BEFORE THE WORKERS' COMPENSATION BOARD

STATE OF OREGON

HEARINGS DIVISION

In the Matter of the Compensation	)	WCB Case No. 06-03726
	)	Claim No. 7988923G
of	)	DOI: 12/9/2005
	)	WCD File No. HAS4901
	)	
DAVID B YOUNG, Claimant	)	**OPINION AND ORDER**

This matter is before the undersigned Administrative Law Judge (ALJ) pursuant to claimant's request for hearing from employer's denial letter dated May 22, 2006. Hearing convened on June 4, 2009, and was continued for further proceedings, including a Worker Requested Medical Examination (WRME), and expert deposition(s). Claimant was present, and is represented by attorney Arthur W. Stevens III. Employer Mountain View Paving and its insurer SAIF Corporation are represented by attorney Tom Harrell. Also present was employer representative Paul Meyer. Hearing was reconvened on March 15, 2011, and evidence was taken. At the conclusion of the hearing the record was held open for the deposition of Dr. Burton, and for claimant's opportunity to present rebuttal evidence relating thereto, subject to employer's right to cross-examination. The documentary record consists of Exhibits A, B, C, D, 1 through 73, and 75 through 221, including Exhibits 107A, and 204A. Exhibit 74 was withdrawn.

The record closed on September 10, 2012, the date of receipt of claimant's reply argument.

ISSUES

Issues are procedure, compensability, attorney fee and costs.

FINDINGS OF FACT

Claimant was a 30-year-old asphalt plant manager when he sought medical attention from Dr. Mullarkey on October 6, 2005, complaining of shortness of breath and frequent coughing in the morning. Claimant told Mullarkey that he had been working in a "somewhat dusty environment" for the past year. (Ex. 2-2). He also told the doctor that he smokes approximately half a pack of cigarettes per day, and had been smoking for 10 years. (*Id.*).

Dr. Mullarkey recorded the following history:

> "He states that he has difficulty at times catching his breath when he is in one of his coughing spasms. Occasionally he will cough so deeply that he will have a dry heave, and very infrequently but occasionally he will vomit. He does detect that for the past 6 to 7 months he has had some wheezes at night. * * * Sometimes he will cough for a straight ½ hour. * * * For the most part it is a dry cough, but then he will struggle with the last bit of coughing and occasionally may bring something up." (Ex. 2-1).

On exam Mullarkey recorded decreased inspiratory and expiratory effort, and noted wheezes throughout both lungs. (Ex. 2-3). She ordered a chest x-ray and pulmonary function testing. (Ex. 2-4).

A chest x-ray obtained on October 11, 2005 revealed abnormal right middle lobe and lingular opacity, suspicious for an infiltrate. (Ex. 5). A pulmonary function test obtained the same day revealed severe restrictive and probably mild obstructive ventilatory defect(s); and severe decreased diffusion capacity suggestive of a severe alveolar capillary process. (Ex. 6). A repeat chest x-ray obtained on November 14, 2005 showed bilateral infiltrates; and further evaluation by chest CT was recommended. (Ex. 12).

In December 2004 claimant had begun working at employer's asphalt production facility just off I-5 a few miles south of Medford. Six months after leaving claimant had summarized his job duties in a narrative statement, which is reproduced here in its entirety:[1]

[1] Spelling and punctuation errors are left intact.

"I started working for Mt View Paving in late December of 2004. I was hired as a plant manager, because they were planning to replace the current one. But until the old plant manager was released in January 2005, I was told to say I was the fabricator there to rebuild the new cold feed bins. These are four rusty metal bins, each of them big enough to fit a volkswagon in. These bins where bought back east, and shipped to Oregon before I even was hired. Any way these bins where old, ,rusty, and worn out, so we where going to rebuild them and up grade our plant. So, my first day there, I cut and grinded my first plate of steel. These are about 5x7 feet in length and welded it in place. While doing this work I am constantly exposed to flying meatel sparks. So much that I do not own one work shirt without hundreds of tiny little burn holes that fly at me from the grinding and welding of the hot metal. But after the guy I replaced was let go, in early January 05, I mainly ran the asphalt plant for the next few months and continued to assist cutting, grinding, our welding on the bins when needed. Then real work was started on the bins, and the company owner's friend Gary Angel was to oversee the work on the bins. To get them on line, a helper was hired named Abel and him and Gary got started. When I was not working making asphalt, I was helping them cut the old metel out and grind the frames to prep them for the new steel. Hassle fabrication in Ashland made the bottom sections and we where to make the top halves and do all the finish work. This involved more grinding and welding. By this time Gary would just tell Able what to do, and leave, so when I was done making asphalt for the morning, I would go and help him for the rest of the day. There was a lot of rusty steel to grind through, so the work was slow and long. I put my time in on those bins up until about September 2005 when we completed the metal work on the bins and went back to mostly making asphalt.

