SUMMARY: Wood dust exposure has been documented in reports from around the world as the cause of asthma, non-allergic pulmonary disease, and sino-nasal cancer. Less frequently, wood dust has been described as causing alveolitis and pulmonary fibrosis, and dermatitis. A summary document by ACGIH in 2005 proposed a TLV of 1 mg/M3 for wood dust in general, and 0.5 mg/M3 for Western Red Cedar, with a sensitizer designation. The conclusion ACGIH reached was based on its extensive review of the literature. The International Agency for Research on Cancer (IARC) designated oak and beech dusts as Type 1A carcinogens, as did the NTP. Most recently, California’s OEHHA added wood dust to the Prop 65 list of carcinogens.

An extensive review of the literature was performed for the development of this report for HEAC, focusing especially on studies from 2005 to current. Overall, there seems to have been a decline in exposure levels compared to older studies, with corresponding decrease in positive associations. In addition, recent research into mechanism of injury and pathophysiology has led to increased biological plausibility for wood dust causation of both cancer and non-malignant disorders.

Exposure monitoring and protection issues are complicated with wood dust, since there are over 12,000 species of trees worldwide, and a wide range of industrial applications. There are studies which show that biological contaminants, such as fungi and molds, and chemical contaminants, such as formaldehyde, can cause or contribute to the pathology. In addition it seems that different types of wood cause injury through different mechanisms on a molecular basis. (Whether that has any major relevance is unclear at this time.)

The US has 34 million asthmatics and 16 million with chronic bronchitis/chronic obstructive pulmonary disease, with estimated 14 million with COPD undiagnosed. 47 million US citizens have metabolic syndrome, characterized by obesity, diabetes, abnormal cholesterol and hypertension. 24% of the US workforce has metabolic syndrome. Obesity is now considered an independent risk factor for lung disease. These figures are all expected to rise over the next 20-30 years.

Because of the history of lung disease findings at higher wood dust exposure levels, with recent lower wood dust dose studies showing no or little adverse effect, and because of the clear carcinogenicity of wood dust vis a vis sino-nasal cancer, and our workforce’s growing risk factors as described above, HEAC support of ACGIH’s position of lowering the PEL to 1 mg/M3 seems reasonable and feasible. Western Red Cedar is a special case and should have a PEL of 0.5 mg/M3 and sensitizer designation.

BACKGROUND:

CAS–none

CHEMICAL FORMULA–None

SYNONYMS: n/a

CHEMICAL AND PHYSICAL PROPERTIES: Wood dust is defined as wood particles resulting from processing or handling of woods. Woods are divided into hardwood—from broad leafed deciduous trees which flower; and softwood, from conifers which do not shed their leaves in the winter, plus tropical woods or “exotics”. 
Chemically, wood is composed of cellulose, hemicelluloses and lignin, plus hundreds of high and low molecular weight compounds which serve a variety of functions, including protecting the wood from attack by insects etc. These include terpenes, lignins, and stilbenes, tannins, flavonoids, quinines, and phenols. In addition, wood can be contaminated by fungi, molds, & insects, which can be irritants or allergenic in their own right. ACGIH notes that 5% to 30% of the wood mass can be composed of these other compounds.

Wood is often treated with a variety of other toxic compounds including copper, arsenic, formaldehyde, and pentachlorophenol as preservatives, lead based paint in the past, epoxy and other glues in production of pulp/wood fiber products and plywood, etc.

Western red cedar is a softwood widely used in construction because of it’s resistance to molds, fungi and other causes of decay. California no longer has large strands of this wood, but imports it from other states and Canada

FORMS, USES AND APPLICATIONS: Rough cutting operations which “shatter” the wood, generate larger particles (20-30 um) which are much less likely to be inhaled. Other processes such as sanding generate smaller ones (1-5 um). The type of operation being performed is an important factor in exposure.

