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REVIEW ARTICLE

Exposure to cement dust and respiratory health for communities residing near cement factories: A systematic review of the literature

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ABSTRACT

Objective: To examine the strength of evidence through systematic review of published literature on the association between effects of exposure to cement dust on respiratory health of communities residing near cement plants. ***Design and data sources:*** A systematic search and review of observational studies in Medline, Embase, and Cumulative Index to Nursing and allied Health Literature (CINAHL), and other sources was conducted. ***Eligibility criteria:*** Peer reviewed articles, published from 1996 to 2022 that investigated effects of exposure to cement dust on human respiratory health (pulmonary functions and symptoms) were included. The studies must have been conducted in communities residing near a cement factory; were original research and written in English. **The search key words were:** cement, cement dust, cement dust exposure, respiratory health, cement respiratory health effect, cement dust exposure respiratory effect, cement pulmonary function, and cement dust pulmonary health, community. ***Results:*** 443 studies were retrieved and screened. Of these, 12 met the inclusion criteria. Out of the 12, seven were of moderate quality based on NIH assessment. Seven of these studies were cross sectional study design. Five studies performed actual measurement of ambient concentration of particulate matter (PM) while the rest assumed high exposure levels based on other studies' findings. Where exposure was conducted, environmental monitoring rather than more precise methods of measuring personal exposure. While most studies reported higher levels of $PM_{2.5}$ and PM_{10} in the exposed compared to the control communities, associations between exposure and respiratory ill health were not inconsistently demonstrated. Additionally, none of the studies conducted source apportionment and chemical characterisation of the PM in the ambient. ***Conclusion:*** This review shows that despite showing higher levels of PM in exposed communities, the existing evidence is insufficient to support an association between exposure to cement dust and respiratory ill health. The low quality of the studies and differences in methodology could explain the disparities in the findings. To improve the quality of evidence, future studies should include panel studies, personal monitoring of the exposure, source apportionment and chemical characterisation coupled with using standardized measurement tools for exposure and outcome

Introduction

The cement industry has grown progressively in both developing and developed countries with an estimated global production of 4.18 billion tons in 2014.[1] The production of cement involves the following three steps: firstly - preparation of raw materials (limestone, shale and sand) which involves mixing/homogenising, grinding and preheating (drying) to produces the raw mill. Secondly, burning of raw mill at high temperatures (900-1500°C) in the pre-calciner to form cement clinker in the kiln. Thirdly, after cooling, the clinker is ground together with the additive. The finished material is then stored in silos, ready for dispatch in bags or bulk. Though necessary for infrastructural development, the production of cement involves the release of undesired emissions such as carbon monoxide (CO), carbon dioxide (CO_2), nitrogen dioxide (NO_2), sulphur dioxide (SO_2) and particulate matter (PM).[2] These emissions significantly contribute to pollution of the ambient air with subsequent effects on the health of the public.[3] PM is of special environmental concern because it constitutes the largest proportion of the emission. The major component of cement dust are particles of size 0.05 to 5.0 micrometer.[4] These particles once inhaled will penetrate into the gas exchange regions of the lungs placing the workers and residents of communities at risk of respiratory illnesses.[5,6] Thus the inherent dusty operation of cement production results in dust exposure of factory workers and residents of communities situated near cement plants.

Some epidemiological studies have reported reduced pulmonary function indices, measured as forced expiratory volume in one second (FEV1), forced vital capacity (FVC), ratio of forced ventilatory capacity in one second and forced vital capacity (FEV1/FVC), and peak expiratory flow rate (PEFR) in individuals who are exposed to cement dust in communities residing near cement factories.[7–9] However, other studies have reported no statistically significant relationship between exposure to cement dust and pulmonary function impairment. Smoking is known to be a confounder for lung health, however not all studies control for it. In one study where smoking was considered, it was found that, smokers exposed to occupational cement dust had significantly lower values for FEV1, FEV1/VC, FEV1/FVC and PEFR compared with the unexposed smokers.[10] Conversely, there was no statistical difference in the pulmonary function indices between smokers and non-smokers in both the exposed and unexposed groups. Nevertheless, smokers in the exposed group did exhibit lower ventilatory function compared to smokers in the unexposed group suggesting that smoking aggravates epithelial damage caused by cement dust.[7] In addition to pulmonary function indices, non-specific respiratory symptoms including cough, wheeze, dyspnoea, sneezing, and phlegm are frequently reported in exposure to cement dust studies.[10–12] However, cough was defined differently, across studies, for instance Legator et al.[13] reported 'persistent cough' while others defined cough as 'morning cough' and 'night cough' and combined to 'composite cough.[14] As a result of the inconsistent findings, there is lack of consensus, on the association between exposure to cement dust and respiratory ill health in occupational and residential settings.

Methods

Literature search

The systematic review protocol is registered: CDR 42017081234. PubMed, Embase and CINAHL were searched for qualifying studies between 2nd December 2017 and 20th February 2018 and a second search was conducted from May 2022 to August 2022 using a combination of the following key search words: community, *cement dust*, $PM_{2.5}$ *and* PM_{10} for the exposure; *and respiratory health, cough, phlegm, bronchitis, chronic bronchitis, pneumonia, asthma and reduced lung function, FEV1, FVC and PEFR* for outcome. A further search was conducted for relevant published and unpublished reports from authors' reference list of eligible and relevant articles during both period of the literature search. The "Google" engine was used to search for abstracts, conference proceedings and unpublished studies. ML conducted the initial search. The studies identified during the search were later screened by two independent reviewers (MN and EN) through reviewing titles and abstracts to identify potentially eligible studies. This was followed by full text screening of the potentially eligible studies. Any disagreements regarding eligibility were resolved by consensus with the help of a third reviewer (SS).