"The asphalt making plant is a whole nother matter. This machinery is extremely out dated and broken down. Now my job as asphalt plant manager is to make asphalt for what we are going to use that day and anybody else that wants to buy asphalt from us. The plant

manager is in charge of the trucks getting loaded and out to the job, but with a plant being 35 years old there are a lot of break downs. So, In between loads, a lot of repairs are made welding up dust holes, putting silicone in dust holes, tighting bolts, unclogging rock jams, making sure there's enough rock in the yard, and oil to make asphalt, or trying to stop massive dust leaks. In the winter we have to dry the rock which requires you to heat the rock up drop it into a truck and put it back into a pile . which creates a massive dust cloud that you cant even see 5 feet in front of you but these are my work duties for the past year and few months." (Ex. 89-1).

At hearing claimant agreed that the narrative statement is accurate. He testified that the grinding wheel he used was a disc that measured nine inches in diameter, and was 1/4 of an inch thick. The wheel mounts on a portable, hand-held electric motor, and the operator places the edge of the spinning wheel against the material that he is working on. (Testimony of claimant). Claimant identified the material safety data sheet (MSDS) for the grinding wheel that he had used (Ex. D), and testified that he had showed the MSDS to his doctor.

The MSDS for the grinding wheel identifies potential health hazards posed by its use, including inhalation of dust particles from the various metal oxides and other materials comprising the tool itself. (Ex. D-1, and -4). The user is also cautioned concerning exposure to the by-products of the grinding operation:

"A greater hazard, in most cases, is the exposure to the dust/fumes from the material or paint/coatings being ground. Most of the dust generated during the grinding is from the base material being ground and the potential hazard from this exposure must be evaluated." (Ex. D-4).

A lot of the grinding that claimant did was to prep the surfaces of the metal bins that he was working on, in order to remove the paint and rust. (Testimony of claimant). Sometimes he would use the grinder when repairing sections of the asphalt plant itself. (*Id.*). When grinding claimant breathed particulate matter. (*Id.*). His wife had bought some paper "cough masks;" but claimant was not always good about wearing them. (*Id.*).

The grinder motor itself weighs four or five pounds; and requires two hands to operate. (Testimony of claimant). While the bins were lying on their side claimant would work inside them with grinding and welding equipment. (*Id.*). He would sometimes lie down to get to the bottom edges the bin that he was working on. (*Id.*). Claimant might grind three to five hours in an eight-hour day – he would typically spend 15-20 minutes at a time grinding; sometimes longer if he was in a position where he could support the grinder with his body. (*Id.*).

Claimant had to cut the side plates out of the old bins, and grind off the rust; then the plates would be welded back in. (Testimony of claimant). He did more cutting and grinding than welding – he is not a great welder. (*Id.*). When welding claimant used gloves and a welding hood, but no breathing apparatus; so he did breathe welding fumes. (*Id.*). He used both wire feed and arc welders. (*Id.*). When welding claimant was usually inside the bins – he would be lying/leaning 45 degrees into the steel wall of the bins, and welding up from the bottom. (*Id.*).

Claimant frequently got fine particulate material on his clothes, face, and hair from work. (Testimony of claimant). The bottom of the shower would be brown when he got done showering after work: there was rust-colored material that came from grinding metal, and from lying on the rusty surfaces holding the grinder above his body; there was fine silt from the augur; and dust from the belt line and the rotating drum. (*Id.*). Claimant assumes that some of the particulates associated with the welding process came from the welding rods. (*Id.*).

Tammie Gilles-Young testified that her husband would come home from work covered in a rust-colored dust, as fine as baby powder – it would be stuck all over his hair and up his nostrils, and sometimes it looked like he had a nosebleed. (Testimony of Tammie Gilles-Young). She would get upset because the bar of soap would be all "icky" from tiny pieces of rust, soot, and rock after claimant had showered. (*Id.*). Mrs. Young testified that after her husband had been working at the asphalt plant for a few months he began coughing a lot in the morning. It eventually got so bad that her husband would get up early, and would go outside and vomit. (*Id.*).