CURRENT WORKPLACE LIMITS AND PUBLIC HEALTH LIMITS: (Usually measured as “inhalable” particulate mass because of effects on both upper and lower respiratory systems)

  - Cal OSHA : current—Wood dust 5 mg/m3
  - Carcinogenicity: Not designated, but OEHHA recently added it to Prop 65
  - Fed OSHA: current-Wood Dust: 5 mg/M3 8 hr PEL, and 15 minute STEL of 10 mg/M3 for all except WRC, which has 8 hr PEL of 2.5 mg/M3 (OSHA originally proposed a soft wood standard of 5 mg/M3 and hardwood 1mg/M3, but based on feasibility issues and critiques of the studies, revised it as above)
  - ACGIH 2005: TLV-TWA 1 mg/M3 inhalable particulate mass—all species except WRC for which the proposed TLV–TWA is 0.5 mg/M3, and Sensitizer designation.
    - Carcinogenicity: IARC-A1-Confirmed human carcinogen: oak, beech
    - A2-Suspected human carcinogen::-birch, mahogany, teak, & walnut
    - A4: Not classifiable as a human carcinogen: all other wood dusts, including Western Red Cedar

MAJOR COMERCIAL USES: Construction, furniture and cabinet making, sawmills, carpentry. Outdoor construction projects = more air moving, larger dust particles generated than indoor furniture making, cabinetry, sawmills. Industry description: “Companies that cultivate and/or manufacture timber, mill lumber, wood, and/or wood products for construction.” Major changes in industry in recent decades due to increased environmental regulation, use of smaller trees and wood fiber boards instead of planed wood and plywood for construction. An industry document indicates over 90% of the timber in USA is on private land

HEALTH ENDPOINTS:
**ASTHMA/ALLERGIC UPPER AIRWAY DISEASE:** Mechanism of allergic asthma unclear, clearest for WRC, although some other studies have shown allergic effects of other woods, equivocal for pine, others not native to US or widely used here.

**NON-ALLERGIC PULMONARY DISEASE:** Chronic non allergic pulmonary disease is the most prevalent health effect with numerous studies showing increased symptoms of chronic bronchitis or chronic obstructive pulmonary disease (COPD), and/or decrements in pulmonary function. Limited studies with good exposure data, and there are numerous other criticisms of the studies, including small numbers, single industrial focus, exposure data obtained by old methods, etc. Generally, an irritant effect is postulated, due to wood dust itself, and/or contaminants.

**OTHER LUNG DISORDERS:** Alveolitis, hypersensitivity pneumonitis, pulmonary fibrosis have all been reported associated with wood dust exposure, but may be actually due to fungi and molds involved in processing.

**CANCER END POINT:** mechanism of carcinogenicity unclear, but new research demonstrates specific mutations. Focus is on sino-nasal cancer, both adeno and squamous. Laryngeal cancer and lung cancer are also of concern.

**DERMATITIS:** Wood dust can cause both Type I and Type IV hypersensitivity as well as irritant dermatitis.

**REVIEW-NON-ALLERGIC PULMONARY DISEASE**

Overwhelmingly studies looking at health effects of wood dust exposure describe non allergic pulmonary effects rather than immunologically mediated problems such as asthma or alveolitis. The mechanism by which these effects are produced is thought to be an irritant effect causing an inflammatory reaction which leads to chronic bronchitis and non reversible airway disease.

The ACGIH TLV document critically reviewed studies of chronic non allergic pulmonary disease in development of its proposed TLV of 1 mg/M3, presented in 2005. Other studies since then were reviewed for this document. Glindmeyer et al (2008) is the most comprehensive analysis published to date, following a cohort of employees in ten different wood processing plants over a 5 year period, with exposure data and medical surveillance, including spirometry. The exposure data was presented in several formats, including the standard inhalable dust used in most studies previously, but also with respirable fraction broken out, because of their focus on pulmonary disorders. The authors also analyzed the exposure data by separating wood solids from the non wood fraction of the dusts (volatiles, water, fungi, etc). Spirometry was performed according to ATS standards and equipment was calibrated and maintained appropriately. Measurements were compared to a very large data base of white and African-American US workers who were non smokers and not exposed to pulmonary hazards on the job.

