

Noise – Protect your Hearing

Noise related damage is a big problem in the arboriculture industry.

- Hearing loss is progressive and permanent.
- It is also totally preventable.
- Hearing loss builds up from exposure to all sorts of loud noise.

How long does it take for particular sounds to become dangerous?

NOISE SOURCE	NOISE LEVEL IN DECIBEL <i>Decibel – used to measure sound level</i>	TIME TO DAMAGE HEARING WITHOUT PROTECTION
Gunshot	120	Instantaneous
Wood chipper	100 – 110	1 to 15 minutes
Chainsaw	100 - 110	1 to 15 minutes
Disk cutter	100 – 110	1 to 15 minutes
Powertools	100	1 to 15 minutes
Waratah	96	30 minutes
Loader	96	30 minutes
Heavy truck	90	1 hour
Lawnmower	90	1 hour
Excavator	87	4 hours

Without proper hearing protection, running a chain saw for less than 5 minutes can cause hearing loss!

What is hearing damage?

Loud noise damages the tiny hairs in your inner ear.

If the hairs are damaged they cannot carry sound to the brain, so you can't hear.

These hairs do not grow back. Once they are gone, so is your hearing!



How can you protect your hearing?



Turn it off



Walk Away



Protect your ears



Limit Exposure

Limit the noise at work as much as you can – this is your first line of defence.

- Turn off equipment and machinery when you're not using it.
- Stay away from someone doing a noisy task if you can.
- If you're in a machine cab, keep the doors and windows closed and use air conditioning.
- Have your hearing checked regularly.
- Don't think a few minutes of noise doesn't matter – in the long term it does.

Use the right PPE – that's your last line of defence.

- Always wear the right hearing protection whenever you're doing a noisy job or around someone else doing one.
- Wear your hearing protection the right way, making sure that nothing is breaking the seal. Don't wear it over hats, hoodies or sunglasses.
- Make sure your earmuffs are in good condition, with no creases or rips in the pads.
- Don't listen to loud music while you work – this defeats the purpose of protecting your ears from noisy equipment.

Limit noise away from work as much as you can.

- Turn down the music when driving, especially to and from work.
 - Wearing hearing protection if you are using power tools or lawnmowers at home, or you're going shooting or hunting.
- ✓ Check your hearing protection daily. If it becomes damaged, replace it immediately.
 - ✓ If you have any concerns about your hearing, PPE or noise exposure, contact your manager.

Respiratory Diseases Associated With Organic Dust Exposure



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Target Audience: Physicians and researchers within the field of allergic disease.

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Learning objectives:

1. Define the important components representing organic dust exposure and mechanisms mediating airway disease consequences.
2. Recognize the spectrum of airway disease and the at-risk populations associated with various organic dust exposures.
3. Describe preventative and therapeutic approaches to mitigate airway disease associated with organic dust exposures.

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Organic dusts are complex bioaerosol mixtures comprised of dust and particulate matter of organic origin. These include components from bacteria, fungi, pollen, and viruses to fragments of animals and plants commonplace to several environmental/occupational settings encompassing agriculture/farming, grain processing, waste/recycling, textile, cotton, woodworking, bird breeding, and more. Organic dust exposures are linked to development of chronic bronchitis, chronic

obstructive pulmonary disease, asthma, asthma-like syndrome, byssinosis, hypersensitivity pneumonitis, and idiopathic pulmonary fibrosis. Risk factors of disease development include cumulative dust exposure, smoking, atopy, timing/duration, and nutritional factors. The immunopathogenesis predominantly involves Toll-like receptor signaling cascade, T-helper 1/T-helper 17 lymphocyte responses, neutrophil influx, and potentiation of manifestations associated with allergy. The true

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Abbreviations used

ACO- Asthma-COPD overlap
AHR- Airway hyperresponsiveness
BALF- Bronchoalveolar lavage fluid
COPD- Chronic obstructive pulmonary disease
FeNO- Fractional exhaled nitric oxide
FEV₁- Forced expiratory volume in 1 second
FVC- Forced volume capacity
HP- Hypersensitivity pneumonitis
Ig- Immunoglobulin
IL- Interleukin
IPF- Idiopathic pulmonary fibrosis
LPS- Lipopolysaccharide
MyD88- Myeloid differentiation factor 88
OA- Occupational asthma
ODTS- Organic dust toxic syndrome
Th- T-helper
TLR- Toll-like receptor
TNF- Tumor necrosis factor
WRA- Work-related asthma

prevalence of airway disease directly attributed to organic dust, especially in a workplace setting, remains challenging. Diagnostic confirmation can be difficult and complicated by hesitancy from workers to seek medical care, driven by fears of potential labor-related consequence. Clinical respiratory and systemic presentations coupled with allergy testing, lung function patterns of obstructive versus restrictive disease, and radiological characteristics are typically utilized to delineate these various organic dust–associated respiratory diseases. Prevention, risk reduction, and management primarily focus on reducing exposure to the offending dust, managing symptoms, and preventing disease progression. © 2024 American Academy of Allergy, Asthma & Immunology (J Allergy Clin Immunol Pract 2024;12:1960-71)

Key words: Organic dust; Asthma; Obstructive lung disease; Hypersensitivity pneumonitis; Fibrosis; Occupational

INTRODUCTION

Organic dusts refer to a complex mixture of dust and particulate matter of organic origin including components from bacteria, fungi, pollen, and viruses to fragments of animals and plants.¹ Organic dust exposures have been linked to development of chronic occupational airway diseases such as chronic bronchitis, chronic obstructive pulmonary disease (COPD), asthma, asthma-like syndrome, byssinosis, hypersensitivity pneumonitis (HP), and idiopathic pulmonary fibrosis (IPF). Establishing the true prevalence of airway disease directly attributed to organic dust, especially in a workplace setting, remains challenging. The diagnostic confirmation of occupational respiratory diseases can be difficult and complicated by hesitancy from workers to seek medical care, driven by fears of potential labor-related consequences.² Workers with worsening symptoms tend to leave their jobs, and those without immediate symptoms remain in the workplace, which has been referred to as “the healthy worker effect.”³ This review addresses risk factors contributing to disease development, immunopathogenesis, disease entities, diagnostic approaches, and management strategies.

EPIDEMIOLOGY

Industries commonly affected by organic dusts include agriculture and food production with high variability in concentration exposure typically related to tasks performed rather than herd size.³ In the dairy industry, milking and animal-handling activities provided a higher risk of concentration exposure.³ In the avian industry, hatchery workers experience an increased prevalence of respiratory symptoms including cough and/or phlegm.⁴ Furthermore, workers assigned to avian-sorting rooms also had a decrease of 11% in forced vital capacity (FVC) compared with those working in incubation rooms.⁴ Moreover, in a Norwegian study of farmers, livestock farmers had increased risk of chronic bronchitis (adjusted odds ratio [aOR] 1.9; 95% confidence interval [95% CI] 1.4–2.6) and COPD (aOR 1.4; 95% CI 1.1–1.7) compared with crop farmers after adjustment for potential confounders including age, gender, and smoking status.⁵ A meta-analysis by Guillien et al⁶ demonstrated a positive association between farming exposure and airflow limitation or chronic bronchitis in 10 of 22 studies (odds ratio [OR] 1.77; 95% CI 1.50–2.08) with cattle, swine, poultry, and crop farming associated with either airflow limitation or chronic bronchitis.

