FILE NAME: Talc (TALC)

DATE: 2004 June

DOC#: TALC177

DOCUMENT DESCRIPTION: Article from Law Journal Website - Is Toxic Tort Reform Leading to the Same Disability Claims with New Labels

 June 2004.



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# Publications

Article - June 7, 2004

**IS TOXIC TORT REFORM LEADING TO THE SAME DISABILITY CLAIMS WITH NEW LABELS (AND HOW DEFENDANTS CAN USE THIS TO THEIR ADVANTAGE)?**

by Sheila Flanagan

## 1. Introduction – The Changing Face of Occupational Lung Disease

Complex litigation in Northern California has seen a recent rise in silicosis and welding fume related lawsuits against an historical backdrop where asbestos related lawsuits were the norm. This trend, which is in part due to the self re-invention of some local plaintiff firms to stay profitable in the face of the threat of a federalized compensation program for asbestos-related injuries, raises many questions about personal injury litigation in general, and whether it is realistic to reform the legal system one toxic tort at a time. It also raises some interesting questions about the claimed disease processes involved in silica, welding fume and asbestos litigation, among others, and whether the more recent litigants are really a different population with different ailments and different exposures, or whether they are really litigants from the same pool of workers (construction workers, automobile mechanics, shipyard workers, electricians, plumbers, welders, etc.) against largely the same pool of defendants with re-labeled disease categories for the current legal climate.

Considering this question more closely from a defense standpoint illuminates how these cross-over diseases can be used to defendants' advantage. It is not always so easy to establish occupational lung disease specific to silica versus welding fumes or asbestos, talc, metal dusts, other minerals or inorganic compounds. It remains plaintiff's burden to prove it was your client's product or premises that caused plaintiff's illness, and that requirement includes proof that it was a substance or dust with some relationship to your client's product, premises or liability.

Welding fume exposure cases are a particularly illustrative example of the broad spectrum of exposure claims that are compressed together in the latest personal injury cases. Welding fume exposure encompasses a mixture of many fine fumes and gases produced from working with a variety of elements and compounds usually over many years. These include chromium, iron, steel, nickel, arsenic, asbestos, manganese, silica, beryllium, cadmium, nitrogen oxides, phosgene, acrolein, fluorine compounds, carbon monoxide, cobalt, copper, lead, ozone, selenium and zinc, to name just some. Focusing on the specifics of a given injury to a limited number of materials at a limited number of job sites or employers may help to shift the liability away from your client. In the alternative, the generic nature of many occupational lung diseases may assist you in spreading the liability amongst a larger group of materials and job sites decreasing your client's proportionate share of the liability.

For years, defense attorneys have discussed putting the absent chairs in play at the defense table in asbestos trials to emphasize what small aspect of any plaintiff's exposure one defendant might have had. When you add the chairs of the products, manufacturers and distributors of all of the

many minerals and compounds that produce chronic obstructive lung disease, interstitial fibrosis and/or dyspnea, the number of chairs in play may, in fact, fill a very large court room. This article considers some of the similarities and differences in the presentation of lung diseases depending upon the cause or causes. In some cases, it may be possible to shift the cause of a plaintiff's pulmonary complaints squarely away from a defendant's products and onto others with a different injurious component. In other cases, it may only be possible to spread the causes more evenly across causative factors and apportion among defendants more broadly. Below, categorized by common forms of medical presentation, is a discussion of the many potential causative agents for each type of presentation.

## 2. Common Symptoms of Occupational Lung Disease

### A. Dyspnea

Probably the most common symptom recited by all plaintiffs and their attorneys' medical experts is dyspnea, or shortness of breath. Dyspnea is a generic term for difficult or labored breathing with a wide range of etiologies and is a common symptom of lung disease due to silica, welding fumes, asbestos, talc, cadmium, ceramic fibers, beryllium, mica, kaolin, iron and steel dust, cotton, grain and wood dusts, ammonia, isocyonates, sulfur dioxide and coal mining, as well smoking, asthma, sleep apnea, heart disease and weight gain.

### B. Pulmonary Function Abnormalities

Basic pulmonary function abnormalities can be broken down into three basic interpretive categories: obstruction, restriction and reduction in diffusion capacity. Obstructive lung disease is due to a decrease in the amount of air that you can exhale from your lungs. This is caused by narrowing or blockage of the airways. Obstructive disorders include emphysema and asthma. Less air is inhaled and exhaled because the airways and air sacs lose their elasticity, the walls between many of the air sacs are destroyed and/or become thick and inflamed, and the cells in the airways may produce more sputum than usual, clogging the airways. Restrictive lung disease is caused by a decrease in the amount of air that the lungs can hold. The lungs become "stiff," reducing their ability to expand for inhalation due to processes such as sarcoidosis and pulmonary fibrosis.

