

GRAIN DUST AS A CAUSE OF OCCUPATIONAL LUNG DISEASE

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Abstract

Grain dust is a complex material with a long history of causing respiratory diseases ranging from bronchitis and chronic obstructive pulmonary disease through immediate allergy, asthma and allergic alveolitis to organic dust toxic syndromes. The roles of fungal and actinomycete spores, bacteria, endotoxins and mycotoxins, insects and mites and their products and of other materials differs between the different syndromes although with some the etiology is not clear. Exposure and patient factors also differ so that with asthma, predisposition to sensitisation by normal, everyday exposure to allergens is important while with allergic alveolitis and organic dust toxic syndrome, intense exposure is of overriding importance. The different conditions are compared and contrasted and the roles of different components of the dust in their etiology is discussed.

Introduction

The harmful effects of grain dust have long been known. In 1713, Ramazzini, in his classic treatise on *Diseases of Workers* referred to the complaint of those who sifted and measured wheat and barley grain that they were "so plagued by this kind of dust that when the work is finished they heap a thousand curses on their calling". They suffered from cough, inflammation of the eyes and shortness of breath and rarely reached old age. He associated these symptoms with the fine dust from grain that had heated and "crumbled to dust". This description probably includes asthma, also noted by Ramazzini in millers and bakers, and extrinsic allergic alveolitis. However, relatively little further attention was paid to the health effects of grain dust until quite recently. Since 1713, the roles of fungal and actinomycete spores in asthma and allergic alveolitis have been recognised, as has the allergenicity of storage mites, and grain dust has come to be recognised as more than just a nuisance. However, although causes have been found for some symptoms, the causes of others are still not proven.

The nature of grain dust

Grain dust is a complex material and its composition changes with stage of harvest, drying and storage, particularly with the way in which the grain has been stored. It may consist of starch grains and fragments of awns and testa from the seed; soil; spores of fungi and actinomycetes, bacteria and metabolites from all

these microorganisms; whole bodies, fragments and excreta from insects and mites; excreta from rodents and birds; and residues from pesticides. The term may be used both for dust produced at harvest and during handling and drying before the grain is placed in store and for that produced after storage. Both contain allergenic components and materials that have other effects on health but they differ microbiologically.

The microflora of grain dust produced at harvest and during handling and drying prior to storage differs greatly from that produced by stored grain, just as the microflora of the grain differs. Grain produced at harvest consists mainly of bacteria and the spores from fungi that colonise ears and straw during grain development and ripening. The fungi include species of *Cladosporium* (50-75% of the total airborne spores), *Alternaria* (about 25%), *Verticillium* (up to 10%), *Epicoccum*, *Puccinia* and *Ustilago* (all < 4%) (Lacey, 1980). However, dust from stored grain contains the spores of fungi characteristic of storage, especially *Aspergillus* and *Penicillium* spp. but the species differ according to the type of grain and grain store, storage conditions particularly water content and temperature, whether spontaneous heating or arthropod infestation have occurred and on the way in which the grain has been handled and cleaned. The predominant species are often indicative of the conditions in which the grain has been stored and if spontaneous heating to 50-65°C has occurred these may include *Aspergillus fumigatus*, a potential human pathogen, and the thermophilic actinomycetes implicated in allergic alveolitis (Lacey, 1980). Settled dust from Canadian grain stores yielded *Eurotium* from 81% (with 53% containing > 2000 cfu g⁻¹) and *Penicillium* (mostly *P. aurantiogriseum* (*P. cyclopium*)) from 100% (95% > 2000 cfu g⁻¹). Even *A. fumigatus* was present in 94% of samples, with 34% containing > 2000 cfu g⁻¹ (Lacey, 1980). Mycotoxins, the toxic secondary metabolites of fungi, have also been found in grain dust, including aflatoxins, secalonic acid, zearalenone and, perhaps, other unidentified mycotoxins (Whidden et al., 1980; Ehrlich et al., 1982; Burg and Shotwell, 1984; Palmgren, 1985).