Eligibility criteria

Only studies meeting the following criteria were included:

i. Peer reviewed comparative studies of any design published from 1996 to 2022,
ii. Population included communities near a cement factory and a control group
iii. Measured exposure to cement dust by central monitoring of ambient air or personal exposure or both, or used proxy measures based on routine data
iv. Measured respiratory ill health outcome: either as symptoms such as cough, phlegm, sinusitis, asthma, emphysema, bronchitis or lung function indices (FEV1, FVC, FEV1/FVC ration, PEFR) or both.
v. Original research and written in English,
vi. Studies conducted any part of the world.

Data extraction

A Microsoft excel spreadsheet was used to extract the data that included full description of study (name of author, title of study, name of journal, year of publication, location), period of study, study design, age and sex of study participants, type of exposures assessed (particulate matter of aerodynamic 2.5 ($PM_{2.5}$), particulate matter of aerodynamic 10 (PM_{10}), suspended particulate matter (SPM), and respirable suspended matter (RSPM)); and health outcomes assessed either as respiratory symptoms (cough, wheeze, dyspnea, nose irritation, asthma, pneumonia, acute or chronic bronchitis) and/or lung function (FEV1, FVC, ratio FEV1/FVC, PEFR).

Assessment of methodological quality of eligible studies

The NIH Quality Assessment Quality Tools for Observational Cohort and Cross Sectional and Quality Assessment Cross sectional studies[15] was used to assess methodological quality and risk bias on all studies that met the eligibility criteria. Each study was evaluated for whether there was a clear research question or research objectives; clearly defined study population; sample size justification or participation rate for eligible persons; clearly stated inclusion and exclusion criteria; defined exposure and outcome variables. Cohort studies or panel were assessed whether sufficient timeframe was allocated in order to reasonably expect to see association between exposure to cement dust and respiratory outcomes. Cross sectional studies were assessed whether independent variables were clearly defined and implemented consistently across all study participants. Furthermore, we assessed whether the investigators were able to confirm that the exposure/risk occurred prior to the development of the condition or defined a participant as a case and controlled for potential confounders. Each of the above attributes was scored as either: "Yes", "No", "Not applicable" or "Not reported". The overall score for each individual study was allocated a percentage to allow for comparability.[16,14]

Studies were classified according to how the investigators reported. Where the study design was not explicitly stated, the authors used standard definitions to assign the study design, otherwise the studies were deemed as unclassified.

Results

A total of 443 articles were retrieved from PubMed (41), Embase (190) and CINAHL (139) while 63 studies were from other sources. Two hundred and ninety (290) studies were excluded as irrelevant. Of the remaining143 studies, 84 investigated effects of exposure to cement dust on the health of factory workers, 32 were duplicates, 17 were commentaries, 2 were written in Spanish, 2 ex-vivo and 2 were toxicological studies while one did not meet the criteria having investigated the effect of PM_{10} on non-respiratory health of school children and therefore excluded. Two studies were further excluded because they assessed exposure concentrations without associating to respiratory ill health. In communities near cement factories. Eleven (11) studies, met the inclusion criteria for analysis but one was further omitted from analysis for having been of weak methodological quality (figure 1). Of the remaining ten (10) studies, five were conducted in Asia (Korea (2) and India (3)), one in USA (Texas) and five conducted in Africa (Zambia (3), Nigeria (1) and Ethiopia (1)). Details of the studies, are presented in Table 1.