In the waste and recycling sector, organic dusts and volatile organic compounds were the highest during the sorting of materials in compost stations.⁷ In the textile industry, recognized to have high endotoxin exposure, byssinosis is an occupational respiratory disease due to exposure to cotton, hemp, or flax. Hinson et al⁸ reported a prevalence of byssinosis of 44% among workers directly exposed to cotton dust in a textile company in Benin, West Africa. In the wood-processing sector, Neghab et al⁹ observed that 35% of Iranian sawmill workers experienced a 5% cross-work shift decrease in forced expiratory volume in 1 second (FEV₁). This occurred despite the existence of recommended occupational exposure limits and precautions offered by occupational safety organizations.⁹

IMMUNOPATHOGENESIS

Organic dusts are bioaerosols composed of a diverse and wide array of organic particles, encompassing fragments from both animal and plant sources, alongside pro-inflammatory mediators such as gram-negative endotoxins or lipopolysaccharides (LPS), gram-positive peptidoglycans, and fungal (1–3) β -D-glucans, among others.^{10,11} These exposures induce release of pro-inflammatory cytokines/chemokines that typically drive the recruitment and activation of neutrophils, induce airway hyperresponsiveness (AHR), generate free radicals, and promote lymphocyte activation.^{12,13} With repetitive exposures, airway remodeling and chronic disease develop.¹⁴ Whereas LPS is a notable element within organic dust, it is the “whole” composition (as opposed to 1 isolated agent) of organic dust mixtures that drive pathogenesis. For example, individuals exposed to pig barn dust had a more pronounced inflammatory response as evidenced by elevated levels of interleukin (IL)-6, IL-8, and total cell counts in sputum samples compared with exposure to LPS alone, despite the concentration of LPS being several-fold higher than what is encountered in pig barns.¹⁵

Organic dust engages innate signaling pathways, mainly through the recognition of pathogen-associated molecular pattern molecules typically by Toll-like receptors (TLRs). The TLR2, TLR4, TLR9, and the common adaptor protein myeloid differentiation factor 88 (MyD88) have been implicated in the

acute inflammatory response to agricultural-related organic dust exposures.¹⁶⁻²¹ In murine studies, swine confinement facility organic dust extract-induced acute airway inflammatory responses (ie, neutrophil influx, release of tumor necrosis factor [TNF]- α , IL-6 and neutrophil chemoattractants, and AHR) were nearly completely abrogated in MyD88-deficient mice.¹⁸ Furthermore, striking reduction in airway inflammatory responses remained after daily exposure for 1 week in MyD88-deficient mice,²² but there was enhanced airway mucous cell metaplasia.²³ Correspondingly, acute organic dust exposure in humans is also associated with cellular infiltration and increased cytokine production ascribed to TLR signaling. Namely, Hedelin et al²⁴ demonstrated increases in nasal lavage total and blood neutrophils, monocytes, and basophils following a 3-hour exposure to swine dust with associated increases in monocyte expression of TLR2 and TLR4. This response was reduced following the installment of fine particle separators.²⁴

Highlighting the importance of TLR signaling pathways, a missense mutation in *TLR4* (Asp299Gly) has been associated with hyporesponsiveness to inhaled LPS exposure with decreased nuclear factor kappa B activity, decreased IL-1 α production, and decreased airway epithelial TLR4 receptor.²⁵ Single nucleotide polymorphisms in the *TLR10-TLR1-TLR6* gene cluster have also been associated with *ex vivo* whole blood IL-6 (but not TNF- α) hyperresponsiveness to organic dust and gram-positive components in agricultural workers.²⁶ The CD14 is a receptor for LPS, and the CD14/-159 T allele polymorphism has been associated with increased circulating levels of CD14 and increased prevalence of wheezing without association in lung function in farmers.²⁷ In this same study, there was no association between *TLR4*/Asp299Gly and lung function or wheeze in farmers.²⁷ Otherwise, there is a paucity of data understanding the role of genetic susceptibility in organic dust-associated respiratory diseases.

Compared with the robust acute inflammatory response to a 1-time organic dust exposure, repetitive exposures result in a comparative reduction in pro-inflammatory cytokines/chemokines levels but a persistence of lung neutrophils, lymphocytes, and recruited monocyte/macrophages in animals studies, which has been termed the chronic inflammatory adaptation response.^{22,28} Similarly, a 1-time organic dust exposure induces an increase in AHR in mice, and this response is lost with repetitive exposures,²⁸ consistent with the adaptation response but differs from experimental allergy asthma murine models. Correspondingly, pig farmers demonstrated an attenuation of symptoms, lung function, bronchial responsiveness, and markers of airway inflammation compared with first-time exposed naive individuals.¹⁵ Nevertheless, these pig farmers had markers of low-grade, persistent inflammation compared with never-exposed individuals.¹⁵

Organic dust exposures can induce the recruitment of CD4⁺ T-helper (Th) 1 cells, Th2 cells, Th17 cells, innate lymphoid cells, airway epithelial cells, and monocyte/macrophage activation responses.²⁹⁻³¹ In animal models, organic dust extract exposure induces lung alarmin IL-33 release, Th1/Th17 responses, and increased serum immunoglobulin (Ig) E levels without evidence for a Th2-mediated response or airway eosinophil influx.^{22,32} The Th17 cells and IL-17 are recognized to induce the recruitment of neutrophils and monocytes/macrophages, contributing to the pathogenesis of asthma and COPD.^{33,34} In severe equine asthma syndrome, a naturally

occurring chronic organic dust airway disease in horses that shares similarities with asthma in humans, a similar Th17 response has also been demonstrated.³⁵

Regulators of organic dust-associated airway inflammatory response include the anti-inflammatory cytokine IL-10. In a cross-sectional study of 625 veterans with farming experience, higher baseline blood concentrations of IL-10 were associated with higher FEV₁/FVC and inversely associated with whole blood stimulated Δ TNF- α and Δ IL-6.³⁶ In IL-10-deficient mice, organic dust extract-induced airway inflammation, neutrophil influx, and lung pathology were elevated, and this response was reversed with IL-10 supplementation.³⁷ Furthermore, short-term, lung-delivered recombinant IL-10 favorably hastened airway inflammatory recovery processes following acute, high-dose inhaled LPS exposure in mice.³⁸ In addition, airway cathelicidin (LL-37), an antimicrobial and LPS-neutralizing peptide implicated in tissue repair,³⁹ was increased in farmers with and without COPD versus healthy urban persons.⁴⁰ In advanced stages of COPD, LL-37 levels have been found to decrease, possibly highlighting the role of this peptide in antimicrobial protection.⁴¹

RISK FACTORS

Cumulative exposure to organic dust represents an important factor in disease development. In a cross-sectional study in Ethiopia, there was a high incidence of chronic respiratory symptoms in flour mill workers.⁴² The presence of chronic respiratory symptoms was associated with working in the mixing department (aOR 5.3; 95% CI 1.68–16.56), work experience of 6 to 9 years (aOR 5.1; 95% CI 2.05–12.48), work experience 10 or more years (aOR 2.5; 95% CI 1.01–6.11), and working 8 or more hours per day (aOR 2.4; 95% CI 1.16–5.10).⁴² The degree of exposure is also important. Andersson et al⁴³ demonstrated that each year of high exposure to soft paper dust (defined as > 5 mg/m³) was associated with a 0.87% predicted decrease in FEV₁ (95% CI -1.39 to -0.35) and a 0.54% decrease in FVC (95% CI -1.00 to -0.08).⁴³ In contrast, a Danish register-based cohort study did not find an association between the cumulative organic dust exposure and COPD in the farming or wood industry despite lagging their variables 10 years to consider the period of disease development of COPD and the healthy worker survivor effect.⁴⁴ Instead, they noted a decreased risk of COPD in the highest exposed group (adjusted rate ratio 0.63; 95% CI 0.56–0.70).⁴⁴ However, it was suggested that tobacco smoke may have confounded the results to suggest a need for additional longitudinal studies.⁴⁴ Indeed, the confluence of tobacco smoking is important. Guillemin et al⁴ demonstrated lower forced expiratory flow between 25% and 75% of vital capacity and forced expiratory flow at 50% of vital capacity in duck hatchery workers who smoked compared with nonsmoking workers.

The role of atopy as a risk factor is less clear. Dairy farmers who had persistent airflow limitation were more likely to have at least 3 positive tests for allergen-specific IgE compared with dairy farmers without persistent airflow limitation.⁴⁵ House dust endotoxin has been associated with atopic and nonatopic asthma in farming populations across the United States. In a large case-control study involving a cohort of farmers and their spouses, Carnes et al⁴⁶ demonstrated that increasing endotoxin levels was associated with higher odds of current asthma (unadjusted OR 1.30; 95% CI 1.14–1.47), and moreover, endotoxin was

TABLE 1. Risk factors associated with development of organic dust–associated respiratory diseases

Risk factor	Respiratory disease
Cumulative exposure	COPD/chronic bronchitis HP IPF
Very high concentration, 1-time exposure	ODTS
	HP
Tobacco smoking	COPD/chronic bronchitis Byssinosis
Composition of organic dust (eg, endotoxin)	Atopic and nonatopic asthma COPD/chronic bronchitis Byssinosis
Age* (eg, young adults)	Obstructive lung disease
Atopy*	Asthma
Timing of exposure (eg, late- vs early-life exposure)	Atopic and nonatopic asthma
	COPD/chronic bronchitis
Micronutrient deficiencies* (eg, zinc deficiency)	COPD/chronic bronchitis

*Additional studies are needed to fully understand its impact in the development of respiratory diseases.

associated with atopic asthma (aOR 1.38; 95% CI 1.09–1.74) and nonatopic asthma (aOR 1.24; 95% CI 1.07–1.43) after adjustment for sex, smoking status, race, and season.