Bacteria may also be important components of grain dust, both before and after harvest. In Polish grain stores, they formed 75% of the total viable microflora and most were Gram-negative rods, mainly *Pantoea* (*Enterobacter*) *agglomerans* (also referred to as *Erwinia herbicola*), which are important as sources of endotoxin, lipopolysaccharide components of the cell walls (Dutkiewicz, 1978a). Of the species of Gram-negative bacteria isolated from terminal elevators on the lower Mississippi, *P. agglomerans* was predominant in settled dust in the warmer months and *Pseudomonas* and *Klebsiella* in winter while numbers of total bacteria remained constant throughout the year (DeLucca and Palmgren, 1987).

Although infestation of grain by insects and mites can occur without fungi, there is often a close interrelationship with moulding. Fungi provide food for both, while the insects and mites may improve the environment for fungal colonisation by causing heating through respiration, often in grain that is initially too dry for fungal growth, thereby releasing water, and by providing sites for fungal colonisation through their feeding damage.

Exposure to grain dust

Exposure of workers to grain dust depends on many factors and may differ greatly between stores. The dustiness of the grain, the way in which it is handled and the use of dust control equipment and ventilation all affect exposure. Dust concentrations between 0.18 and 781 mg m⁻³ have been found in Canadian grain elevators, with mean concentrations in different areas over a work shift up to 109 mg m⁻³. However, because workers were not continuously exposed in the worst areas, their mean exposure did not exceed 67 mg m⁻³ (Farant and Moore, 1978). In another

study (Cotton et al., 1989), mean concentrations in the general work area of elevators with dust control equipment were $6.1 \pm 9.2 \text{ mg m}^{-3}$ and in those without, $13.6 \pm 22.1 \text{ mg m}^{-3}$ with, respectively, 1.9 ± 3.2 or $6.2 \pm 14.3 \text{ mg m}^{-3}$ in the breathing zones of workers, averaged over an 8 h shift.

Much of the dustiness of grain results from the presence of fungal spores. Concentrations of airborne fungal spores in the Canadian elevators often exceeded $10^6 \text{ spores m}^{-3}$ air and numbered up to $3.6 \times 10^6 \text{ spores m}^{-3}$ in eight elevators in the Duluth-Superior area of the U.S.A. (Whidden et al., 1980). Fungi identified included both field and storage species of which, in the American study, 2.5×10^5 were *Ustilago* spores. Plating yielded up to 9.4×10^4 colony forming units (cfu) of viable fungi m^{-3} air, out of up to 3.4×10^5 cfu total microorganisms m^{-3} (including bacteria and yeasts). Elevators could be ranked by mean spore concentration but there were few differences between job categories. By comparison, some English combine harvester drivers were exposed to 15 to 35×10^6 spores m^{-3} while concentrations of up to 10^{10} spores m^{-3} air were found in moist barley silos during unloading (Lacey, 1980).

The most studied airborne mycotoxins are aflatoxins produced by *Aspergillus flavus*. Aflatoxins have been detected in the air while harvesting maize and handling it in store. Generally, workers were exposed to up to 1600 ng aflatoxin m^{-3} around combine harvesters, 24 ng m^{-3} inside the driving cab, 1120 ng m^{-3} in elevators, 8330 ng m^{-3} loading trucks and railroad cars and 1760 ng m^{-3} when feeding cattle, with the greatest exposure, 13000 ng aflatoxin m^{-3} air, near a conveyor (Burg and Shotwell, 1984). In settled dust, most aflatoxin appears to be in particles of 7-11 μm aerodynamic size (Sorensen et al., 1981).

Health effects of grain dust

Grain dust can cause irritation of the nose, throat, skin and eyes as well as a range of acute and chronic respiratory symptoms, including allergy, asthma, cough, grain fever, bronchitis and chronic obstructive pulmonary disease. The incidence and relative importance of the various forms of disease is uncertain. Epidemiological studies are few and mostly limited to Canada and the U.S.A. (Becklake, 1980). In Manitoba, symptoms reported by grain elevator agents included chronic cough, sputum, dyspnoea and grain fever, with both immediate hypersensitivity, demonstrated by prick tests, and positive precipitin tests (Tse et al., 1973). A similar range of symptoms was found in a group of English farmers, of which 20% were affected by harvesting dust (Lacey, 1980). However, in neither study was there any significant correlation between positive skin or precipitin tests and clinical disease, although a later study of grain farmers and elevator agents showed a good correlation between skin and bronchial provocation tests (Warren et al., 1974). The effects of grain dust cannot easily be separated from other factors, especially smoking, and the role of microorganisms is sometimes poorly understood. It is estimated that some 75% of Canadian grain elevator agents complain of symptoms on exposure to dust and 27% of systemic symptoms (Tse et al., 1973) and that there is an excess 5-15% in the occurrence of respiratory symptoms in non-smoking grain handlers (Broder, 1989); more symptoms were reported where workers completed questionnaires themselves (DoPico et al., 1984).