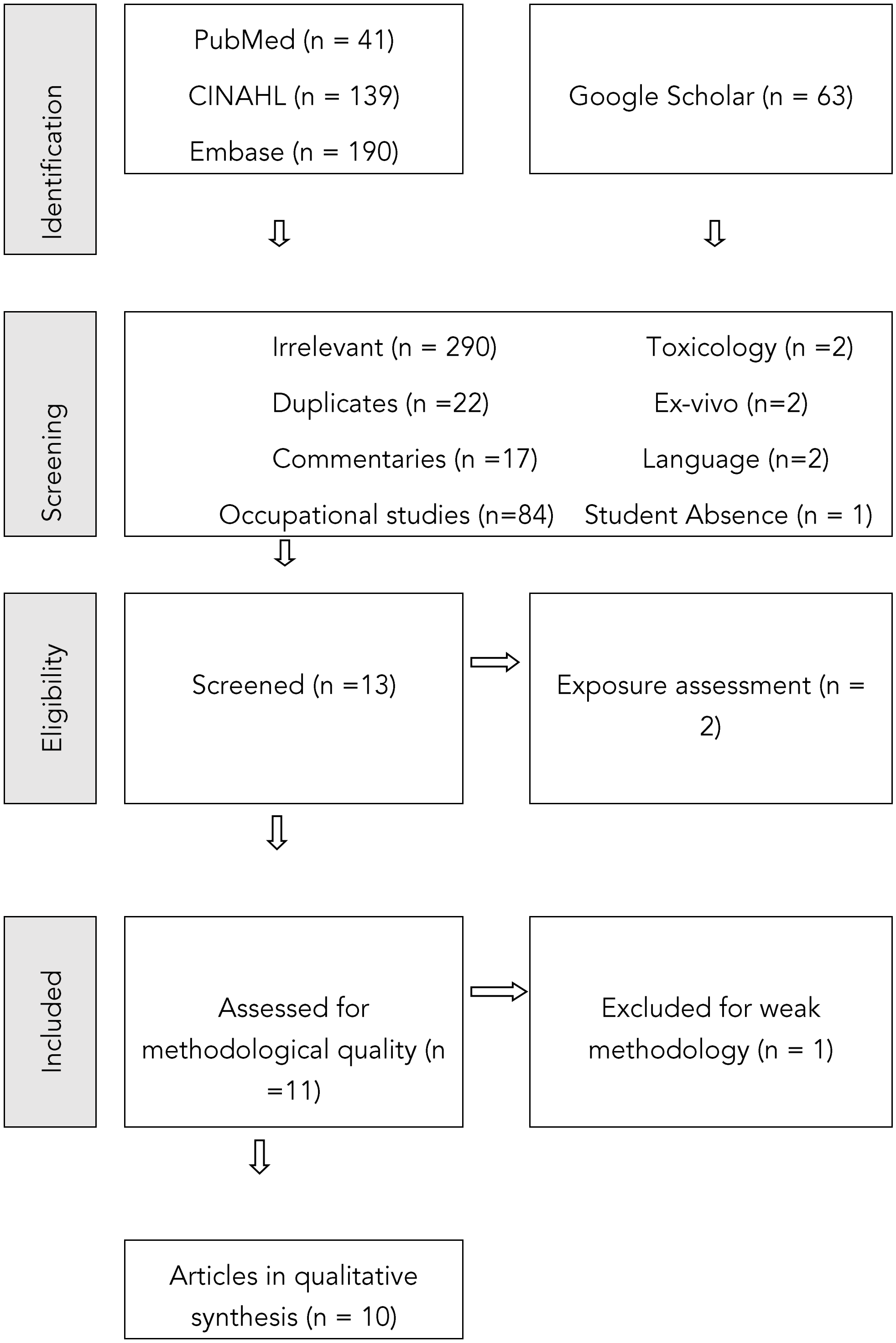

Figure 1: Selection procedure

Table 1. Summary of studies conducted in community settings

Author	Participants	Exposure	Outcome	Confounders adjusted
		Cross sectional studies		
Merenu et al 2015, Nigeria	Expo n=244 (radius1km) Cont n=270 from plant	NM	FVC 2.5L (p=0.001 95% CI 0.21,0.59) FEV1 2.2 (p=0.024 (95% CI 0.03, 0.37) FEV% 84.9% (p=0.000295% CI -7.88, 2.52]	NR
Nkhama et al. 2015,Zambia[a]	Exp n=223 (1km) Cont n=197 (18 km)	NM	Eye irritations,78.2 vs. 49.9% (p value<0.001) Nose irritation 66.9 vs. 29.4%, (p-value<0.001) Sinus irritations, 73.7 vs. 53.3% (p value<0.001)	Age, gender, marital status, occupation, smoke status, Lighting/cooking
Nkhama et al 2015, Zambia[a]	Exp n=223 (1km) Cont n=197 (18 km)	NM	Cough morning 37.6 vs. 23.5 23.5%, (p value =0.003); Cough night (48.1 vs 14.6, p value<0.001); Increased cough with phlegm" (55.9 vs. 13.9%; p value <0.001). (45.0 vs. 30.6%, p value =0.002 Pneumonia 20% vs. 3.5%; (p value<0.001).	Residence, age, gender, Smoking status, presence of carpet, lighting and cooking Energy
Sul Ha Kim et al. 2013	Exp n=319 (within1km) Cont n=1292 (>5 km)	45.5μg/m^3, 95% CI 37.8-53.3) >1km [PM_{10}]; 38.5μg/m^3 95% CI 32.3-44.7 [PM_{10}]; 25.5μg/m^3 95% CI 18.7 -32.3) <1km [$PM_{2.5}$]; 19 (95% CI 14.1-24.6 >5 km [$PM_{2.5}$]	Obstructive =9.7% MEG; 85% (LEG) Restrictive =21.6% (MEG), 12.4 (LEG) OR 2.63 95%CI 1.50-4.611	NR

Author	Participants	Exposure	Outcome	Confounders adjusted
Cross sectional studies				
Merhaj et al. 2013, India	Exp n=2000 (~2-31km) Cont n= NS (~40 km)	TSPM, PM_{10}, RSPM 8805µg/m^3	Eye irritation 97 vs. 12 % Shortness of breath 96 vs. 10% Cough 96 vs 15% Wheeze 96 vs. 21% Asthma 49 vs. 1% Chronic bronchitis 57 vs. 0 Emphysema 9vs.0	
Legator et al. 1998, Texas	Expo n=58 Cont n=54	NM	Coughing up blood, 2% v. 0(p-value 1.00) Pneumonia, 4 vs.5% (p-value 1.00) Lung disease, 4 vs. 0 (p-value 0.49) Wheezing, 11 vs. 2% (p- value: 0.20 Persistent cough, 15 vs. 5% (p-value: 0.16) Persistent bronchitis, 11 vs.2% (p-value:0.20) Shortness of breath, 18 vs.5% (p-value: 0.09)	Smoking
Case controls studies				
Hyun Seung Lee et al 2016 Korea	Expo n= 1.046 (1 km) Cont n= 317 (>5 km)	NM	14.3% MEG with 17.8% HRCT OR 2.56 (95% CI 1.64 – 3.99)	
Panel study studies				
Nkhama et al 2017	Expo n=63(1 km) Cont n=55 (18 km)	$PM_{2.5}$, PM_{10} ranged from 2.39-24.93 µg/m^3 and 7.07-68.28 µg/m^3 Exposed vs control community 1.69 -6.03 µg/m^3	Cough, 46.3 vs. 13.8‡; Phlegm production, 41.2 vs 9.6‡ Nose irritation, 49.0 vs.12.5‡; Wheeze 13.9 vs 3.9‡ FEV1† 6% points lower than control, FVC† 4% points lower than control Cough OR 1.02(95%1.01-104) Phlegm=1.02 (95%1.01- 10.3) Nose irritation OR 1.01 (95% 1.01,1.02)	

