.2002.001255>
- Polatli, M., Tuna, H. T., Yenisey, C., Serter, M., & Cildag, O. (2008). Lung function and IFN-gamma levels in the sera of silica-exposed workers. *J Interferon Cytokine Res*, 28(5), 311-316. <https://doi.org/10.1089/jir.2007.0093>
- Reynolds, C. J., MacNeill, S. J., Williams, J., Hodges, N. G., Campbell, M. J., Newman Taylor, A. J., & Cullinan, P. (2017). Chronic obstructive pulmonary disease in Welsh slate miners. *Occup Med (Lond)*, 67(1), 20-25. <https://doi.org/10.1093/occmed/kqw147>
- Rushton, L. (2007). Chronic obstructive pulmonary disease and occupational exposure to silica. *Rev Environ Health*, 22(4), 255-272. <https://doi.org/10.1515/reveh.2007.22.4.255>
- Sen, S., Mitra, R., Mukherjee, S., Das, P. K., & Moitra, S. (2016). Silicosis in Current Scenario: A Review of Literature. *Current Respiratory Medicine Reviews*, 12, 56-64.
- Ulvestad, B., Ulvestad, M., Skaugset, N. P., Aaløkken, T. M., Günther, A., Clemm, T., Lund, M. B., & Ellingsen, D. G. (2020). Pulmonary function and high-resolution computed tomography in outdoor rock drillers exposed to crystalline silica. *Occupational and Environmental Medicine*, 77, 611 - 616.

Appendix 1 – Data Extraction Spreadsheet and List of All the References Considered



Data extraction Si & COPD final for IIAC.xls



Silica_and_COPD_All_Papers.xlsx

Appendix 2 – Current prescription for COPD

Disease Number	Name of Disease or Injury Miscellaneous conditions not included elsewhere in the list	Type of job Any job involving
D12	<p>Chronic obstructive pulmonary disease – COPD where, with maximum effort, where there is evidence of a forced expiratory volume in one second which is:</p> <p>(i) at least one litre below the appropriate mean value predicted, obtained from the following prediction formulae which give the mean values predicted in litres:</p> <ol style="list-style-type: none"> 1. For a man, where the measurement is made without back-extrapolation, $(3.62 \times \text{Height in metres}) - (0.031 \times \text{Age in years}) - 1.41$; or, where the measurement is made with back-extrapolation, $(3.71 \times \text{Height in metres}) - (0.032 \times \text{Age in years}) - 1.44$ 2. For a woman, where the measurement is made without backextrapolation, $(3.29 \times \text{Height in metres}) - (0.029 \times \text{Age in years}) - 1.42$; or, where the measurement is made with back- extrapolation, $(3.37 \times \text{Height in metres}) - (0.030 \times \text{Age in years}) - 1.46$ or <p>(ii) less than one litre.</p>	<p>Exposure to coal dust (whether before or after 5th July 1948) by reason of working–</p> <ol style="list-style-type: none"> (a) underground in a coal mine for a period or periods amounting in aggregate to at least 20 years; (b) on the surface of a coal mine as a screen worker for a period or periods amounting in aggregate to at least 40 years before 1st January 1983; or (c) both underground in a coal mine, and on the surface as a screen worker before 1st January 1983, where 2 years working as a surface screen worker is equivalent to 1 year working underground, amounting in aggregate to at least the equivalent of 20 years underground. Any such period or periods shall include a period or periods of incapacity while engaged in such an occupation.



www.iom-world.org

[email: iom@iom-world.org](mailto:iom@iom-world.org)

IOM Edinburgh

Research Avenue North
Riccarton, Edinburgh,
EH14 4AP

Tel: 0131 449 8000

IOM Chesterfield

Tapton Park Innovation Centre
Brimington Road, Tapton,
Chesterfield S41 0TZ

Tel: 01246 383 110

IOM Stafford

Brookside Business Park,
Cold Meece, Stone
Staffordshire, ST15 0RZ

Tel: 01785 333 200