This study was initiated, according to the authors, in part because of criticisms of previous studies as being too small, single industry, foreign workforce, questionable endpoints, such as symptoms review only, etc., The exposure assessment revealed geometric mean values of personal inhalable dust samples ranging from 0.82 to 2.51 mg/M3 across the ten plants, with geometric standard deviations ranging from 2.1-2.8. The respirable dust fraction was 0.10-0.23 mg/M3 with geometric standard deviation in the range of 2.0-3.5. Their conclusion was “Exposure to WS was not associated with significant adverse effects” Separating out the wood dust solids from other wood components such as terpenes, and possible contaminants such as fungi, may be removing the actual cause of disease in different wood processing operations. If fungi in sawmills or chemicals such as glues in plywood manufacturing operations are part of the etiology for lung diseases seen in those industries, calculating disease rates based on their removal is artificial unless one postulates new control mechanisms for that part of the hazard.
Other studies more recent than those discussed in the ACGIH TLV document include Veneri’s study on upper and lower airway disorders in woodworkers published in 2007 (abstract only - Italian) which analyzed medical surveillance reports of 197 woodworkers with median dust exposure of 2.1 mg/M3. Each had an examination and spirometry. They found decrease in FEF 25-75 correlated with no use of respiratory protection and weakly with length of service. A pathologic decrease in vital capacity correlated with wood dust exposure. They concluded that chronic irritation of the upper and lower respiratory tract are caused by exposure to wood dust below the European legal exposure limit of 5 mg/M3. Focusing on FEF25-75 is not a useful endpoint however, since it is a highly variable measurement of small airway function on spirometry. In 2008 Jacobsen et al published a study investigating the relationship between changes in lung function and cumulative exposure to wood dust in 1112 woodworkers (927 male, 185 female) and 235 controls. This was a 6 year longitudinal study, involving a questionnaire, spirometry, personal dust sampling at baseline and passive dust monitoring on follow-up. The spirometry methods were described and appropriate. A significant decrease in exposure was identified from the initial period 1997-98 to 2003-04. At baseline it was 0.94 +/- 2.10 mg/yr/M3, and at follow-up it was 0.60 +/- 1.60 mg/yr/M3. The results showed a significant decrease in lung function among female woodworkers compared to controls, but not males. They note that the inhalable dust concentrations in their study were low. Schlunssen et al (2002) evaluated respiratory symptoms and lung function among Danish woodworkers, with questionnaires (n=2033, plus 474 controls), spirometry pre and post shift, (n=2423) and passive dust sampling (n=1579). The arithmetic mean for inhalable dust was 1.19 +/- .86 mg/M3, which they noted was low. Again, the most significant finding was a dose response relationship seen in women smokers with high dust exposure, and inhalable dust concentrations and asthma symptoms. Fransman et al (2003) studied work exposure and respiratory symptoms in 112 New Zealand plywood workers, but again exposures were low (majority did not exceed 1 mg/M3), with only 57 dust samples, and symptoms were the outcome measure. “Asthma symptoms” were more common in the mill workers than the general population, were associated with duration of employment, and were reported to lessen or go away on weekends or vacations. They did look at endotoxin levels, abietic acid, terpenes and formaldehyde in a smaller number of samples each. Symptoms were more common in workers with high exposure to formaldehyde, but did not show a strong relationship to the other exposures. Aside from the reliance on self report symptoms, disease classification may be an issue in this study also. Rusca et al (2008) looked at exposure to bioaerosols in 111 sawmill workers in Switzerland. They found that the airborne concentration of fungi exceeded the Swiss recommended limit and was associated with “bronchial syndrome” (cough and expectoration) but there was not an associated decline in lung function.

**REVIEW: ASTHMA**

Asthma is a chronic inflammatory disorder of the airways characterized by reversible (fully or partially) bronchial tube spasm. Symptoms include wheezing or coughing, shortness of breath, with wheeze, cough and/or prolonged expiration on chest examination, and decreased FEV1 on spirometry. Common findings on histopathology include inflammatory cell infiltration, hypertrophy of smooth muscle, hyperplasia of goblet cells, airway edema, and denudation of airway epithelium. Asthma affects approximately of the population. Identifiable predisposing factors include atopy and obesity. There are an estimated 34 million people with diagnosis of asthma in US, and over 40 million with allergic rhinitis. About 9-15% of adult onset asthma is considered to be occupational, with rates varying by country and level of industrialization.
Asthma due to wood dust exposure has been reported for decades. Asthma due to a specific wood dust exposure is the subject of many individual case reports from around the world, usually due to exotic woods not used frequently in the US. The cases describe symptoms related to a specific wood exposure, and are verified by IgE, prick and/or challenge testing. (Algranti 2005, Bosomba 1991, Bush 1983, Eire 2006, Fernandez-Rivas 1997, Garces 1995, Gozalo 1988, Hinjosa 1986, Kospohl, Obata 2000, Reijuli 1994) Best studied is Western Red Cedar, used widely in this country, where the specific allergen has been identified as plicatic acid, and the disease severity related to dose and duration of exposure. (Chan-Yeung 1992 & 1994, Cockcroft 1984, Coke 1990, Paggiori 1987, Vedal 1986 & 1988). Eastern white cedar has also been found to be allergenic, with the same etiologic agent plicatiic acid (Cartier 1982, Malo 1994). In contrast, pine dust as an allergen was investigated by Skovsted et al (2003), evaluating 365 exposed and 116 non exposed workers. Some exposed and some non exposed were found to have IgE antibodies to pine, but small percentages. A study evaluated health effects of exposure to the tree which produces natural rubber latex, evaluating 103 workers and 76 unexposed, and found a dose dependent increase in wheeze, nasal symptoms, asthma and reduced spirometric values. (XX).