Author	Participants	Exposure	Outcome	Confounders adjusted
		Unclassified Studies		
Hyun Seung Lee et al,2016 Korea	Expo n=1,046 (1 km) Cont n=317 (>5 km)	NM	9.1% LEG with 11.4% on HRCT; 14.3% MEG with 17.8% HRCT	
Priyanka et al. India, 2013.	N	SPM -281.07 to 342.45μg/m^3 322.29 to 387.20 μg/m^3 172.25 to 213 μg/m^3	RS= 28.35 - 52.54% (five sites)	NR
Tiwari et al	Expo n=200 (< 1.5 km)	SPM- range 150.84 (rainy) to 340.1μg/m^3 (winter) RPM range 83.48 (rain) to 132.28μg/m^3 (winter)	Respiratory disease 19.63%; Eye irritation 17.78%,	

Cont = control, Expo = exposed,; total dust; RD, respirable dust; RPM, respiratory particulate matter; SPM, suspended particulate matter; TSPM, total suspended particulate matter; RSPM, respirable suspended particulate matter; S, respiratory symptom; LFT, lung function test; FEV1, forced expiratory volume in one minute; FVC, forced ventilatory volume; LEG, less exposed group; MEG, more exposed group; NM, not measured; [a], P<0.05; [b], P<0.0001; C, researchers assumed exposure levels based on existing literature; NS, not stated; HRCT, high resolution computed tomography.† percentage predicted value

Description of the eligible studies

Assessment of individual studies: Bias assessment

Using the NIH quality assessment criteria,[13] studies with total scores equal or more than 90% were graded as strong (low risk of bias), those scoring 70-90% as fair quality (moderate risk of bias) while those scoring below 70% were considered weak (high bias). Three studies were assessed to be of low risk of bias (good quality), seven were of moderate risk of bias (fair quality) while one was graded as having high risk of bias (weak) and excluded from analysis. For the 10 studies that remained, seven studies used cross sectional study design.[13,14,17–21] one used panel.[22] while two studies were not clearly reported by the authors.[23,24] Most studies scored moderate because they neither clearly defined the exposure measurement, nor adequately defined the exposed and control populations while others did not measure the exposure and failed to control for confounders.

Population

Definition of study groups varied widely among studies. The majority of the studies used the community near a cement factory as exposed while identifying communities far from the plant as control. The exposed groups were drawn from communities near cement plants within a radius of range 1-14 km and mostly situated on the leeward side of the cement plants. In these studies, the distance from source for the control groups ranged from 14 – 19.2km[17,19,22–25] to 70km.[18] However, two studies.[20,21] identified two groups from the same community, based on distance from the plant, as representing different exposure levels; 1-5 km for the more exposed group (MEG) and greater than 5 km for the least exposed (LEG). Additionally, the definition of individual participants differed from one study to another; attributes included the age range and the number of years a participant should have lived in the exposed community prior to the study. For instance, Nkhama et al.[19] included participants aged more than 15 – 59 years old, while Sul et al[20] and Seung et al.[21] included participants aged 40 years and older while a study in the USA[13] used participants aged 16 – 76 years. The number of years that participants lived in the exposed community ranged from two[19,22,25] to 5 years[18]. The remainder of the studies did not specify the age range of the participants.

Exposure assessment

Broadly, two approaches of assessment of the exposure were used. Five of the ten studies carried out actual measurements of PM concentrations in the ambient air.[17,20–24] The rest assumed that the concentration of PM in the ambient air was high in the respective exposed communities based on previous studies and/or retrospective data routinely collected by environmental monitoring agencies. All of the studies that measured the exposure used environmental monitoring employing a stationary/central station in the study community. The type of exposure measured varied from one study to another but included $PM_{2.5}$, PM_{10}, suspended particulate matter (SPM), respirable particulate matter (RPM) and atmospheric dust in various permutations. Regardless of the exposure of interest that was measured, the five studies consistently demonstrated elevated PM concentrations in the exposed communities compared to their respective controls. For instance, Mehraj et al.[17] measured total SPM and RSPM using high volume respirable sampler and reported an average of 1208.78 μg/m^3 SPM and 880 μg/m^3 RSPM, respectively. Nkhama et al.[22] measured 24-hour $PM_{2.5}$ and PM_{10} concentrations in the ambient air at a fixed site for the exposed similar results: the mean seasonal concentrations of $PM_{2.5}$ and PM_{10} as high as 24.93 μg/m^3 and 68.28 μg/m^3 respectively in the exposed. Furthermore, Tiwari et al. assessed ambient concentrations of SPM and RPM over three seasons in four sites during winter, summer and rainy months.[23] SPM ranged from 150 to 275 μg/m^3 while RPM ranged from 78.49 to 105.15 μg/m^3 with the lowest concentration reported in the rainy and the highest in winter.[23] The findings from these studies were above limits set in the Indian National Air Quality Standards; 70 μg/m^3 annual and 150 μg/m^3 for 24-hour for PM_{10}, and 75 μg/m^3 in 24-hour mean concentration for $PM_{2.5}$ for urban areas.[20] The concentrations of SPM and RSPM in the ambient air were above the permissible limit of 200μg/m^3 for SPM, 100 μg/m^3 for RPM. Nevertheless, the studies used different methods of measuring exposure: 24-hr duration using vacuum pump[22], 8-hour duration using respirable dust samplers,[24] high volume respirable sample.[17]

The five studies[13,18–20,25] that did not measure concentration assumed high exposure levels in the exposed communities based on previous studies or on data routinely collected by other agencies. For instance, Legator et al.[13] in Texas conducted a randomised cross-sectional study and assumed exposure concentration levels based on routine data retrospectively collected by the Texas Natural Resource Conservation Commission, 1998.[26]

Health outcomes assessment

The health outcomes measured across all the reviewed studies were classified as symptoms and respiratory diseases, and pulmonary functions. Studies investigated symptoms in different combinations; common symptoms being cough, wheeze, production of phlegm and difficulty in breathing while respiratory diseases included asthma, emphysema, bronchitis and pneumonia. Other health outcomes measured were irritations of mucous membranes of the sinuses, eyes and nose. In all the studies, symptoms or diseases were participant self-reported and not diagnosed by health professionals. Most of the studies defined the symptoms/disease according to one of the following standards: American Thoracic Society (ATS), European Respiratory Society (ERS) or the British Thoracic Society (BTS).[27,28] The symptoms and diseases were measured as either prevalence or incidence, and measures of association were also reported where applicable. Pulmonary function was measured FEV1, FVC, FEV1/FVC, VC and PEFR. These lung indices were reported as percentage predicted or absolute numbers. The following sections discuss each health outcome separately.