Paul Meyer is a paving contractor, and owns the asphalt plant where claimant worked. (Testimony of Paul Meyer). He is a working owner-operator, and his many tasks include bidding jobs, operating a paving machine, and occasionally babysitting" the operation of the asphalt plant. (*Id.*).

Meyer described the operation of the asphalt plant, which basically entails processing rock in the "pug mill," where it is coated with oil to make asphalt. (Testimony of Paul Meyer). The process starts with the dumping of rock into large metal bins, which are 12 feet long, nine feet wide, and six feet deep. (*Id.*). There is a three-foot vertical section at the top of each bin; then the sides slope down on a 45 degree angle to the flat bottom. (*Id.*). The material flows out the bottom of the bin onto a conveyor. (*Id.*). Because the rock is wet in the winter it has to be dried before it can be processed into asphalt; and this drops the output of finished product from about 75 tons an hour to 50 tons per hour. (*Id.*). Photographs of the plant (Ex. 64-12 through -14) show areas where the main operations take place; and one photo (Ex. 64-15) shows an aerial view of the entire operation.

In the original plant, which was built in 1970, Meyer had only one bin; and he had to have a third party blend the asphalt material, which cut into his profit. (Testimony of Paul Meyer). Figuring that multiple bins would allow him to mix a greater variety of product, in 2004 employer had bought four used bins from a party in Tennessee. When the bins were delivered, however, Meyer found that they were rusty and pin-holed; and were unusable without rehabilitation. (*Id.*). He had claimant and others work on the bins for a few months, but when the work was not proceeding fast enough Meyer contracted with a third party to cut out the insides of the bins and refabricate them. (*Id.*).

Meyer admits that there is dust at the plant – if the augur plugs up the operator will have to stop the augur, and open up the augur door so that the fine silica dust can drop to the ground. (Testimony of Paul Meyer). The silica dust is typically a white, light beige, or light gray color. (*Id.*). Meyer also admitted that dumping dry rock into a truck will produce a cloud of dust, but he said that most of claimant's work day was spent in the control shack, away from significant exposure to dust. (*Id.*).

On November 20, 2005 claimant was seen in the emergency room by Dr. Howard, who recorded the following history:

> "This 30-year-old man has been ill, actually for about eight months, with intermittent shortness of breath, wheezing and cough. He did have a diagnosis of asthma and was put on inhaled bronchodilators. However, he had an x-ray in October that demonstrated pneumonia.

> * * * He is a cigarette smoker. He works as an asphalt paver. The work is dusty and smoky." (Ex. 14-1).

Howard diagnosed multi-lobe pneumonia; and ordered a CT scan, which revealed multifocal alveolar disease of each lobe of both lungs. (Ex. 15).

On December 9, 2005 Dr. Ordal saw claimant for a pulmonary consultation, and recorded the following history:

> "[Claimant] is a manager of an asphalt plant. He has been there for about the past year. He works mostly in the office. When he is out around the dust he does wear a double-banded facemask. He does feel he has had significant exposure. * * * He has smoked about one pack per day for eight years." (Ex. 27-1).

Diagnoses included chronic bilateral pulmonary infiltrates of uncertain etiology. (Ex. 27-2). Dr. Ordal ordered additional laboratory studies. (Ex. 27-4).

After undergoing allergy testing (Ex. 28), blood tests (Ex. 29), and a new chest x-ray (Ex. 32), claimant returned to Dr. Ordal on January 11, 2006. Ordal reviewed the diagnostic tests with claimant, but was unable to clearly identify the source of what he characterized as "[b]ilateral patchy pulmonary infiltrates[.]" (Ex. 33). Ordal told claimant and Mrs. Young that a video-assisted thorascopic (VAT) lung biopsy and cardiothoracic surgery would be required. (*Id.*). He then offered the following observation:

> "It is possible that this process is related to some form of work exposure although [claimant] says he does take precautions and wears a double-banded facemask at the asphalt plant and works mainly in the office." (*Id.*).

A new chest x-ray obtained on January 18, 2006 revealed findings suspicious for mediastinal lymphadenopathy and moderate interstitial lung disease. (Ex. 34). The next day claimant was admitted to Rogue Valley Medical Center (RVMC), where Dr. Folsom recorded the following history:

> "[Claimant] is a 30-year-old male. In June 2005 he developed a nonproductive cough. He states that it was very severe and at times

would be so severe he would vomit. He sought medical help for this in October 2005. At that time he was started on Azmacort, albuterol, antibiotic, and had several treatments with corticosteroids. Chest x-ray and CT scan showed bilateral pulmonary infiltrates.