Timing of exposure may also be important. Exposing mice to Amish organic dust extracts prior to the onset of experimental allergen-induced asthma sensitization and challenge resulted in reduced airway inflammatory outcomes.⁴⁷ In contrast, when mice were exposed to swine confinement organic dust extracts after allergen-induced asthma sensitization and challenge phase, there was potentiation of airway inflammatory outcomes.⁴⁸ Among U.S. farmers, a nonlinear relationship between endotoxin and asthma was described, with higher endotoxin levels associated with current asthma (OR 1.30; 95% CI 1.14–1.47) and modifiable by early-life farm exposure.⁴⁶ Notably, the association between dust endotoxin and asthma was higher in individuals not born on a farm (OR 1.67; 95% CI 1.26–2.20) than in those who were (OR 1.18; 95% CI 1.02–1.37). This highlights that the “protective effect” of early-life farming exposure may be linked to endotoxin exposure.^{46,47}

The impact of age as a risk factor for the development of organic dust airway disease has been difficult to establish. Earlier studies (1990s) demonstrated that a relative excess of respiratory symptoms and reduced lung function were higher among swine producers aged 26 to 35 years, which may have reflected more intense exposures.⁴⁹ It has also been demonstrated that “young (7–9 wk old)” mice had a more robust inflammatory response to swine confinement organic dust exposures than “older (12–14 mo old)” mice.⁵⁰

Micronutrient deficiencies represent global health concerns⁵¹ and may represent an additional risk factor. Nutritional zinc insufficiency has been associated with lower pulmonary function (FEV₁/FVC $P = .03$; trends for FEV₁ $P = .056$) among veterans with history of farm exposure in COPD individuals.⁵² In animal studies, dietary vitamin D⁵³ and docosahexaenoic acid (omega-3 fatty acid)⁵⁴ supplementation demonstrated reduced airway inflammatory indices following exposure to organic dust extracts.

Thus, nutritional approaches may potentially reduce organic dust–associated airway inflammation, but future studies in humans are necessary. A summary of the risk factors and respiratory disease(s) associated with organic dust exposure is shown in Table 1.

RESPIRATORY DISEASES ASSOCIATED WITH ORGANIC DUST EXPOSURES

Organic dust toxic syndrome

Organic dust toxic syndrome (ODTS) is a complex disease entity that can develop acutely, usually occurring within hours of exposure to very high dust concentrations, also referred to as acute febrile syndrome or grain fever. The risk of its development increases with concentration and duration of exposure. Symptoms include fever with generalized malaise, myalgias, dyspnea, nonproductive cough, chest tightness, and nausea.⁵⁵ Laboratory testing typically reveals leukocytosis with neutrophilia. Chest imaging, oxygen saturation, and pulmonary function test may be unremarkable.⁵⁶ Most cases of ODTS are mild, with symptoms usually resolving within 24 hours, but can persist for 2 to 5 days.⁵⁵ In contrast to HP, ODTS lacks prior sensitization to antigens driving its pathogenesis. Furthermore, the role of corticosteroid therapy remains uncertain in ODTS.⁵⁷

Asthma

Adult exposure to organic dusts in industries such as farming, soft paper, and cotton are associated with an increased risk of developing asthma.⁵⁸ A comprehensive meta-analysis revealed that exposure to dust from paper/wood, flour/grain, and textiles can raise the risk of asthma by 48%,⁵⁹ but information regarding preexisting asthma was not included. Reducing exposure also decreases the frequency of asthma exacerbations.⁶⁰ The term “asthma-like syndrome” refers to acute, nonallergic airway responses commonly seen in agricultural settings, characterized by chest tightness, wheezing, and/or shortness of breath. This syndrome may also manifest as a cross-shift decline in FEV₁, often linked to acute neutrophilic airway inflammation. Unlike classic asthma, this syndrome can occur upon first exposure, suggesting an inflammatory rather than an allergic reaction.

Byssinosis

Byssinosis refers to a specific respiratory disease directly caused by cotton dust (textile industry). Breathlessness, cough, and chest tightness are more severe at the start of the work week but may evolve to include productive cough and exertional dyspnea with repeated exposures.⁶¹ Tobacco smoking is an additional risk factor.⁶² Ocular and nasal irritation may also be present.⁶³ Disease progression has been categorized into distinct stages. Initially, there is a stage of irritation (0–5 y), which often improves upon cessation of exposure. The next phase is temporary incapacity, usually occurring after 10 or more years. The final stage involves complete disability, characterized by chronic bronchitis and emphysema.⁶⁴ Diagnosis is based on the World Health Organization grading system and Schilling criteria^{65,66} that includes symptoms and the weekday affected.⁸ The severity of byssinosis correlates with a more rapid decline in pulmonary function.^{65,66} Note, swine confinement workers also report worsening respiratory symptoms with weekday exposure and improvement when away from work,⁶⁷ and these symptoms are rarely related to allergy to porcine proteins.^{68–70}

TABLE II. Characteristics and diagnostic features associated with various organic dust-associated respiratory diseases

Organic dust-associated disease	Clinical symptoms	Causative agents	Diagnostic procedures	Lung function testing	Imaging
Asthma	Respiratory	Livestock and crop dusts, wood dust, cotton dust, mold, pollens, bacteria, chemicals	Possible eosinophilia ± specific allergen sensitivity	Variable obstructive pattern Cross-shift ↓ in FEV ₁ > 10%	Normal, bronchial wall thickening, air trapping
Asthma-like syndrome	Respiratory	Grain and farming dust, mold, bacteria, chemicals	Allergen skin testing is typically negative ± Airway neutrophils	Variable obstructive pattern Cross-shift ↓ FEV ₁ , < 10%	Normal, bronchial wall thickening, air trapping
Byssinosis	Respiratory	Cotton dust	Allergen skin testing is typically negative	Variable airflow limitation Across workday variability	Normal, bronchial wall thickening to opacities
ODTS	Respiratory and systemic	High concentrations of organic dust	Leukocytosis, specific IgE and IgG testing is typically negative	Normal with possible obstructive or restrictive pattern	Normal, ground-glass opacities possible
COPD, chronic bronchitis	Respiratory and systemic	Livestock and crop dust, wood dust, cotton dust, mold, bacteria, chemicals	± Skin testing, may be positive in ACO	Obstructive pattern with limited reversibility	Air-trapping, bronchiectasis, emphysema
HP	Respiratory and systemic	Mold, bacteria, avian proteins, vegetable and animal dust, chemicals	Specific IgG detection, ↑ BALF CD8 ⁺ T cells, airway neutrophils	Restrictive pattern, reduced DLCO	Mosaicism, ground-glass opacities, centrilobular nodules, reticulation, traction bronchiectasis
IPF	Respiratory and systemic	Vapors, gases, dust, fumes, metal dust, wood dust, silica dust	Allergy skin testing typically negative, UIP lung biopsy	Restrictive pattern (irreversible at late stage), reduced DLCO	Honeycombing, ground-glass opacities, traction bronchiectasis, reticulation

DLCO, Diffusing capacity of carbon monoxide; UIP, usual interstitial pneumonia.