Respiratory symptoms

Four studies investigated cough: Nkhama et al (cross sectional, exposed N=223; control, N=197);[25] Nkhama et al (panel study, exposed N=67; control N=55);[22] Mehraj et al. (cross sectional study, exposed N=1000, control N=1000);[17] Legator et al (cross sectional, exposed N=45, control N=43).[13] Nkhama et al.[25] and Merhaj et al.[17] used the ATS and World Health for aging and Health Council recommended guidelines respectively, while Legator et al.[13] did not report how cough was measured. In a cross sectional study, Nkhama et al.[18] reported higher proportion of participants with cough in the exposed compared to the control community (57% vs. 17.4%; p-value=0.001). Moreover, regardless of the time of the day more people in the exposed community, compared to the control, reported suffering from cough: "cough in the morning" (37.6 vs. 23.5%, p value=0.003) and "cough at night", (48.1 vs. 14.6, p value <0.001). These results were consistent with the two other cross-sectional studies conducted by Merhaj et al.[17] and Legator et al.[13] 96% vs. 15% and 15% vs. 5% (p-value=0.16) respectively. In the fourth study, a panel by Nkhama et al.[22] the incidence rate (per 100-person days) for cough was 46.3 and 13.8 in the exposed and control communities respectively.

Two of these studies[22,25] further investigated whether there was association between exposure and cough. Nkhama et al., in both cross sectional and panel studies, demonstrated statistically significant association between exposure to cement dust and cough; OR 5.64 (95% CI 3.63-8.67; p value < 0.001 and OR 1.02 (95% CI 1.01-1.04) for the cross sectional and panel studies respectively. Both studies controlled for residence, age, marital status, education, occupation, smoking status, floor carpet and type of energy used for cooking and sex, age, height, weight, smoking history, socioeconomic status, asthma and meteorological variables, respectively. However, Legator et al.[13] reported a non-statistically significant difference in prevalence of cough between the exposed and control communities (15% vs. 5% of cough, p= 0.16) after adjusting for smoking only.

The prevalence of wheeze was higher in the exposed community compared to the control in three cross sectional studies[13,17,25] (Mehraj et al., 96% vs. 21%; Nkhama et al.,45.0 vs. 30.6%, p value=0.002; Legator et al., 11% vs.2% p=0.20). One panel study[22] demonstrated that the incidence rate per 100 person-days was higher in the exposed community (13.9 vs. 3.9). However, an association between exposure cement dust and wheeze was found in only one of these studies (OR1.60 95% (1.01-2.54, p=0.045)).[14]

Two studies[13,17] both cross sectional, investigated shortness of breath. A higher prevalence of shortness of breath was reported in the exposed compared to their respective control communities. Mehraj et al.[17] reported 96% vs. 10% while Legator et al.[13] 18% vs. 5% (p-value=0.20) respectively.

Other symptoms investigated were phlegm[22,25] irritation of the mucous membrane such as eye,[17,19] and nasal irritation.[19,22] In all the studies, the prevalence of the symptoms was found to be higher in the exposed compared to the respective control communities. Nkhama et al.[14,19,22] demonstrated associations between exposure to cement dust and phlegm, nasal and eye irritations in cross sectional and panel studies. The exposed community in cross sectional study was three times more likely to report phlegm (OR 3.30 (95% CI 2.04, 5.34), p value < 0.001), while this reduced significantly towards null in the panel study[22] (OR=1.02 (95%1.01, 10.3). Similarly, nasal irritation was four times more likely to be reported in the exposed community (OR=4.36 (95% CI 2.96, 6.55), p value < 0.001) in the cross sectional study[19] while the likelihood reduced to slightly above the null in the panel study[22] (OR=1.01 (95% 1.01,1.02). Similarly, Nkhama et.al,[1919] in cross sectional study, found that eye irritation was at least twice as likely to be reported in the exposed community compared to the control (OR 2.50 (95%CI 1.65-3.79, p value < 0.001) after adjusting for time where respondents spent most of the time, location of the kitchen and source of energy for cooking. However, the researchers did not investigate this symptom in the panel study. Mehraj at el.[17] also reported higher prevalence of eye irritation (96% vs. 12%), although, the study did not investigate whether there was an association.

In addition to the above symptoms, some studies investigated the prevalence of various respiratory diseases. These included asthma, chronic bronchitis, pneumonia and emphysema in different permutations. The prevalence of all the respiratory diseases was higher in the exposed communities compared to the control. Merhaj et al.[17] and Nkhama et al.[14] demonstrated that asthma was 49% vs 1% and 9.7% vs 1.1% in the exposed compared to the control, respectively. Additionally, the exposed community was almost six times more likely to report asthma compared to the control (adjusted OR 5.67, 95% CI 2.00,16.05, p-value=0.003).[14] Similarly, the prevalence of chronic bronchitis was reported higher in the exposed than in the control communities; 57% vs. 0, 11% vs. 2% (p=0.20) and 2.3% vs. 0.8% (p=0.090) in Merhaj et al., Legator et al., and Nkhama at el., respectively.[13,14,17] However, Legator et al.[13] found that the difference in prevalence between the two communities was not statistically significant while Nkhama et al.[25] found only marginal association between residence in the exposed community and chronic bronchitis (OR 3.45 (95% CI 0.49 – 24.56; p-value=0.098). In the case of pneumonia, Legator et al.[13] and Nkhama et al.[25] found the prevalence to be higher in the exposed communities: 5% vs 4% (p=1.00) and 20.1% vs. 3.5% (p value<0.001), respectively. Furthermore, Nkhama at el.[25] demonstrated that the exposed community was four times more likely to report pneumonia compared to the control (OR 4.38 95% CI 1.28 – 14.95; p-value=0.021).