Exposure to oak and beech dust was found to cause sore throat and bronchial hyperresonsiveness but not decline in FEV1 with increasing dose/duration.

Perez Rios et al published in Allergy in 2009 “A meta-analysis on wood dust exposure and risk of asthma”. A quality scale for study selection was applied and nineteen studies were included, (3 cohort, 12 case control and 4 mortality studies). The pooled RR was 1.53 (95% CI 1.25-1.87). For studies of Caucasians alone the results were 1.59 (95% CI 1.26-2.00), and studies of Asian populations 1.15 (95%CI 0.92-1.44)

In addition to wood dust itself, other candidates for allergic asthma and rhinitis in wood processing industries include molds, glues and other agents used in the processing or finishing of wood products. Winck et al did prick allergen tests with 3 common fungi in cork production and had negative results in all asthmatic and non asthmatic workers tested.

Williams (2006) did a “critical analysis of studies concerning reports of respiratory sensitization to certain wood dusts used in the US (oak, beech, pine, and western red cedar) and concluded that the effects seen in the studies were due to underlying bronchial hyperactivity, not allergic sensitization.

**REVIEW: CANCER:**

Wood dust has been associated with cancer of the respiratory tract in a number of studies, and has been identified as a carcinogen by IARC, NTP and most recently, by California’s OEHHA. While there are studies suggesting a link between wood dust exposure and laryngeal and lung cancers, good documentation is available only for nasal cancer.

Cancer of the nose and paranasal sinuses (sino-nasal cancer) is quite rare in the US, with incidence rates about 1/million. Multiple studies have been done showing an elevated risk of this cancer in wood workers from a variety of trades. A recent analysis of cancer and occupation from the extensive registries in the Scandinavian countries revealed a high RR for sino-nasal cancer and wood trades. In 2009 Pukkala, from the Finnish Cancer Registry evaluated records of 15 million people aged 30-64 years and the 2.8 million incident cancers diagnosed in them through the personal identity code linkage system used in all the Nordic countries. The results are presented as “Standardized Incidence Ratio-SIR” the number of cases observed divided by the expected number. Occupations were grouped into 53 active and one group of economically inactive folks. “Wood workers“ was one of the 53 codes. For nasal adenocarcinoma in male wood workers, the SIR was 5.50(4.60-6.56).
Most of what we now know about carcinogenesis has been discovered since the 1970s, with the development of ability to look at specific changes to parts of the genome. In the past 5 years there has been major work looking at the mechanism of wood dust and carcinogenesis. In brief, it is believed that 5-10 sequential mutations are required to transform a normal cell into a cancer cell. (See attached diagram from the latest edition of Harrison’s internal medicine text). A significant mutation is inactivation of the tumor suppression gene P53. Several studies have investigated this recently. Homila et al found a high prevalence of TP53 mutations in a large group of samples from 358 sino-nasal cancers with data on occupational exposure, and found the mutation levels significantly elevated with duration of exposure 24 years (OR 3.5, 96% CI 1.2-10.7. In a further experiment, they reported the actual sequence change for 159 of the TP53 mutations, identifying C-T transitions, base changes in the coding region, G-T transversions and identified the actual codons most frequently affected.