COPD/chronic bronchitis

The prevalence of COPD/chronic bronchitis among non-smokers varies between 2% and 4.2%⁷¹ with a systematic review demonstrating associations between farming exposure (ie, cattle, swine, poultry, and crop farming) and airflow limitation or chronic bronchitis.⁶ Livestock farmers were more likely to have chronic bronchitis (OR 1.9; 95% CI 1.4–2.6) and COPD (OR 1.4; 95% CI 1.1–1.7) than crop farmers.⁵ Farmers with allergy have significantly lower FEV₁, and the effects of farming and specific agents on COPD were substantially greater in farmers with atopy.⁵ Despite modernization efforts in the dairy industry that have reduced COPD prevalence,^{72,73} traditional dairy farming remains a risk factor for COPD with additive smoking effects observed.⁷⁴ In poultry work, prevalence rates of COPD were higher in individuals with longer exposure regardless of smoking status.⁷⁵ In cotton work, particularly those workers exposed to both jute and hemp dust, the frequency of chronic bronchitis in retired workers who previously smoked was higher (20%) than currently smoking workers (17%).⁷⁶ Working in dense dust areas, active smoking, being older than 40 years of age, being an exsmoker, and working in the factory for a period exceeding 15 years were significantly associated with bronchitis and emphysema development.⁷⁶

Asthma-COPD overlap

Asthma and COPD are prevalent respiratory conditions that can overlap in 15% to 20% of patients,⁷⁷ referred to as asthma-COPD overlap (ACO). Individuals with COPD displaying asthmatic characteristics, as well as asthma patients with a history of smoking who develop non-fully reversible airflow obstruction, fall into this ACO category.⁷⁸ Diagnosis of ACO in COPD patients typically reflects the presence of reversible airflow obstruction, type 2 inflammation with airway or peripheral blood eosinophilia, or a previous physician's diagnosis of asthma.⁷⁹

The association of ACO with work-related asthma (WRA) or organic dust exposure is not well established. A U.S. Behavioral Risk Factor Surveillance Survey found that 51.9% of adults with WRA and 25.6% of those with non-WRA had also been diagnosed with COPD.⁸⁰ Those with concurrent WRA and COPD experienced more severe asthma exacerbations and outcomes than those with non-WRA and no COPD. In a study identifying ACO in an occupational asthma (OA) cohort of 304 subjects by Ojanguren et al⁸¹ in Montreal 86% were diagnosed with OA alone and 14% with occupational ACO. The occupational ACO group was older, required higher doses of inhaled corticosteroids, had longer exposure to offending agents, was more frequently exposed to low molecular weight agents, and showed less atopy

TABLE III. Exposure reduction and environmental controls for management and prevention strategies of organic dust exposure—associated lung disease

Focus area	Description
Environmental risk assessment	Applicable to agriculture, textile, woodworking, and construction industries Avoidance of high dust and endotoxin exposure tasks. However, this may not be socioeconomically feasible ¹²⁴
Environmental monitoring	Regular air quality monitoring Use of air sampling devices to measure the concentration of organic dust particles
Environmental controls	Implementation of engineering controls Improving ventilation systems ¹²⁵ Incorporating dust extraction and collection systems. Using mobile recirculating ventilation systems ¹²⁶ Ensuring that machinery and equipment are well-maintained
Work practice controls	Modification of task execution to reduce dust exposure Wetting down surfaces to prevent dust from becoming airborne Using tools and machinery that produce less dust Ensuring proper cleaning and maintenance procedures Adopting newer, cleaner technologies ¹²⁷
PPE: education and training	Incorporation of PPE: masks, respirators, and protective clothing. Proper training and education are required Workers should be educated on Risks of organic dust exposure Recognizing hazardous conditions Proper use of control measures and PPE Regular training sessions can reinforce this knowledge and keep workers informed about new practices or equipment
Policy and regulation compliance	Compliance with local and international health and safety regulations Adherence to occupational exposure limits for different types of dust Implementation of recommended safety practices
Worker health surveillance and education	Baseline/prework health examinations Regular health examinations, including lung function tests, chest x-rays, and allergy testing Education on trigger avoidance and proper medication use Support groups and counseling can also be beneficial Smoking cessation because smoking represents an additive effect Medical plan with rescue inhalers, bracelets as necessary

PPE, Personal protective equipment.

compared to the OA group.⁸¹ In Finland, a study highlighted the significant association between the risk of ACO and the presence of mold odor in the workplace.⁸² A Finnish study demonstrated that asthma patients exposed to vapors, gases, dust, or fumes in their occupation were more likely to develop ACO than those without such exposures.⁸³

Hypersensitivity pneumonitis

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis, is a complex syndrome caused by a non-IgE-mediated allergic reaction to organic particles or low molecular weight agents involving type III (immune complex-mediated) and type IV (delayed-type hypersensitivity) reactions.⁸⁴ Symptoms can vary widely depending on the duration and intensity of exposure and typically include cough, fever, chills, dyspnea, and fatigue. These symptoms can appear acutely, often 4 to 8 hours after exposure, or develop insidiously, particularly if the exposure to the antigen persists. Lung inflammation is characterized by lymphocytic and frequently granulomatous features that can result in lung fibrosis.⁸⁴ The occupational causes of

HP, been recently described in a systematic review,⁸⁵ includes farmers (farmer lung) and bird breeders or pet bird owners (bird fancier lung) as well as woodworkers, cheese manufacturers, and textile workers. Diagnosing HP involves a combination of clinical evaluation, lung imaging, lung function tests, and sometimes lung biopsies.⁸⁶ The primary treatment for HP is avoidance of the offending antigen. Corticosteroids are often used. In advanced fibrotic stages, management is more complex, requiring additional intervention including possible lung transplantation.

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis is a chronic, progressive, fibrosing interstitial pneumonia of unknown cause, primarily occurring in older adults. It is defined by the presence of radiological and/or histological usual interstitial pneumonia and has a poor prognosis with a median survival of 2.5 to 4 years.⁸⁷ Although organic dust exposures have been proposed in the development and/or exacerbation of IPF, their direct link to IPF remains unclear.⁸⁸ An international collaborative review

TABLE IV. Key areas of future research and development to advance knowledge and care of individuals with organic dust–associated respiratory disease

Focus area	Comment
Mechanism of disease	<ul style="list-style-type: none"> • In-depth understanding of the cellular and molecular pathways involved to aid in developing targeted therapies and interventions • Research into understanding resolution and recovery following organic dust exposure to develop novel targeted approaches • Further evaluation of the role of organic dust in the pathogenesis of IPF because little is known. Understanding and differentiating IPF from other forms of ILD that are directly caused by organic dust exposure, such as HP, which can present with fibrosis but has a distinct pathogenesis and clinical course from IPF
Long-term health effects	<ul style="list-style-type: none"> • Longitudinal studies to track the long-term health impacts of organic dust exposure • Understanding the progression of diseases over time and identifying any late-onset effects
Genetic and environmental interactions	<ul style="list-style-type: none"> • Understanding how genetic predisposition interacts with environmental exposure to organic dust • Deciphering why some individuals are more susceptible than others to developing airway diseases
Improved diagnostic tools	<ul style="list-style-type: none"> • Developing more sensitive and specific diagnostic tools for early detection of airway diseases related to organic dust • Tools needed range from biomarkers, imaging techniques, and lung function tests
Better exposure assessment methods	<ul style="list-style-type: none"> • Advancing methods for assessing and quantifying organic dust exposure in different environments • Improving the understanding of dose-response relationships and knowledge of accurate exposure limits
Preventive strategies and interventions	<ul style="list-style-type: none"> • Developing more effective preventive strategies and interventions, including improved personal protective equipment, workplace modifications, and educational programs
Treatment modalities	<ul style="list-style-type: none"> • Exploring new treatment options, including pharmacological and nonpharmacological therapies • Assessing the efficacy of existing treatments in managing symptoms and slowing disease progression
Impact of climate change	<ul style="list-style-type: none"> • Understanding how climate change might influence the generation and distribution of organic dust and the subsequent impact on airway disease
Public health policies	<ul style="list-style-type: none"> • Developing and evaluating public health policies and guidelines to protect workers in high-risk industries • Assessing the economic impact of such diseases on individuals and health care systems
Global perspectives	<ul style="list-style-type: none"> • Considering the global diversity in types of organic dust and varying workplace standards • Developing a global consensus and applicable guidelines through international collaborative research

ILD, Interstitial lung disease.

and meta-analysis assigned an attributable fraction for occupational exposures to the burden of IPF of 26%, calculated from 11 studies.⁸⁹ In this study, several exposure categories (vapors/gases/dust/fumes, metal dust, wood dust, silica dust) but not agricultural dust were significantly associated with IPF.⁸⁹ However, a U.S. multicenter, case-control study identified several occupations that were associated with IPF, including farming, livestock raising, hairdressing, raising birds, stone cutting/polishing, and jobs with exposure to metal dust and vegetable/animal dust.⁹⁰ Awadalla et al,⁹¹ in a multicenter, case-control study in Egypt, demonstrated that the risk of IPF was higher in women working in farming, raising birds, and with occupational exposures to animal feeds, dust, and pesticides. Moreover, a case-control study in Italy found that farmers, veterinarians, and gardeners had a particularly high risk of developing IPF, and the risk increased with increased lengths of exposure.⁹²

The issue of incorrectly diagnosing chronic HP as IPF has been raised. A 2013 study revealed that, out of 46 patients initially diagnosed with IPF following established guidelines, 20 were later found to have HP.⁹³ Many of these cases were associated with bird-related antigens, particularly from feather bedding.⁹³ De Sadeleer et al⁹⁴ studied 244 patients with IPF to demonstrate that IPF patients with a history of exposure to mold or birds had improved survival rates compared with those without such exposure, although survival was less than that of HP.