The more exposed group (MEG) was more than twice likely to have emphysema compared to the least exposed group (LEG) OR 2.56 (95 % CI 1.64–3.99) after adjusting for sex, age, BMI, smoking history, residency, and use of firewood.[20] Similarly, Merhaj et al.[17] and Legator et al.[13] measured emphysema and reported higher prevalence in the exposed compared to the control: 9% vs.0% and 2% vs. 0% (p-value=1.00) respectively.

Nkhama et al.[22] also demonstrated that a 1 μg/m^3 increase in $PM_{2.5}$ increased the odds of cough, phlegm and nose irritation by about 2% controlling for season, smoking status and asthma. However, it had an opposite effect on the odds of wheeze (p-value < 0.05). Similarly, an increase in PM_{10} concentration reduced the odds of cough, phlegm, wheeze and cough, but was only statistically significant for phlegm and nose irritation. Furthermore, PM_{10} seemed to have delayed effect with regards to phlegm and nose irritation; a statistically significant effect for phlegm and nose irritation was observed 3–5 days after exposure (lag 3 and 5); phlegm lag 5 [OR = 1.00 (0.06–1.00)]; and nose irritation lag 3 [OR = 1.00 (1.00–1.10)] and wheeze [OR = 1.00 (0.06–1.01)].

Pulmonary Function

Three studies investigated pulmonary functions.[18,20,22] A Nigerian study demonstrated that the mean FVC, FEV1 and FEV% were found to be lower in the exposed compared to the control communities.[18] Furthermore, the same study found obstructive lung impairment more frequently in the exposed community than the control (17.2% vs. 7.8%; (p=0.0215)).

The second study,[20] evaluated both a pre- and a post-bronchodilator pulmonary function test (PFT) on FEV1 and FVC for a more exposed group (MEG) (N=318) living within a 1 km radius of a cement plant and a less exposed group (LEG) (N=129) living more than 5 km away from the same plant. The FVC% predicted value estimated using the Korean equation showed both lung function impairment to be higher in the exposed compared to the control communities; 9.7% vs. 8.5% for obstructive and 21.6% vs. 12.4% restrictive types of impairments. In that study, adjusting for sex and age, the exposed group was 2.63 (95% CI 1.50,4.61) and 2.55 (95% CI 1.37,4.76) more likely to develop the obstructive and restrictive types respectively compared to the control. The third study[22] found lower performance on lung indices in the exposed community compared to the control: FEV1, and FVC predicted exposed was six and four percentage points lower respectively. However single-pollutant regression models showed a non-statistically significant reduction in FEV1 and FVC over time.[22]

Discussion

This review shows that most studies demonstrated association between exposure to cement dust and poor or impaired lung health. However, the evidence is not strong as the measures of effects were weak in some cases and there were contradictory findings for some symptoms and pulmonary function indices, (FEV1, FVC, FEV1/FVC ratio, PEFR) across studies. Additionally, the majority of the studies were cross sectional and half of the reviewed studies did not perform robust measurement of the exposure nor did they control for other possible sources of PM in the ambient. Due to the heterogeneity of studies; the population studied and variance in results, this review will focus on discussing and placing emphasis methods applied in the studies

The contradictory findings could be attributed to several factors; not least the high variation in sample size, non-uniformity of characteristics of the exposed groups across the studies, and inconsistent methods of measuring the exposure and outcome. For example, comparing 'cough' as health outcome associated with inhalation of airborne dust, Legator et al[13], using a sample size of 58 and 54 in the exposed and control communities respectively, found a higher proportion of participants in the exposed group compared to the control, reporting cough yet the difference was not statistically significant. On the other hand, Nkhama et al.[14] who used a larger sample size of 220 in each of the communities was able to find a statistically significant difference in proportions reporting cough; OR 5.64 (95% CI 3.63-8.67; p value < 0.001 and OR 1.02 (95% CI 1.01-1.04) in a cross sectional and panel study respectively. This could arise from (a) lack of power to detect a difference in the former study compared to the latter, (b) the method of measuring "cough" or (c) the study settings.

The assessment of association between any exposure and outcomes requires precise and accurate measurements of both. Employing different methods for measuring the exposure or outcomes across studies compromised precision thus making it difficult to compare studies. For instance, one study[14] measured cough as "morning cough" and "night cough" which was then analysed as "composite cough" by combining any of the types while Legator et al.[13] reported "persistent cough".