The various pulmonary effects of grain dust contrast in a number of important ways (Table I). Acute inflammation, bronchitis and chronic obstructive pulmonary disease are commonly associated with smoking but may also occur widely in non-smokers in the grain industry with a total incidence exceeding that in the general population (Dosman and Cotton, 1980; Warren and Manfreda, 1980; Broder, 1989). There is wheeze, breathlessness, cough and sputum with airflow obstruction in

Table I. Comparisons of the characteristics of respiratory disorders associated with grain dust

Factor	Symptom	Bronchitis, chronic obstructive pulmonary disease	Asthma	Allergic alveolitis	Organic dust toxic syndrome
<i>Exposure</i>		Dusty but not otherwise defined	Normal, everyday, up to 10^4 pollens, ⁻³ 10^6 fungal spores m^{-3}	Intense, $> 10^6$ and up to 10^{10} spores m^{-3}	Intense but not otherwise defined
<i>Particle size</i>		Not known	Mostly $> 5 \mu m$	Mostly $< 5 \mu m$	Not known
<i>Predisposition</i>		Some evidence that atopy increases incidence	Important; affects readily sensitised atopic subjects	Unimportant, affects mainly non-atopic subjects	Unimportant, may affect several workers in team
<i>Site affected</i>		Bronchi	Bronchi	Alveoli	None characteristic, alveolitis sometimes found
<i>Onset</i>		Gradual development of chronic symptoms	Rapid, within minutes	Delayed, after several hours	Delayed, after several hours
<i>Systemic upset</i>		Not characteristic	Slight	Influenza-like, fever, cough, weight loss, breathlessness	Fever, malaise
<i>X-ray changes</i>		Not characteristic	Not characteristic	Mottling	None characteristic
<i>Serology</i>		None characteristic	No precipitins, IgE	Precipitins, IgG	None characteristic
<i>Skin test reaction</i>		None	Immediate, wheal and flare reaction	Delayed or none, extensive, oedematous	None characteristic
<i>Functional abnormality</i>		Obstructive	Obstructive	Restrictive + diffusion effect	None characteristic
<i>Possible causes in grain dust</i>		Endotoxin or other components	Large fungal spores, e.g., <i>Alternaria</i> , <i>Puccinia</i> , <i>Cladosporium</i> , mites,	Small spores of actinomycetes, <i>Aspergillus</i> , <i>Penicillium</i> spp.	Bacteria, spores of actinomycetes or fungi, mycotoxins, bacterial endotoxins

smokers and non-smokers alike. Forced expiratory volume (FEV₁) may decrease significantly through the season (James *et al.*, 1986) and a decrease in forced expiratory flow (FEF₅₀) related to exposure to respirable grain dust was seen in 74% of a group of Canadian grain elevator workers (Broder, 1989). In Manitoba grain elevator agents, >25% of non-smokers developed chronic cough, dyspnoea and sputum, compared to >50% of smokers and ex-smokers (Tse *et al.*, 1973). Symptoms may be related to a history of wheeze, asthma and hay fever, increased bronchial reactivity and positive responses in skin prick tests to a range of allergens (James *et al.*, 1986) and may or may not be related to atopy (constitutional predisposition to sensitization by normal, everyday exposure to allergens) (Terho *et al.*, 1987; Broder, 1989). In a group of Finnish grain farmers, the effects of atopy and smoking were additive, each doubling the risk of bronchitis. The etiology of the symptoms is uncertain. They may be non-specific reactions to components of the dust or be triggered by endotoxin inhalation or even allergy.