DIAGNOSTIC STRATEGIES

Presentation

Organic dust exposure generally triggers symptoms of cough, chest tightness, wheezing, mucus production, and shortness of breath, even in healthy individuals. In asthma and COPD, exposure can further exacerbate symptoms, reduce lung function, and increase in AHR.⁹⁵ Flulike symptoms can be found in ODS and HP, and progression to irreversible pulmonary fibrosis, restriction, and diminished diffusing capacity of the lungs for carbon monoxide can be seen in HP and IPF.^{84,87} Clinical, functional, and radiological characteristics of organic dust–associated respiratory disease are summarized in Table II.⁹⁶

Testing

Lung function. Lung function testing is recommended in exposed, at-risk persons because spirometry is particularly valuable for workers with preexisting obstructive diseases or smokers^{49,97} to monitor lung function over time and establish a dose-response effect.^{98,99} For example, swine-confinement workers often exhibit an accelerated loss of lung function, evidenced by a decrease in FEV₁ during a work week.^{100,101} Changes in lung function (FEV₁) across a work week, particularly in textile workers, aid in predicting disease.¹⁰² A restrictive ventilatory defect may also be observed in HP or IPF, as well as impaired gas exchange (reduced diffusing capacity of the lungs for carbon monoxide) and/or hypoxemia during exercise.⁸⁶

Measuring fractional exhaled nitric oxide (FeNO) may also be warranted. Exhaled nitric oxide increased from baseline value of 7.5 (range 5.7–13.7) to 13.4 (range 10.5–17.5) parts per billion after swine facility exposure.¹⁰³ In addition, the rise in FeNO post-work shift, along with diminished pulmonary function, established across-shift FeNO as an effective, noninvasive method to assess airway inflammation in textile workers.¹⁰⁴ Serial FeNO measurements at home versus work settings can further validate a dose-response relationship.¹⁰⁵

Laboratory tests. The detection of serum-specific IgE and IgG antibodies of organic dust components can be beneficial to establishing causation. For example, patients with grain dust-induced symptoms may be sensitized to dust mites or cereal flour proteins (ie, wheat, rye, and barley).¹⁰⁶ However, the presence of specific IgE antibodies may not be indicative of symptomatic exposure, as in individuals exposed to corn dust, in whom specific IgE, IgG, and IgG4 antibodies were identified in both symptomatic and asymptomatic subjects.¹⁰⁷ Conversely, a cohort study of workers with asthma-like symptoms following grain dust exposure, confirmed through inhalation challenges, lacked evidence for specific serum antibodies.¹⁰⁸

The identification of antigen-specific IgG antibodies is a key diagnostic criterion for HP (Table I). Elevated antibody titers following exposure, or a decrease in levels after avoiding exposure, can further support this diagnosis. However, the absence of these antibodies does not rule out the disease, and their presence alone is not definitive because they may merely indicate exposure in asymptomatic individuals.⁸⁴ In a study involving asymptomatic swine-confinement workers, the presence of IgG antibodies to specific porcine proteins was noted, whereas IgE-mediated reactions to these proteins were rare.⁷⁰

Skin testing. Allergy skin prick testing may also be useful. Over 15% of grain handlers exhibiting airway symptoms demonstrated sensitization by skin testing to storage mites (*Lepidoglyphus destructor* and *Acarus siro*) and molds (*Candida*).¹⁰⁹ In addition, skin test sensitization to wheat and rye has been linked to a decrease in lung function during a work shift in grain workers.⁹⁸ However, the relationship between asthmatic response and symptoms with positive grain dust-extract skin testing remains unclear.^{108,109} An improvement of quality and standardization of these complex allergen extracts may potentially increase diagnostic accuracy.¹⁰⁶

Bronchoalveolar lavage. Bronchoscopy with bronchoalveolar lavage fluid (BALF) analyses represents an additional tool to characterize the pattern of airway inflammation. The BALF from workers recently exposed to swine environments demonstrated increased levels of neutrophils, lymphocytes, and macrophages compared with those exposed for longer durations.^{110,111} Moreover, higher BALF concentrations of IL-1 β , IL-6, IL-8/CXCL8, and TNF- α were demonstrated in exposed versus nonexposed individuals.¹¹² Notably, swine workers with at least 1.6 years of exposure demonstrated increased levels of IL-6, whereas TNF- α was not detected following acute exposure.¹¹³ In chronic stages of exposure, BALF may reveal an increased concentration of total cells, neutrophils, albumin, fibronectin, and hyaluronan¹⁰⁰ and striking increases in BALF cell mRNA for IL-17A.¹¹⁴

In HP, the BALF cellular profile is typically characterized by marked lymphocytosis (>50%) and a predominance of CD8⁺ T cells (also reflected in a low CD4⁺/CD8⁺ ratio).⁸⁴ This pattern is common in the acute and subacute stages of the disease. Following intense exposure or during resolution stages, a significant increase in neutrophils may also be observed.

The BALF can be used to distinguish between exposed workers who develop HP or alveolitis and those who remain asymptomatic. This distinction is based on a low CD4⁺/CD8⁺ ratio for HP and elevated levels of hyaluronan for alveolitis.^{84,111} Despite these indicators, limited data have been demonstrated for significant differences in cell counts within BALF between farmers exhibiting respiratory symptoms and those without symptoms.¹¹⁵ This highlights a need for further research to validate the efficacy of BALF in differentiating symptomatic from asymptomatic exposed workers because most studies compare exposed workers (including symptomatic and asymptomatic workers) with unexposed controls.^{100,111}

Inhalation challenges. Inhalation challenges have confirmed the capacity of organic dust to induce respiratory symptoms and enabled analysis of lung function and inflammation profiles.^{116,117} Inhalation tests using extracts from durum wheat and corn dust have proven particularly useful in demonstrating AHR in grain handlers.¹¹⁸ These challenges have also been useful in assessing the impact of endotoxins and dust mites in triggering airway inflammation in sensitized patients, although the results have been mixed.^{108,109,119} In cases of suspected HP, inhalation challenges are recommended to help identify the specific causative agent. The effectiveness, applications, and safety of allergen challenge tests in respiratory disorders have been recently reevaluated.¹²⁰ These tests are predominantly viewed as tools for research purposes. In the United States, inhalation challenges to identify the cause of HP are generally not performed, primarily owing to regulatory constraints.

Imaging

Chest imaging, notably high-resolution computed tomography, can assess the disease activity and aid in diagnosis (Table I).⁹⁶ Chest radiograph findings are often nonspecific, including patterns like ground-glass or interstitial opacities, consolidations, or micronodules, but may appear normal in many patients.⁸⁴ High-resolution computed tomography is recommended for symptomatic individuals with abnormal lung function tests, and it is valuable in determining the necessity and location for lung biopsy.¹²¹ In the active or early stages of disease, transient pulmonary infiltrates, isolated diffuse ground-glass opacities, mosaicism, and centrilobular nodules may be observed.¹²² Bronchial wall thickening and air trapping can be identified in obstructive diseases like asthma and COPD/chronic bronchitis.¹²³ In HP, mosaicism and diffuse ground-glass centrilobular nodules may indicate small airway involvement. During the fibrotic stages of HP and IPF, features such as reticulation, traction bronchiectasis, and architectural distortion are observed, signaling irreversible disease.¹²²

MANAGEMENT AND PREVENTION

The management of airway diseases caused by organic dust involves both supportive care and standard medical interventions. The primary focus is on reducing exposure to the



What Is Hypersensitivity Pneumonitis?