Diffusion capacity measures the transfer of carbon monoxide across the capillary bed of the lungs. In the presence of normal amounts of hemoglobin and normal ventilatory function, the primary limiting factor of diffusion capacity is a decrease in the surface area of the capillary bed due to emphysema, loss of capillary bed, increased distance from terminal bronchiole to the capillary bed, and the mismatching of ventilation to blood flow.

Asbestosis, silicosis, welding fumes in non-smokers, [1] and mineral and metal dusts have been shown to cause restriction on pulmonary function tests. Silica, welding fumes in long-term smokers,[2] mineral and metal dusts, coal mining, smoking, and ammonia have been documented to cause obstruction on pulmonary function testing. All of these substances have been shown to produce a decrease in diffusion capacity.

### C. Chronic Obstructive Lung Disease (COPD)/ Emphysema

Silica, welding fumes, coal mining ceramic fibers, iron and steel dust, cotton, grain and wood dust, ammonia, cadmium, isocyonates, sulfur dioxide, as well as smoking have all been documented to cause symptoms of COPD (otherwise known as irreversible obstruction) on pulmonary function testing. COPD may also present with a reduced resting diffusion capacity (DLCO) and over-inflated lungs on chest x-ray. High Resolution CT scans (HRCTs) may show evidence of emphysema and small airways disease.

### D. Interstitial Fibrosis

Asbestosis, silicosis, welding fumes, cadmium exposure, coal workers pneumoconiosis (CWP), talc, aluminum, antimony, barium, beryllium, chromium, cadmium, lanthanides, nickel, tin, titanium, uranium, radon, zirconium, mica and kaolin exposures have all been claimed to produce interstitial fibrosis. Interstitial fibrosis has been categorized by the International Labor Organization (ILO) into three discreet categories: small rounded opacities, small irregular opacities and large opacities. These categories are further particularized by reading small opacities into sizes within their shape subcategory:

Small rounded opacities associated with silicosis and other lung abnormalities are classified by the following letter system:

p = opacity diameter up to 1.5 mm
q = diameter from 1.5 mm up to 3 mm
r = diameter from 3 mm up to 10 mm

Small irregular opacities associated with asbestosis and other lung abnormalities are classified by this letter system:

s = opacity diameter up to 1.5 mm
t = diameter from 1.5 mm up to 3 mm
u = diameter from 3 mm up to 10 mm

The extent of fibrosis or opacities is graded in terms of a profusion score that ranges from 0 for the least pervasive, to 3+ for the most pervasive. Interstitial fibrosis can be further classified by the lung zones where it is found to be the most prevalent. To this end, asbestosis is largely described as irregular opacities in the mid and lower lung zones. Silicosis is described as mostly rounded opacities primarily in the upper and middle lung zones. Welding fumes have been shown to cause interstitial fibrosis in the absence of silica exposure.[3]

Coal Workers Pneumoconiosis also has been described largely as primarily rounded opacities, mostly in the upper lung zones. However, irregular opacities have also been observed in a significant number of U.S. coal miners.[4]

Talcosis presents as both nodular (rounded) and irregular opacities consistent with both silicosis and asbestosis.[5] Mica-related fibrosis is also described as both nodular and irregular opacities, largely at the lung bases. Similarly, Kaolin related fibrosis is described as both nodular and irregular opacities in the mid and lower lung zones. Perlite miners have also shown in one study to present with diffuse peripheral reticular and nodular opacities common to complicated mixed-dust pneumoconiosis.[6]

Aluminum exposures in some forms such as aluminum powder, causes pulmonary fibrosis in the upper most portions of the lung. [7] Antimony, a metal commonly used with lead and in machine bearings, has been shown to cause pneumoconiosis.[8] [9] Barium can cause baritosis, a form of pneumoconiosis with profound evidence of fibrosis on radiographs without much evidence of physiologic impairment. Studies have documented clearing of barium related fibrosis with cessation of exposure.[10]

Exposure to beryllium can cause berylliosis. Berylliosis presents similar to sarcoidosis with non-necrotizing lung granulomas or fibrosis found mostly in the upper lung zones. Beryllium lymphocyte transformation tests of BAL cells or peripheral blood lymphocyte tests may be necessary to distinguish this disease from other forms of occupational lung disease. [11] Chronic cadmium fume exposure has been associated with pulmonary fibrosis. [12] Chromium fumes have been associated with interstitial lung disease and fibrosis in welders. Workers exposed to mica can present with diffuse interstitial fibrosis either in the upper or lower lung zones. Cobalt exposure can cause interstitial fibrosis with concomitant asthma, often referred to as hard metal disease. It can also present with enlarged hilar lymph nodes and be difficult to distinguish from sarcoidosis or berylliosis.[13] [14] Lanthanides, a group of 14 metals commonly used as alloys, have been

associated with granulomatous disease and lung fibrosis in recent years.[15] Copper sulfate exposure can also present in advanced disease as diffuse reticulonodular opacities, particularly of the upper lung zones, in a similar pattern to tuberculosis, or progressive massive fibrosis similar to latter stages of other pneumonconioses.[16] Studies of nickel-exposed workers have seen a significant number of irregular opacities of profusion score 1/0 or greater on radiographs and some, though fewer cases of rounded opacities with the same profusion score.[17] Sepiolite exposures have also been shown to cause interstitial fibrosis. [18]