The methods used to measure the exposure were varied across studies and therefore incomparable. Some studies measured exposure to pollutant levels directly while others used indirect measured. Studies either assumed exposure levels based on previous studies or used an environmental monitoring approach as a proxy measure of individual participant exposure. Even with several studies[17,20,22–24] showing that the ambient air in communities near cement factories has levels as high as high as 45.5μg/m^3, 68.28 μg/m^3, 340.1μg/m^3 ', 387.20 μg/m^3 and 8805μg/m^3, of cement dust and other emissions, relying on retrospective data or extrapolating data from other situations is not a reliable method in assessing relationships. This is because a number of factors, such as temperature, humidity and wind speed,[29 28] can affect the instantaneous PM burden in the ambient air. Environmental conditions thus could result in wide variability in exposure levels in exposed communities and as such assumptions cannot be made about exposure levels especially in studies investigating associations. Environmental monitoring of the ambient air, though better than the first approach, is also below the minimum requirement as it does not measure the actual individual exposure. There are a number of limitations with this approach especially that it does not account for variation in the micro-environment around the individual participant and activity of the participant. For instance, participants may move out of the study for prolonged period of time in a day meaning that the individual stops being exposed for that period of time. This leads to erroneous conclusions about the exposure level for the individual. In additional to inaccuracies of measurements of the exposure, other sources of pollution were not accounted for. Furthermore, there were notable differences in the actual measurements; some studies conducted 24-hour continuous monitoring while the other conducted 8-hour monitoring. The 8-hour[18] monitoring approach is compromised in that not only does it fail to measure individual exposure; the method also fails to reflect daily total exposures for the exposed community. The studies that did not measure exposure but assumed exposure levels based on findings from other studies in different communities could have been incorrect in concluding "effect'.[13,18–20,25] These studies arguably did not help in resolving the question of whether or not exposure to cement dust is associated with respiratory ill health. Additionally, chemical characterization was omitted in most studies in this review. Chemical characterization is essential in establishing the source of the exposure. Although, without knowledge of chemical composition of samples and resultant source apportionment, it is difficult to confidently associate the observed respiratory ill health to exposure to cement dust, the studies in this review are useful as base studies for future and for rigorous studies.

All studies measured self-reported symptoms that were not verified with hospital records. It is possible that self-reporting could have introduced reporting (recall) bias especially because that there had been much media publicity about the adverse effects that cement plant has had on the environment and people living in the vicinity of the plant.[13,14] Except for Legator at el.[13] none of the studies blinded the respondent in the exposed communities. Blinding of respondents is a useful tool in addressing such bias. However, in assessing community environmental exposures and measurement of effects it is difficult to implement this approach.[30]

Findings from this review suggest that exposure to cement dust has variable effects on the respiratory tract. Whereas cough seemed to be consistently related to the exposure, in almost all the studies regardless of study design, other symptoms showed variable relationships. For instance, wheeze, shortness of breath and phlegm was not significantly associated to the exposure in some studies. This implies that studies must strive to measure and report each respiratory symptom separately to reflect the fact that the respiratory effects are not uniform. It is therefore imperative that studies must go further than measuring total dust concentrations but they must also chemically characterise the individual constituents of the emissions. In addition to particulate matter, other emissions such as sulphur dioxide (SO2), nitrogen oxides (NOx) and carbon monoxide CO have been shown to result in cough, wheeze and phlegm.[31]

Limitation

Important limitations were observed for the studies included in this review. Most important is the study design used in investigating this question. Cross sectional studies are ill equipped to answer association questions but were the majority. None of the studies reviewed performed chemical characterisation of the exposure, neither was source apportionment done. In the absence of the former it is difficult to associate the exposure with health outcome while with the latter even if there were observed increases in the exposure within the exposed communities, it is difficult to account for the amount of particulate matter "cement dust" from the cement factories. There was generally incomplete information in the articles leading to some studies being excluded. One study,[16] although included in this review, did not state the sample size and population characteristics of the control community, an essential component of comparative studies. Similarly, Priyanka et al.[24] and Tawiri et al.[23] did not give detailed descriptions of the study participants. The researchers attempted to contact some authors of the studies but received no response by the time this systemic review was completed.

Limitations related to this systematic review included insufficient literature on studies that have assessed the effect between cement dust exposure and respiratory health. Moreover, the studies were varied in terms of sample size, measurement of exposure, specific respiratory symptoms measured and statistical methods making it impossible to conduct a conduct meta-analysis.

Conclusion

Despite studies included in this review showing some degree of association between exposure to cement dust and respiratory ill health, the existing evidence is insufficient to draw firm conclusion. Most studies used a cross sectional design which has an inherent weakness in providing evidence of causation or associations. Other weaknesses included suboptimal measurement of exposure and outcomes. To improve the quality of evidence of association between exposure to cement dust and respiratory ill health, it is recommended that future studies should employ methods that increase accuracy in measuring the exposure and outcomes. These should include personal monitoring of the exposure, source apportionment carried out and chemical characterisation coupled with using standardized measurement tools for exposure and outcome at predetermined intervals. Highlighted in this review is that even without strong evidence as a result of methodological weaknesses, there are sufficient indicators that the quality of air in communities around the cement plants is poor and that the burden of respiratory symptoms and diseases is much higher compared to other communities. It is thus recommended that cement factories should institute measures to reduce emission into the ambient so as to improve the quality of air for communities residing near the plants. Results from this review, could help to improve the study designs for future research, and inform public health policy even in the midst of the current uncertainty on the relationship of exposure to cement dust and respiratory health.

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Responses for the Review

Line number	Reviewer's comment	Author's response
59	What type of impairment? How was it assessed?	Some epidemiological studies have reported reduced pulmonary function indices, measured as forced expiratory volume in one second (FEV1), forced vital capacity (FVC), ratio of forced ventilatory capacity in one second and forced vital capacity (FEV1/FVC), and peak expiratory flow rate (PEFR) in individuals who are exposed to cement dust in communities residing near cement factories. However, other studies have reported no statistically significant relationship between exposure to cement dust and pulmonary function impairment. Smoking is known to be a confounder for lung health, however not all studies control for it. In one study where smoking was considered, it was found that, smokers exposed to occupational cement dust had significantly lower values for FEV1, FEV1/VC, FEV1/FVC and PEFR compared with the unexposed smokers. Conversely, there was no statistical difference in the pulmonary function indices between smokers and non-smokers in both the exposed and unexposed groups. In addition to pulmonary function indices, non-specific respiratory symptoms including cough, wheeze, dyspnoea, sneezing, and phlegm are frequently reported in exposure to cement dust studies
61-62	These are unspecific symptoms	
64-65	Is there an statistical association? Considering that smoking is a very common habit, how can changes in the lung function differentiated between tobacco use and cement dust?	
66	What kind?	
67-68	In the general population or among those working in cement factories?	
350	Individual pathologies should be detailed	The following statement has been included; This review shows that most studies demonstrated association between exposure to cement dust and poor or impaired lung health. However, the evidence is not strong as the measures of effects were weak in some cases and there were contradictory findings for some symptoms and pulmonary function indices, (FEV1, FVC, FEV1/FVC ratio, PEFR) across studies.