Medically Reviewed by Paul Boyce, MD on January 18, 2025 | Written by Loraine Fick

4 min read

You may not think it's a big deal when you [breathe](#) in dust, but for some people, it could bring on a lung disease called hypersensitivity pneumonitis. It's an allergic reaction to particles in the dust, and it can cause symptoms like [coughing](#) and shortness of breath. You can get things back to normal if you get treated early and avoid breathing the stuff you're allergic to.

There are a variety of things that can cause hypersensitivity pneumonitis

when you breathe them in, including fungus, molds, bacteria, proteins, and chemicals.

Normally, the immune system -- your body's defense against germs -- causes [inflammation](#) in your lungs as it clears away the things you're allergic to. After a while, the inflammation stops. But in some people who are "hypersensitive," the lungs stay inflamed and cause the symptoms of hypersensitivity pneumonitis.

If you catch it early and stop breathing in more particles, your [lungs](#) can heal. If you breathe them in over and over, your lungs will stay inflamed, and scars may develop, which can make it hard to breathe normally.

It's hard to tell how many people have hypersensitivity pneumonitis because many don't get diagnosed or are mistakenly thought to have another lung disease, like [asthma](#).

Particles That Cause

Problems

You can breathe in troublesome particles in your home, at work, or almost any other place you usually go. It may take months or years before you become allergic to them.

Some sources of particles that can cause hypersensitivity pneumonitis include:

- Animal fur
- Fungus that grows in air conditioners, humidifiers, and heating systems
- Bird droppings and feathers
- **Mold** that grows on hay, straw, or grain animal feed
- Bacteria in water vapor from hot tubs

You may be more likely to get hypersensitivity pneumonitis if you have a job that puts you in contact with these particles, like farming, veterinary work, and lumber mill

operations. But most people who breathe them in won't get the [lung disease](#), so experts think certain genes play a role.

How You Get a Diagnosis

To find out if you have hypersensitivity pneumonitis, your doctor will want to know about the kinds of dust you may have been in contact with. They'll ask you questions like:

- Do you have any pet birds?
- Do you have a hot tub?
- Have you been around any water damage, especially from a humidifier, heater, or air conditioner?

Your answers to these questions will also help your doctor figure out the best treatment.

Your doctor will also listen for abnormal sounds in your lungs and will check the oxygen levels in your

[blood](#). You may also get tests like:

- [Blood](#) tests
- Chest X-ray or [CT scan](#)
- Tests to see how well your lungs are working
- Lung [biopsy](#) (removing a small piece of lung tissue)

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Symptoms and Types of Hypersensitivity Pneumonitis

There are three types, based on how long you have had the disease and how severe your symptoms are.

Acute. This type is brief and severe. It feels like you caught [the flu](#), and it happens after you've been around a lot of dust. Your symptoms should get better in a couple of days if you don't breathe in any more dust but will probably return if you do. Your symptoms could include:

- [Cough](#)
- Shortness of breath
- Tight feeling in your chest
- [Fever](#)
- Chills
- [Sweating](#)
- Tiredness

You might have these symptoms from 12 hours to several days.

Subacute. It can happen when you have low-level contact with the dust

over time. It may start out with a fever. Then shortness of breath, tiredness, and coughing can start over weeks or months. This type of hypersensitivity pneumonitis tends to get worse with time.

Chronic. This is a long-lasting form that happens after a low but long period of contact with dust. You may get symptoms like shortness of breath, tiredness, coughing, and weight loss that slowly get worse. This kind of hypersensitivity pneumonitis can lead to permanent [lung](#) scarring.

Treatment

The most important thing you can do is avoid the dust that caused your hypersensitivity pneumonitis. If you have a chronic form of the disease, you may take a [steroid](#) medicine to help curb [inflammation](#). They have side effects like [weight gain](#) and higher [blood sugar](#).

Early studies suggest that [drugs](#) that

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Acute Respiratory Illness Following Occupational Exposure to Wood Chips -- Ohio

The inhalation of organic dust contaminated with microbes has been recognized as an occupational hazard for persons who work with decomposing vegetable matter (1-6). An outbreak of illness caused by such inhalation occurred in Ohio in 1983. The investigation that followed is described below.

On June 21, 1983, five employees at a municipal golf course became ill with an influenza-like syndrome within hours after manually unloading a trailer truck full of wood chips. Physicians from the city health department examined and tested all golf-course employees who had helped in the unloading and requested assistance from the National Institute for Occupational Safety and Health in evaluating the outbreak (7). On June 24, a questionnaire was administered to those employees exposed to wood chips, and their medical records were reviewed. The investigators inspected the unloaded wood chips, collected samples, and interviewed the wood chips' vendor.

The wood chips were brought to the golf course in an enclosed, 40-foot trailer. Eleven employees participated in some aspect of the unloading process. Although fresh chips had been ordered, the vendor included old chips that had been stored in the front of the truck for approximately 1 year. Unloaded chips from the front were grossly moldy, and cultures revealed a wide variety of mesophilic and thermophilic bacteria and fungi.

A case was defined as the presence in an employee of at least five of the following six symptoms after exposure to the wood chips: malaise, fever, difficulty breathing, chest tightness, headache, and cough. Except for cough, which was reported by two persons who did not meet the case definitions, each symptom was reported more frequently by ill persons than by well persons ($p < 0.05$) (Table 1).

All five ill employees had worked in very dusty conditions without respiratory protection while unloading the front of the trailer on the afternoon of June 21. The time from beginning of unloading until onset of illness ranged from 4 hours to 16 hours (median 13 hours). None of the workers were hospitalized, but one reported to a local emergency room, and two were too ill to work the following day. Within 48 hours, symptoms were very much improved; within 72 hours, all affected workers had completely recovered.

The other six employees included three who had unloaded fresh chips from the back of the trailer on the morning of June 21, one supervisor who had briefly checked on the unloading process, and two workers who finished unloading the front of the trailer on the morning of June 22 but wore air-purifying respirators. Thus, all five workers who had unloaded the moldy wood chips without respiratory protection became ill, compared with none of the other six workers.

The mean total white blood count in ill workers (11,000) was significantly higher than in those who remained well (8,100); a significantly greater mean absolute polymorphonuclear leukocyte count was also found among the ill (ill: 8,300, well: 5,600) ($p = 0.008$). The erythrocyte sedimentation rate was elevated in all five ill workers but in only two of the six who did not become ill. Except for one individual who had radiographic changes due to previous surgery, all those who became ill had normal chest radiographs and spirometry. Furthermore, none had positive tests for precipitating antibodies against a standard panel of 11 antigens associated with hypersensitivity pneumonitis extracts of three types of wood chips and 12 microbial organisms isolated from the wood chips (8). Tests for complement fixing antibodies as evidence of histoplasmosis produced low titers in both the acute and convalescent sera of the ill workers.

On the basis of clinical and epidemiologic evidence, the investigators concluded that this episode probably represented an outbreak of self-limited, acute toxic reaction associated with inhalation of large amounts of dust heavily contaminated with microbial toxins from decomposing vegetable matter. Reported by P Asmussen, MPH, WL Duff, ER Heidtman, Middletown City Health Dept, CJ Burrell, AM Richmond, MD, Middletown Social and Health Center, Ohio; Hazard Evaluations and Technical Assistance Br, Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note

Editorial Note: In 1975, an apparent toxic pulmonary illness was reported among 10 farmers who became ill several hours after removing moldy silage (1,2). The authors of that report referred to the illness as "pulmonary mycotoxicosis" because the etiology presumably involved toxic components of inhaled fungal organisms (1). Others have recognized an apparently identical syndrome but have applied other names to it. Thus, it has been variously referred to as (1) "silo unloader's syndrome" to contrast it with silo filler's disease, a toxic pulmonary edema following inhalation of the oxides of nitrogen in freshly filled silos (3); (2) "precipitin test negative farmer's lung" to emphasize its clinical similarities to and its pathogenetic differences from farmer's lung disease, an immunologic lung response to microbial antigens in moldy hay (4); and (3) "organic dust toxic syndrome" (ODTS), a generic designation to emphasize that mycotoxin exposure is not a necessary prerequisite and that the syndrome is not restricted to either silo exposures or farming occupations. A striking similarity has been recognized between ODTS and "mill fever" in cotton textile workers, "grain fever" in grain elevator workers, and "humidifier fever" in building occupants exposed to air from highly contaminated ventilation systems (3,5). Similar to the current report, moldy wood chips were etiologically linked to symptoms of ODTS in individuals exposed to dust from wood chips that had been stored in basements as a fuel source for wood-burning furnaces (6).