Allergic rhinitis and asthma occur in about 30% of people who are described as atopic, developing symptoms of rhinitis or asthma within minutes of exposure to airborne allergens. Concentrations of fungal spores may reach 10^6 m^{-3} out of doors but usually do not exceed 10^4 - 10^5 spores m^{-3} , even in summer. The incidence of atopy and allergy among workers newly employed in the grain industry is probably similar to that in the general population. However, the low incidence of asthma among some groups of grain workers suggests that those who develop sensitivity to grain dust soon leave the industry (DoPico *et al.*, 1986). Grain workers are exposed to many allergens and their exposure, both when harvesting grain and later when handling stored grain, may be more intense than that experienced by the general population although the species to which they are exposed may also differ. Storage mite and grain weevils can both be important allergens in grain stores (Frankland and Lunn, 1965; van Hage-Hamsten *et al.*, 1988). Among Danish grain elevator workers, 6.4% had storage mite allergy and 15.9% were sensitised to mites, with *Acarus siro*, *Lepidoglyphus destructor* and *Tyrophagus longior* present in 75% of grain and all dust samples. (Revbech and Andersen, 1987). On English farms, mite specific IgE was present in 59% of farmers with work-related respiratory symptoms and in 60% with cough but in only 9% of symptomless farm workers (Blainey *et al.*, 1988). Also, 33% of workers in a modern bakery were sensitive to one or more storage mite allergens (Musk *et al.*, 1989). Immediate hypersensitivity to non-mite allergens can also sometimes be common. Of 17 workers in one study, most reacted to crude grain dust (8) and smaller numbers to grain mill dust (4), *Ustilago* (3), *Cladosporium* (1), *Helminthosporium* (1), *Candida* (4) and *Aspergillus* spp. (1) (Warren *et al.*, 1974). Positive skin tests to bacterial extracts may sometimes be found (Dutkiewicz, 1978 b).

Extrinsic allergic alveolitis (hypersensitivity pneumonitis) is a T-lymphocyte dependent granulomatous inflammatory reaction, chiefly of the peripheral gas-exchange tissue, and is more dependent on intense exposure to airborne antigen than to constitutional predisposition (Newman Taylor, 1987). The presence of precipitating IgG antibodies (precipitins) to the offending antigen can aid diagnosis but they are chiefly evidence of exposure. Their relevance to the disease has to be determined by other tests. Alveolitis may be caused by spores of fungi or actinomycetes in concentrations exceeding 10^6 to 10^9 or more spores m^{-3} . A minimum of 10^8 spores m^{-3} may be necessary to cause acute symptoms (Rylander, 1986). Acute dyspnoea, sometimes of delayed onset, was found in 44% of Manitoba farmers, mostly when handling old grain (Warren and Manfreda, 1980). Precipitins against crude grain dust extract were detected in two out of 17 farmers, against *Faenia rectivirgula* in another two and against both antigens in one (Warren *et al.*, 1974). Allergic alveolitis in grain farmers, caused by *P. agglomerans* with precipitins to both bacteria and lipopolysaccharide (Dutkiewicz

et al., 1978b, 1985), and in malt workers to *Aspergillus clavatus*, are recorded.

Organic dust toxic syndrome includes pulmonary mycotoxicosis, silo unloader's disease and, perhaps, grain fever and precipitin-negative farmer's lung. Its etiology is unknown but it is associated with the inhalation of large amounts of airborne dust. Symptoms resemble allergic alveolitis in being influenza-like with leucocytosis but prior sensitisation, respiratory symptoms, radiographic changes or precipitins do not necessarily occur. More than one worker in a group often develops symptoms. Organic dust toxic syndrome was more common than all other farm-related respiratory illnesses in New York and more than one worker was affected in 75% of outbreaks. Farm workers affected were younger than in allergic alveolitis (May et al., 1986). Suggested causes include fungal and actinomycete spores, bacteria, mycotoxins and endotoxins but although these have been abundant in source material, the aerobiology of the environment at the time of exposure, nor of the occurrence of airborne endotoxins or mycotoxins, has rarely been studied (Emanuel et al., 1975; Rylander and Snella, 1982; Pratt and May, 1984; May et al., 1986; Dutkiewicz et al., 1989).

Mycotoxins may be inhaled with fungal spores and cause lesions in the lung, which is perhaps more susceptible to damage than is suggested by LD₅₀ values for the toxin, or other organs after absorption. The risk of primary liver cancer was significantly increased in Swedish grain millers, although the overall cancer incidence was not elevated, perhaps because of exposure to aflatoxins or pesticides (Alavanja et al., 1987). Other occurrences of cancer in which aflatoxins might be implicated are documented by Abramson, 1989. Apart from acute effects, mycotoxins may decrease the body's defences against other infections, through immune suppression or cytotoxic effects on alveolar macrophages (Gerberick and Sorenson, 1983; Gerberick et al., 1984; Pier and McLoughlin, 1985).