E. Pleural Plaques

Pleural plaques are a relatively unusual finding and not common to many exposures. Asbestos is the most commonly found cause of pleural plaques and asbestos-related pleural plaques commonly are calcified. Talc, mica, kaolin, zeolite, sepiolite, wollastonite and shale exposures have also been documented to cause pleural plaques. However, these are without calcification.[19]

F. Lung Cancer

There are many epidemiologically proven occupational and environmental factors that increase an individual's risk of lung cancer. In complex litigation we may be familiar with lung cancer due to asbestosis, silicosis, and smoking. Epidemiologic studies also show an increased risk of lung cancer due to arsenic, nickel, chromium, aluminum, iron and steel founding, beryllium, radon, sulfuric acid, diesel exhaust, glass manufacturing, paint spray, mining, cadmium, chloromethyl ethers (BCME and CMME), certain coal processing, uranium and antimony.[20] [21] [22] [23] [24] [25] These elements are also found outside the occupational setting. As one example, cadmium is found in significant amounts in cigarettes with elevated amounts of cadmium in serum blood levels.[26]

Recent epidemiology suggests there is no increased risk of lung cancer from welding fumes. IARC sponsored a study of lung cancer risk in welders in the early 1990s that suggested a 34% increased risk of lung cancer in welders. [27] However, multiple large studies of welders since then have revealed negligible to no increased risk in lung cancer in this population.[28] [29] [30] [31] [32]

### 3. Use the Generic Presentation of These Indistinguishable Plaintiffs to Your Clients' Advantage:

Clearly, there is a wide variation in the radiologic and pulmonary function presentation of a broad spectrum of claimed occupational lung diseases. There is also significant crossover of what may have been asbestos-related lawsuits to silicosis and welding fume complaints. The Plaintiffs' Bar has learned to used the gray zones between disease categories to their advantage. In the right cases, there is no reason why defendants can't claim their own advantage.

As an example, many texts use the following three steps to diagnose silicosis: 1) history of sufficient exposure to silica (variable dependent upon the exposure level), 2) chest radiographs with evidence of nodular opacities consistent with the disease, and 3) no other concomitant illness that mimics silicosis.

It is this last step that, as defense counsel, we should not pass over too quickly. So many occupational lung diseases present almost exactly the same. So many plaintiffs also have very similar pulmonary function and radiographic presentations. Add to this that very few plaintiffs really present with a text book case of a specific occupational disease absent, at a minimum,

confounding factors for smoking and aging if not also weight gain, asthma, allergies and heart disease.

The lesson we can take from so many similar presenting occupational lung disease claims, is to use the generic presentation of a given plaintiff's disease process to your client's advantage in discovery:

- In deposition, determine what other occupations and other substances plaintiff may have had exposures to, and get details about duration and job duties.
- Evaluate the similarities and differences a plaintiff's claimed symptoms and exposures have to your client's liability area, and what alternative exposures may present similarly.
- Ask your medical experts about the other possibilities and point out other types of exposures that you may have uncovered in the discovery process.
- Thoroughly review medical records for evidence of alternative causation for a plaintiff's symptoms. Certain occupational diseases can be identified through blood testing and biopsies. If it is alleged that your client exposed plaintiff to silica, but his medical records show he has tested positive to beryllium lymphocytic transformation, your battle may be won. Similarly, if the allegation is that your client is in part responsible for plaintiff's exposure to welding fumes causing lung disease, but on biopsy the mineral dust analysis reveals mostly talc and peri-bronchiolar talc dust laden macrophages, then an alternative causation has been established.

Finally, it may not be viable to shift causation completely to another substance or disease process in many cases. However, this is not reason to give up alternative theories of causation, as there is always the opportunity to apportion causation between more substances, more disease processes, or more entities.

Bibliography:

Amandus, HE et al, "Significance of Irregular Small Opacities in Radiographs of Coal Miners in the U.S.A.," British Journal of Industrial Medicine, 1976;33: 13-17.

Antonini, JM et al, "Pulmonary Responses to Welding Fumes: Role of Metal Constituents," *Journal of Toxicology and Environmental Health*, Feb. 13 2004; 67(3): 233-49.