Epidemiologically, ODTS often occurs in small outbreaks, with illness affecting all or most individuals who have had intense exposure to microbially contaminated vegetable dust (3,9). The syndrome is clinically characterized as an acute febrile illness with respiratory symptoms; onset usually occurs 4-12 hours after exposure. General malaise, headache, and cough are common symptoms, while dyspnea is variably present. Chest auscultation usually reveals normal breath sounds; the chest x-ray is remarkably clear; and pulmonary function may be only slightly impaired. Leukocytosis with a predominance of polymorphonuclear leukocytes is the rule, and serologic testing for precipitating antibodies associated with farmer's lung disease is usually negative.

With removal from exposure, ODTS is a self-limited illness, occasionally resolving within 24 hours, often within several days, and sometimes only after a few weeks. To date, no deaths have been reported, and there is no evidence for residual pulmonary fibrosis. Some individuals, however, have been hospitalized with severe symptoms, and a few have undergone diagnostic bronchoscopy and lung biopsy. Bronchoalveolar lavage has revealed a predominance of PMNs, and biopsy has demonstrated an acute inflammation without granulomas, as well as an assortment of microorganisms in the airways (1).

ODTS probably occurs much more frequently than is currently recognized. Only serious solitary cases or those that occur in suspicious clusters are likely to come to medical attention, and when a history of

environmental exposure is elicited, these are often misdiagnosed by physicians as silo filler's disease or farmer's lung disease (3). Because the incidence, etiologic agent(s), and pathogenesis of ODTD remain unknown, physicians are encouraged to report to appropriate health authorities any influenza-like illness following intense exposures to organic dust. Based on current understanding, symptomatic treatment alone should suffice. Prevention measures should include storing vegetable matter in a way that limits microbial growth and wearing appropriate respiratory protection when intense exposure to organic dusts cannot be avoided.

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This page last reviewed 5/2/01

Residents Feedback about Palm Bay Tree Recycling Site

Contacts with various residents near and around the Palm Bay Tree Recycling Site (PBTR)

3/6/25 and 3/7/25...

Spoke to a "Savon" apt 2201 in the apartments directly across from the PBTR Business. We observed the gentleman and his aunt using a leaf blower to remove dust from the driveway area into the street.

He stated "the dust is a problem, My aunt has asthma and has difficulty breathing"

Spoke to numerous residents at the Mercury Cove Apartment Complex (behind the PBTR Business)

No one at the residential area would give their names, however, they were willing to discuss the issues

Bldg 421

Elderly woman states she has one lung and stays indoors mostly because of the noise during the day and the dust

Bldg 481

A gentleman in his 40/50's who doesn't speak English asked to have his relative a 18 year old female to speak

She said "it is very dusty and they stay in most of the time"

Bldg 491

A female around 25+ years of age was walking her dogs

She states "I work during the day and don't hear the noise. However, at night there is a weird smell and lots of dust."

A female in her 20/30 states "she visits her sister and nephew and her sister and nephew have developed severe allergies because of the dust"

A male 30+ was working on his car and stated "he and his wife's car on many days has blankets of dust on their vehicles"



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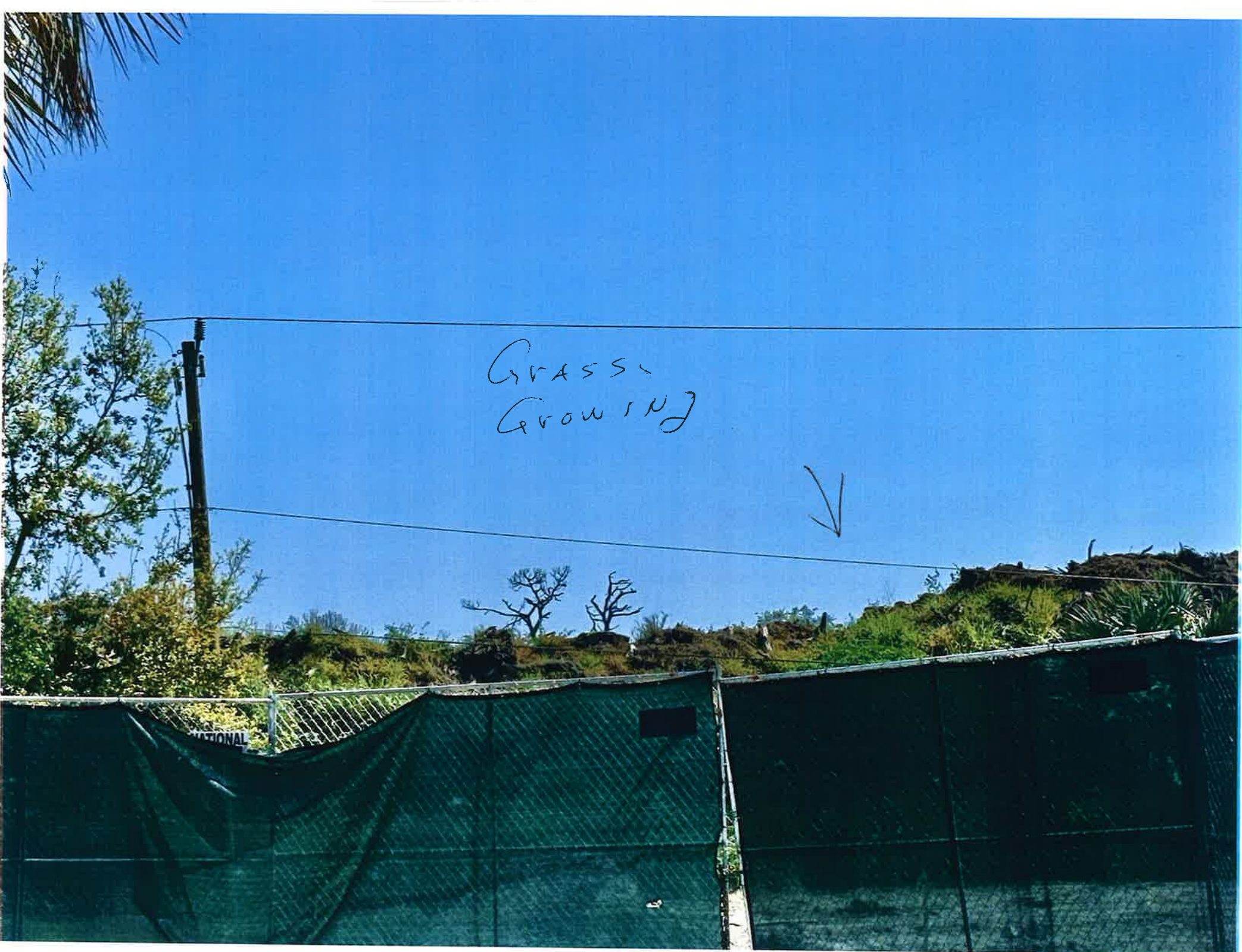








Grass
growing



Large
wood
pust
FLAKES
on
Auto
Held



Health Hazards Associated with Wood Dust and Fungi from Mulch Producing Facilities

8 May 2014

Presentation to the Environmental Sustainability Board

James and Cynthia Nickel
4904 Green Bridge Rd., Dayton, MD 21036

Contributors: Rob and Leslie Long
2701 Woodbine Rd., Woodbine, MD 21797

Mulch, Fungi and Wood Dust

Peer Reviewed Studies

- While mulch is generally considered "safe", the context is typically residential application, not piles of mulch covering acres 20 ft. high being churned daily.

Here are but five studies that begin to touch on the potential consequences.

- Fulminant Mulch Pneumonitis: An Emergency Presentation of Chronic Granulomatous Disease
 - Infectious Diseases Society of America
- Fungal spores: hazardous to health
 - US National Library of Medicine, NIH
- Adverse Human Health Effects Associated with Molds in the Indoor Environment
 - American College of Occupational and Environmental Medicine
- Pulmonary responses after wood chip mulch exposure.
 - US National Library of Medicine, NIH
- Binding of *Aspergillus fumigatus* spores to lung epithelial cells and basement membrane proteins: relevance to the asthmatic lung.
 - I.M. Bromley and K. Donaldson

Fungal Spores: Hazardous to Health

- “Fungi have long been known to affect human well being in various ways, including disease of essential crop plants, decay of stored foods with possible concomitant production of mycotoxins, superficial and systemic infection of human tissues, and disease associated with immune stimulation such as hypersensitivity pneumonitis and toxic pneumonitis. The spores of a large number of important fungi are less than 5 micron aerodynamic diameter, and therefore are able to enter the lungs. They also may contain significant amounts of mycotoxins. Diseases associated with inhalation of fungal spores include toxic pneumonitis, hypersensitivity pneumonitis, tremors, chronic fatigue syndrome, kidney failure, and cancer.”