How wood dust could cause these mutations has also been studied in papers relevant to pulmonary tissue damage. In 2009, Pylkkanen et al in Finland used dust from pine, birch and oak, generated dusts with 90% less than 5 um, and exposed human bronchial epithelial cells from The American Type Culture Collection in 3 concentrations (10, 50, &500um/ml and 5 duration periods from 0.5-24hr. All wood dusts caused cytotoxicity, dose dependant and statistically significant at 2 & 6 hrs compared with controls. Exposure to all three woods stimulated reactive oxygen species (ROS) and induced capase-3 protease activity. Maata et al studied wood dust particle induces pulmonary inflammation in mice to identify the mechanisms by which lung diseases develop. They used oak and birch, created fine dusts with 90% 5nm or less, and used titanium dioxide as a control dust., instilled intranasally 2 xs week for 3 weeks, and then airway hyper reactivity to methacholine was measured by body plethysmography. The mice were then killed and blood, bronchial alveolar lavage fluids and tissue samples were taken. The results showed that repeated exposure to he oak and birch dusts caused influx of inflammatory cells into the lungs, and on a molecular level was associated with increase in several cytokines, chemokines and chemokine receptors in the lung tissue.

REFERENCES

Aguna EN et al: The prevalence of occupational asthma and rhinitis among wood workers in south-eastern Nigeria. 2007; 9(1)52-5

Ahman M et al: IgE-mediated allergy to wood dusts probably does not explain the high prevalence of respiratory symptoms among Swedish woodwork teachers. Allergy 1995; 50:559-62


Basomba A et al: Occupational Rhinitis and asthma caused by inhalation of Balfouradendron riedelianum (Pau Marfim) wood dust. Allergy 1991; 46(4) 316-8


Brodkin CA & Rosenstock L: The relation between chronic respiratory symptoms and ventilator capacity in adults. Occup Med 1993 8(2) 363-74

Carson M et al: Occupational Exposure to wood dust. Health Effects and exposure limit values. Rev Epidemiol Sante Publique 2002; 50(2) 159-78 (ABSTRACT ONLY-ARTICLE IN FRENCH)

Cartier A et al: Occupational asthma caused by eastern white cedar (Thuja occidentalis) with demonstration that plicatic acid is present in this wood dust and is the causal agent. J Allergy Clin Immunol 1986; 77(4) 639-45


Cockcroft DW et al: Recurrent nocturnal asthma after bronchoprovocation with Western Red Cedar sawdust: association with acute increase in non-allergic bronchial responsiveness. Clin Allergy 1984 14 (1)61-8

Cote J et al: Outcome of patients with cedar asthma with continuous exposure. Am Rev Resp Dis 1990; 141(12) 373-6


Demers PA et al: Pooled reanalysis of cancer mortality among five cohorts of workers in wood related industries. Scan J Work, Environ Health 1995;21(3)179-190


Eire MA et al: Occupational rhinitis and asthma due to Cedroarana (Cedrelinga catenaeformis Ducke) wood allergy. J Investig Allergol Clin Immunol 2006; 16(6)385-387


Fernandez-Rivas M et al: Occupational asthma and rhinitis caused by ash (Fraxinus excelsior) wood dust. Allergy 1997; 52(20 196-9

Frew AJ et al: Lack of rome for mononuclear cel-derived histamine realeasing factors in occupational asthma due to western red cedar. Clin Exp Allergy 1993; 23(10) 861-7


Gozalo RF & Pelta FR: Occupational asthma due to exotic wood: Nesorgordonis papaverifera (danta or kotibe): Rev Mal Respir 1988; 5(1) 71-3 (ABSTRACT ONLY-ARTICLE IN FRENCH)


Hausen BM and Herrmann B: Bowmakers disease: an occupational disease in the manufacture of bows for string instruments 1990;115(5) 169-73 (ABSTRACT ONLY-ARTICLE IN GERMAN)


Heikkila P et al: Asthma incidence in wood processing industries in Finland in a register based study.Scan J Work Environ Health 2008; 34(1) 66-72

Herbert FA et al : Respiratory consequences of exposure to wood dust and formaldehyde of workers manufacturing oriented strand board. Arch Environ Health 1994; 49(60 465-70

Hessel PA et al : Lung health in sawmill workers exposed to pine and spruce. Chest 1995; 108(3) 642-6

Hinojosa M et al: Occupational asthma caused by African maple (Obeche) and Ramin: Evidence of cross-reactivity between these two woods. Clin Allergy 1986 16(2) 145-53