Endotoxins are lipopolysaccharide components of the cell walls of Gram-negative bacteria that can produce febrile reactions on inhalation, complement activation, macrophage activation, mitogenicity and death (Salkinoja-Salonen et al., 1982). Inhalation of endotoxin by rabbits resulted in leucopenia followed by leucocytosis after several hours; fever occurred after a latent period of 1-2 hours but daily exposure led to tolerance. Antibodies against endotoxin could be detected after 5 days and there was an acute interstitial pneumonitis (Snell, 1966). Clinical effects are likely with more than 10⁵ Gram-negative bacteria or 0.1 µg endotoxin m⁻³ air (Rylander et al., 1983). These concentrations may be found in grain stores without undue moulding. Up to 1.3 x 10⁶ cfu m³ microorganisms were found in the air of Polish grain stores, with *P. agglomerans* (up to 4.3 x 10⁵ cfu m³) predominant (Dutkiewicz, 1978 a). However, respirable dust in two grain terminals on the lower Mississippi contained only 0-7.9 ng endotoxin m⁻³ and no viable bacteria. Settled dust contained 1.7-5.6 ng endotoxin g⁻¹, with most in September-November and least in January-March (DeLucca and Palmgren, 1987).

Opportunistic pathogenic fungi, e.g., *A. fumigatus*, can occur widely in grain but are usually only numerous when heating to 40-55°C occurs. They can cause pulmonary infections but usually only in immune-compromised subjects, i.e., those suffering from diseases affecting the immune system, being treated with immunosuppressive drugs or receiving radiation therapy. *A. fumigatus* may also grow in tuberculosis cavities, forming an aspergilloma or fungus ball.

Conclusion

Respiratory disorders associated with grain dust have long been known but there is still much uncertainty about the causes of some syndromes and of their

relative importance. Different disorders have frequently been confused and new studies require careful clinical descriptions and an integrated interdisciplinary study of all possible causes. Synergistic interactions between different components of grain dust need to be considered to determine their relevance.

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LA POUSSIERE DES GRAINS EN TANT QUE CAUSE DE MALADIE PULMONAIRE PROFESSIONNELLE

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RESUME

La poussière des grains est composée de fragments de végétaux, de paille, de terre, de bactéries, d'actinomycètes de spores de champignons, d'insectes, d'acariens et de leurs produits, endotoxines et mycotoxines. Plusieurs de ces substances peuvent contribuer au développement de maladies pulmonaires chez les travailleurs agricoles et, parfois, dans la population en général. Les maladies peuvent comprendre la bronchite chronique, l'allergie de contact immédiate, allant de la rhinite et de ses symptômes "rhume des foins" à de l'asthme, l'alvéolite allergique, le syndrome de toxicité des poussières organiques, l'infection, la fièvre due aux endotoxines et, peut-être, les mycotoxicoses pulmonaires. Avant la moisson, les champignons pathogènes poussant sur le végétal, ainsi que les moisissures saprophytes qui poussent sur la paille et les épis mûrissants, contribuent grandement à l'ensemencement de l'air en spores, cause d'asthme dans la population en général autant que chez les travailleurs agricoles. Après stockage, la composition de la poussière dépend des conditions de stockage et du degré de colonisation du grain par les micro-organismes, les insectes et les acariens. Plusieurs champignons ainsi que, tout spécialement, les acariens, sont des allergènes bien connus tandis qu'une grande concentration de spores et d'actinomycètes en suspension peuvent être la cause d'alvéolite allergique. Une trop longue exposition aux micro-organismes en suspension dans l'air s'avère également importante dans le développement du syndrome de toxicité des poussières organiques, mais les agents de l'étiologie et les mécanismes de la maladie n'ont, jusqu'à présent, pas encore été identifiés, bien qu'il ait été suggéré que les spores de champignons, les endotoxines bactériennes et les mycotoxines puissent être les causes possibles. La relation entre les micro-organismes en suspension dans l'air et la bronchite n'est pas claire, bien que l'allergie semble contribuer à la maladie et qu'elle apparaisse plus communément chez les travailleurs qui manipulent le grain, plutôt que chez les autres travailleurs agricoles.