W.G. Sorenson – US Library of Medicine, NIH

Wood Dust: Hazardous to Health

- "Cancers have been associated with wood dust exposure. The National Institute for Occupational Safety and Health (NIOSH) considers both hardwood and softwood dust to be potentially carcinogenic to humans. The three types of cancers associated with wood dust exposure are nasal and sinus cavity cancer, lung and other cancers, and Hodgkin's disease. The wood and cancer relationship was studied by Milham (1974), who conducted a mortality study involving the AFL-CIO United Brotherhood of Carpenters and Joiners of America. This study supports the hypothesis that wood contains carcinogenic agents. The cancer mortality patterns found were:
 - Excess lung cancer in acoustical tile applicators and insulators.
 - Excess gastrointestinal cancer in pile drivers.
 - Excess leukemia lymphoma group cancers in millwrights, mill workers, and lumber and sawmill workers.
 - Excess lung and stomach cancer in construction workers with the greater excesses found in workers in major urban areas.
- Hodgkin's disease has also been associated with wood dust."

Wood Dust Exposure Hazards AEX-595.1-2006

Thomas L. Bean, in collaboration with Timothy W. Butcher and Timothy Lawrence
Ohio State University

Wood Dust and Fungi Risk Mitigation

- None
 - Grinding wood and frequent churning of mulch piles ejects dust and spores into the air.
 - Fungal Spores can't be seen with the naked eye [< 5 microns] and are airborne to greater distances than mulch dust. Mulch processing requires the use of water to reduce mulch dust both as a irritant and to mitigate against mulch fire risk.
 - Moisture stimulates the growth of mold.
 - Fungi/fungal spores are dormant when dried.
 - It is inevitable that you will have both, mulch dust AND fungal spores airborne, spread by the prevailing winds.

Howard County Test Case

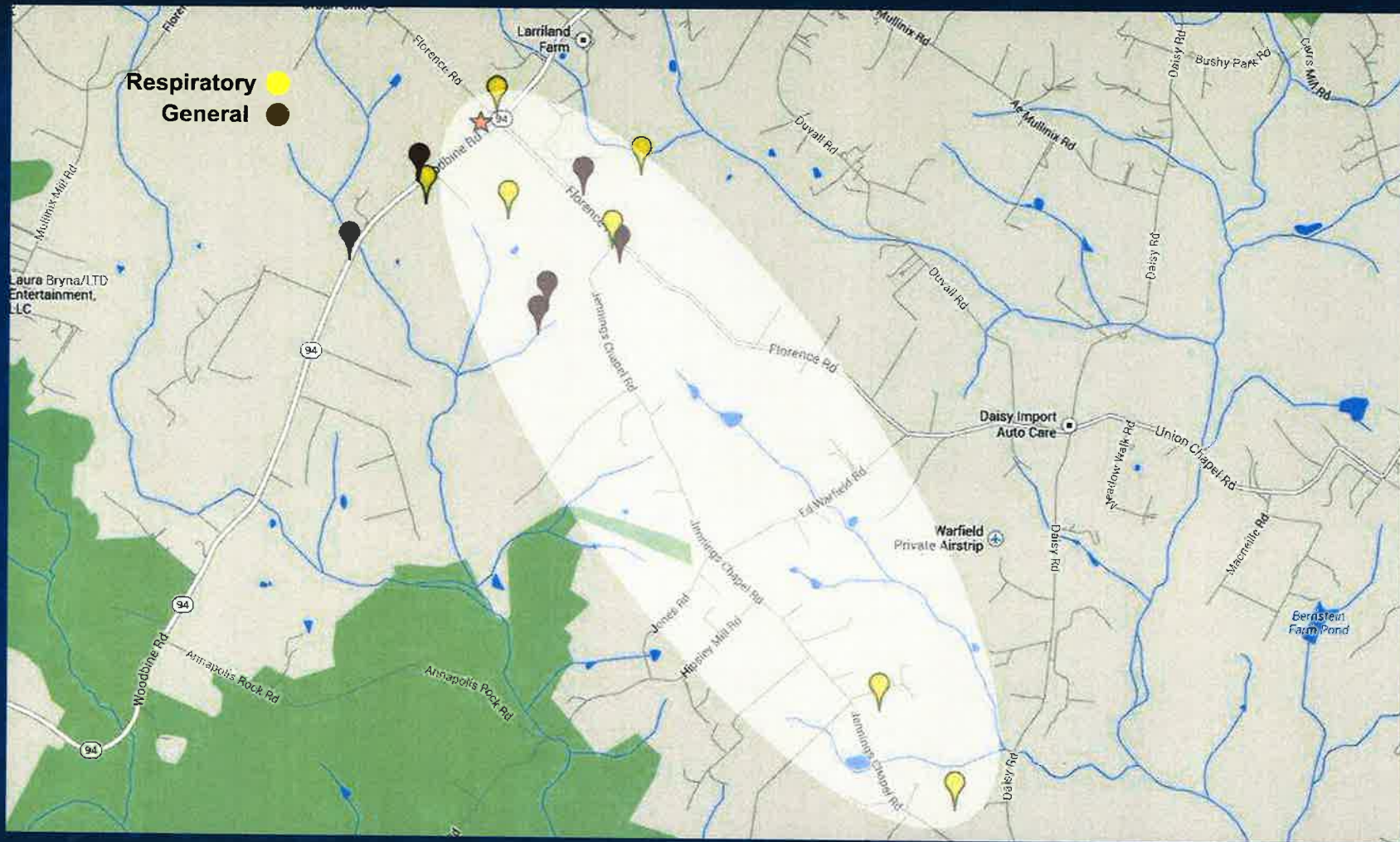
Sponsored by Oak Ridge Farms, LLC

- Oak Ridge Farms, LLC has inadvertently provided Howard County with a test case of the possible consequences of mulching and composting facility on Agricultural Preservation Properties.
- The residents of Woodbine, including farmers and livestock, have been the unwitting subjects in this test case.
- We can learn a lesson from this experiment on Woodbine Residents.
- Observation, sample collection and analysis of the Woodbine experience is necessary.

Requests to DPZ for Zoning Inspections Oak Ridge Farms

- Residents of Woodbine made 17 requests to DPZ [Nov-Dec 2013]
- 9 Requests explicitly stated respiratory related issues
 - All with health issues were age 51 and older
 - 2 residents under care at Johns Hopkins were tested and found to have wood particulate matter in their respiratory system
 - Distance between the Oak Ridge facility and most distant health issue was 3.1 miles [airborne fungi spores can travel longer distances than wood dust]
- 8 Requests were of a general nature
 - Traffic
 - Pollution & contaminants
 - Decline in air quality
 - Odor
 - Noise, e.g., “louder than a combine”
 - Residents can feel the vibrations of the grinding equipment
 - Occurring 6 or 7 days a week

Inspection Requests - Clustering



Prevailing North West Winds Affecting Residents to 3.1 Miles
Florence and Jennings Chapel Roads may provide “corridors” for wind

Summary - Woodbine Test

- Woodbine Residents have been unwitting participants in a “test” resulting from operations by Oak Ridge Farms, LLC.
- In a relatively short period of time, residents and livestock are showing symptoms commonly associated with fungal spore contamination and wood dust inhalation.
- The clustering of those affected is consistent with prevailing winds.
- The affected persons in Woodbine are more than 3 miles away from the Oak Ridge Farms, LLC facility.
- Horses and livestock are showing respiratory distress.

Summary – Health Hazards

- Fungal Spores and wood dust from mulch are known health risks to humans.
- Hazards of mycotoxins and mycotoxigenic fungi are well documented in peer reviewed studies within the US and Internationally.
- Wood dust has been long established as a carcinogen.
- **There are no mitigation strategies.**