Becker, N, "Cancer Mortality Among Arc Welders exposed to Fumes containing Chromium and Nickel. Results of a third follow-up: 1989-1995," *Journal of Occupational and Environmental Medicine,* April 1999; 41(4): 294-303.

Churg, Andrew and Francis Green, *Pathology of Occupational Lung Disease,* Igaku-Shoin, New York, 1988.

Cooper, D., Prendergrass, EP et al, "Pneumoconiosis among Worker in an Antimony Industry," *American Journal of Roentgenology,* 1968; 103: 495-508.

Cugell, DW, "The hard metal Diseases," *Clinical Chest Medicine,* 1992; 13: 269-279.

Danielsen, TE et al, "Incidence of Cancer among Welders and other Shipyard Workers with Information on Previous Work History," *Journal of Occupational and Environmental Medicine,* January 2000; 42(1): 101-9.

Doig, AT "Baritosis: A benign pneumoconiosis," *Thorax,* 1976, 31: 30 – 39.

Elmes PC, "Perlite and Other Nuisance Dust", *J R Soc Med* 1987; 80: 403-440.

Funahashi, A et al, "Welders' Pneumonconiosis: Tissue Elemental Microanalysis by Energy Dispersive x-ray analysis," *British Journal of Industrial Medicine,* 1988; 45: 14-18.

Gamble et al, "An epidemiologic study of a group of talc workers," *American Review of Respiratory Disease,* 1979; 119: 741-753.

Groth, M et al, "Respiratory Symptoms in Danish Welders," *Scandanavian Journal of Social Medicine,* 1989; 17:271-276.

Gustavsson, P et al, "Occupational Exposure and Lung Cancer Risk: A Population-based Case-referent Study in Sweden," *American Journal of Epidemiology,* July 1 2000; 152(1):32-40.

Hendrick, DJ et al, *Occupational Disorders of the Lung: Recognition, Management, and Prevention,* W.B. Saunders, London, 2002.
IARC Monographs. *Chemicals and Industrial Processes Associated with Cancer in Humans,* Lyon, France, IARC, 1979.

IARC Monographs. *Some Metals and Metallic Compounds,* Lyon, France, IARC, 1980.

IARC Monographs. *Beryllium, Cadmium, Mercury and Exposures in Glass Manufacturing Industry,* Lyon, France, IARC, 1994.

IARC Monographs. *Chromium, Nickel and Welding,* Lyon, France, IARC, 1990.

IARC Monographs. *Silica, some Silicates, Coal Dust and para-Aramid Fibrils,* Lyon, France, IARC, 1997.

Liss, GM "Health Effects of Welding and Cutting Fume – An Update," Ontario Ministry of Labour, Final Report December 1996.

McDonald, JW et al "Rare Earth Pneumoconiosis: Analytical Scanning electron microscopy and literature review," *Modern Pathology,* 1995; 8: 859-65.
Stark, P "Vineyard Sprayers Lung – A Rare Occupational Disease, *J Can Ass Radiology,* 1981; 32: 183-184.

Meyer, KC, "Beryllium and lung disease," *Chest,* 1994; 106: 942-946.

Mitchell J, Manning GB, et al, "Pulmonary Fibrosis in Workers Exposed to Finely Powdered Aluminum," British Journal of Industrial Medicine, 1961; 18: 10-20.

Muir, DCF et al, "Prevalence of Small Opacities in Chest Radiographs of Nickel Sinter Plant Workers," *British Journal of Industrial Medicine,* 1993; 50: 428-31.

Pontkonjak, V et al, "Antimoniosis: A Particular Form of Pneumoconiosis. Etiology, Clinical, and X-ray Findings," *Internal Archives of Occupational and Environmental Health,* 1983; 51: 199-207.

Rizzato, G et al "The Differential Diagnosis of Hard Metal Lung Disease,: *Scientific Total Environ,* 1994: 150:177-83.

Schnorr, TM et al, "Mortality in a cohort of antimony smelter workers, *Am J of Industrial Medicine,* 1995; 27:759-770.

Shaham, J "Biological Monitoring of Exposure to Cadmium, a Human Cardinogen, as a result of active and passive smoking,"*Journal of Occupational and Environmental Medicine,* 1996; 38:1220-1228.

Simonato et al, "A Historical Prospective Study of European Stainless Steel, Mild Steel, and Shipyard Welders, *British Journal of Industrial Medicine,* 1991; 48: 145-154.
Steenland et al, "Lung Cancer in Mild Steel Welders," *American Journal of Epidemiology,* 1991; 133: 220-229.

Smith, TJ et al, "Pulmonary effects of chronic exposure to airborne cadmium," *American Review of Respiratory Disease,* 1976; 114: 161-169.

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