Homila R et al: Profile of TP53 gene mutations in sinonasal cancer. 2009 XXXXXXXXXXX


Innocenti A et al: Asthma and systemic toxic reaction due to cabreuva (Myrocarpus fastgiatus Fr. All.) wood dust. Med Lav 1991 82(5) 446-50


Jeebhay MF & Quirce S: Occupational asthma in the developing and industrialized world: a review. In J Tuberc Lung Dis 2007;11(2) 122-33

Kauffer E et al: Site comparison of selected aerosol samplers in the wood industry. Ann Occup Hyg 2009 advanced publication on line 12/31/09


Kespohl S et al: Identification of an obech (Triplochiton scleroxylon) wood allergen as a class I chitinase. Allergy 2005; 60 (6) 808-14

Kos PD: Wood dust exposure in wood industry and forestry Coll Anthropol 2005; 29(1)207-11


Liou SH et al: Respiratory symptoms and pulmonary function among wood dust exposed joss stick workers. Int Arch Occup Environ Health 1996 68(3) 154-60

Llorente JL et al: Genetic and clinical aspects of wood dust related intestinal-type sinonasal carcinoma: a review 2008. 1434


Maattan J et al: Immunolmodulatory effects of oak dust exposure to murine model of allergic asthma. Toxicol Sci 2007; 99(1);260-6

Maatta J et al: Mechanisms of particle induced pulmonary inflammation in a mouse model: exposure to wood dust. 2006 93 (1) 96-104


Moran CA et al: Primary adenocarcinomas of the nasal cavity and paranasal sinuses. Ear Nose Throat j 1991 70(12); 281-8


Obata H et al: Occupational asthma due to exposure to African cherry (Makore) wood dust. Inter med 2000; 39(11) 947-9

Paggiaro PL & Yeung C M: Pattern of specific airway response in asthma due to western red cedar (Thuja plicata): relationship with length of exposure and lung function measurements. Clin Allergy 1987 17(4) 333-9


***Post WK: Occupational estimated by a population specific exposure matrix and 25 years incidence rate of chronic nonspecific lung disease (CNSLD): the Zutphen Study


Reijula K et al: Sauna builder’s asthma caused by obeche (triplochiton scleroxylon) dust. Thorax 1994; 49:622-23


Sandven EW & Levy F: Serum IgG antibodies to mold spores in two Norwegian sawmill populations: relationship to respiratory and other work related symptoms. Am J Ind Med 1993 24(2 207-22

Scarselli A et al: Occupational exposure levels to wood dust in Italy, 1996-2006; Occup Environ Med 2008; 65-567-574

Schlunssen V et al: Cross shift changes in FEV1 in relation to wood dust exposure: the implications of different exposure assessment methods. Occup Environ Med 2004 61(10); 824-30
Schlunssen V et al: Detreminants of wood dust exposure in the Danish Furniture industry—results from two cross sectional studies 6 years apart. Annals of Occup Hyg 2008 52(4) 227-238


Shamssain MH: Pulmonary function and symptoms exposed to wood dust. Thorax 1992;47;84-87


Skovsted TA et al: Only few workers exposed to wood dust are detected with specific IgE against pine. Allergy 2003; 58(8) 772-779


Toren K et al Health effects of working in pulp and paper mills: Malignant Diseases. Am J Ind Med 1996; 29-123-130


Vanderplas O: Increase in non-specific bronchial hyperresonsiveness as an early marker of bronchial response to occupational agents during specific inhalation challenges. Thorax 1996 51(%) 472-8


Vedal S et al: Symptoms and pulmonary function in western red cedar workers related to duration of employment and dust exposure. Arch Environ Health1986;41(3)179-83

Veneri L: Study on prevalence of upper and lower airways disorders in woodworkers, using data from medical surveillance reports and exposure registers (Abstract only—article in Italian) G Ital Med Lav Ergon 2007; 29 (3 Suppl):833-5

Whitehead LW: Health effects of wood dust—relevance for an occupationa standard. Am Ind Hyg Assoc J 1982 43(9) 674-8

Williams PB: Critical analysis of studies concerning reports of respiratory sensitization to certain wood dusts. Allergy Asthma Proc 2005; 26(40 262-7

Wu X et al: A case-control study of wood dust exposure, mutagen sensitivity, and lung cancer risk. Can Epi biomarkers and prev 1995; 4; 583-588