Line number	Reviewer's comment	Author's response
352	Which ones?	
358	Which where those used more frequently?	The subsequent statement of the paragraph explain in detail the frequent methods used (372)
360	Cough is a very common and unspecific symptom, not a disease, thus assessment of symptoms rather than pathologies should be disregarded from the final analysis. In this case it would be interesting to analyse why the author choose cough as an indicator.	The studies defined the symptoms (e.g cough) according to the validated tools such American Thoracic Society (ATS), European Respiratory Society (ERS) or the British Thoracic Society (BTS). • Cough was defined or explained according to the standard. • This has also been stated in the methodology section (health outcome assessment)
363	Before continuing with the discussion, it's important to state what was considered as cough in each study. These criteria might lead to different result	Four studies assessed cough. Of these, three studies used the ATS and measured cough as; • Cough was considered as "morning cough" and "night cough" which was then analysed as "composite cough" by combining any of the types While one study defined cough as; • Persistent cough
365	This needs to be developed deeper	This has been revised and deleted
369-371	Each type should be defined in both, the introduction and the discussion	As advised, the following statement has been added both to the introduction and discussion sections; "Legator measured 'persistent cough' while others defined cough as 'morning cough' and 'night cough' and combined to 'composite cough"
372	It's important to specify which method was used	Studies either assumed exposure levels based on previous studies or used an environmental monitoring approach as a proxy measure of individual participant exposure. Even with several studies[16,20,22-24] showing that the ambient air in communities near cement factories has levels as high as high as 45.5µg/m^3, 68.28 µg/m^3, 340.1µg/m^3

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		', 387.20 μg/m^3 and 8805jhhμg/m^{3,} , of cement dust and other emissions, relying on retrospective data or extrapolating data from other situations is not a reliable method in assessing relationships. This is because a number of factors, such as temperature, humidity and wind speed,[28] can affect the instantaneous PM burden in the ambient air. Environmental conditions thus could result in wide variability in exposure levels in exposed communities and as such assumptions cannot be made about exposure levels especially in studies investigating associations. Environmental monitoring of the ambient air, though better than the first approach, is also below the minimum requirement as it does not measure the actual individual exposure. There are a number of limitations with this approach especially that it does not account for variation in the micro-environment around the individual participant and activity of the participant.
376	Measurable levels are required	Revised and added OR 5.64 (95% CI 3.63-8.67; p value < 0.001 and OR 1.02 (95% CI 1.01-1.04) in a cross sectional and panel study respectively
388	Multivariate analysis is required. Was cement factory the only air pollution in the area? Usually where a cement factory is located there are several other industries… What about motor vehicles?	Most of the studies did not control for other sources of pollutants. Therefore, the following statement has been added; "In additional to inaccuracies of measurements of the exposure, other sources of pollution were not accounted for"
394-395	What kind of statistical analysis was used across the different investigations to set a causal association?	Odds ratios were the major measures of association used. To control for confounding linear and logistic as well generalised estimating equations were used.

Line number	Reviewer's comment	Author's response
399	So, is the available information useless?	This statement has been revised the statement and reads as follow: • "Although, without knowledge of chemical composition of samples and resultant source apportionment, it is difficult to confidently associate the observed respiratory ill health to exposure to cement dust, the studies in this review are useful as base studies for future and for rigorous studies." Additionally: Results from this review, could help to improve the study designs for future research, and inform public health policy even in the midst of the current uncertainty on the relationship of exposure to cement dust and respiratory health.
404	This is speculative	Thank you for pointing out this one, we have deleted.
409	Not really. According to the information provided above, there's not strong data to support any conclusion	The paragraph has been revised and this statement has been deleted.
411-412	Not a single value of association stats was shown in the discussion	Well noted. Values of association have been added in second and third paragraphs of the discussion section.
414-415	From where can this be concluded?	Most studies used cross sectional study design and demonstrated association. Additionally, results from the panel study reported similar results. The panel involve repeated measurements of both the exposure and the outcomes on the same individual, providing even more accurate estimate in the variation of respiratory effects due to the exposure.

Line number	Reviewer's comment	Author's response
416	Are differences between cement dust among factories? If it's a standardized process, it might be assumed that pollutants will be the same anywhere. This needs to be discussed deeper in both, the introduction and discussion	The process of cement manufacturing is similar. The following statement has been added to the introduction: The production of cement involves the following three steps: i. Preparation of raw materials (limestone, shale and sand) which involves mixing/homogenising, grinding and preheating (drying) to produces the raw mill. ii. Burning of raw mill at high temperatures (900-1500 °C) in the pre-calciner to form cement clinker in the kiln. iii. After cooling, the clinker is ground together with the additive. The finished material is then stored in silos, ready for dispatch in bags or bulk. Several emissions with capacity to pollute the ambient air are released during these production phases.
416-418	What about particulate pollution?	In addition to particulate matter, other emissions such as sulphur dioxide (SO2), nitrogen oxides (NOx) and carbon monoxide CO have been shown to result in cough, wheeze and phlegm.