"The patient denies traveling either into the desert or out of the country. He does have a number of industrial exposures. The patient is a manager at an asphalt plant and he states that he is subjected to a fair amount of dust, oil, and particulate matter. The patient notes that while he should wear a double-banded face mask and while this is provided he is not always as good as he ought to be about wearing it. The patient has a dog at home. No birds are noted. He has been living in a new home for the past several years. Unfortunately, the patient continues to smoke a pack of cigarettes a day, although he states he is cutting down." (Ex. 36).

On January 19, 2006 Dr. Folsom performed a VAT, with biopsy of the left upper[2] and lower lobes. (Ex. 37). Four days later Dr. Treger completed a surgical pathology report, diagnosing a desquamative[3] interstitial pneumonia (DIP) pattern with giant cells. (Ex. 41). Treger included the following note in his report:

"Although a DIP pattern can be associated with heavy smoking, the frequent presence of multinucleated giant cells within the intra-alveolar component in not usually found in DIP and, hence, requires consideration of other entities. The primary consideration in this case would be a giant cell interstitial pneumonia (usually a manifestation of hard metal disease) or a manifestation of an extrinsic allergic alveolitis." (*Id.*).

On January 26, 2006 Dr. Mullarkey saw claimant again, noting that he did not have wheezing or coughing during his recent hospitalization, or during the first two days following his discharge. Following his return to work on January 25,

[2] Although the narrative portion of the operative note identifies the source of the upper lobe section as the "right upper lobe" (Ex. 37-1), the surgical pathology report describes the specimens as originating in the left upper lobe and left lingual. (Ex. 41).

[3] Desquamative means, "Relating to or marked by desquamation." *Stedman's Medical Dictionary* 468 (26th ed. 1995). Desquamation means, "The shedding of the cuticle in scales or of the outer layer in any surface." *Id.*

however, claimant had awakened with a resumption of the coughing and wheezing. (Ex. 44).

Dr. Ordal saw claimant again on January 27, 2006. His chart note includes the following conclusion:

> "I had a long discussion with [claimant] and [his wife]. The biopsies demonstrate findings consistent with desquamative interstitial pneumonia but also with giant cells suggestive of hard metal disease or extrinsic allergic alveolitis. I think it is more likely than not that this is related to his exposure at work and I have strongly recommended that he no longer work at the asphalt plant. I believe that it is imperative that he avoid the smoke, dust, fumes and other pollutants to which he is exposed there." (Ex. 45).

On January 30, 2006 claimant filed a Form 801, identifying the claimed condition as "progressive cough * * * pathology revealing mass caused by exposure at work." (Ex. 48).

On February 16, 2006 claimant returned to Dr. Ordal, who wrote:

> "[Claimant] is seen back in regards to his biopsy proven desquamative interstitial pneumonia, which includes features of extrinsic allergic alveolitis suggestive of hard metal disease. * * * When seen on January 27 it was strongly recommended that he no longer work at the asphalt plant and that he avoid exposure to smoke, dust, fumes and the other pollutants to which he is exposed there. I told him at that time that I thought it was more likely than not that his lung disease was related to his exposure at work. [Claimant] states that his employer refused to accept this. [He] has actually continued to work there although he states now that he is not returning there to work. * * * His breathing is worse on days that he has been working at the asphalt plant although he does better on weekends." (Ex. 52).

On February 28, 2006 claimant gave a recorded statement. He told the SAIF investigator that he was still working five days per week, with two or three hours per week overtime. (Ex. 54-2). He runs the asphalt plant: "You dump dry rock in

one end and out the other end comes heated oil soaked asphalt." (Ex. 54-3). Claimant loads the rock hopper with a front end loader. (Ex. 54-4). He would spend two or three hours in the loader in an eight-hour day when making asphalt. (Ex. 54-5).

Claimant is responsible for controlling the amount of oil being added to the rock; and how much heat, up to 340 degrees. (*Id.*). He described the process:

> "[There is] an exhaust fan * * * that sucks dirt and steam and the heat, it pulls the heat through the drum out and it goes through a water wash, a water jet filtration and then so there's steam, there's still steam. When you open up the door we drop a ton at a time into a bed of a truck and there's steam that comes off that then the oil smoke too. There's smoke and there's dust just from the * * * rock just being * * * dried." (*Id.*).

If claimant had spent two or three hours on the loader, he would have spent the other five to six hours standing next to the asphalt plant/machine – the control shack is just five feet from the machine. (Ex. 54-6). During the winter months claimant would usually make asphalt every day, but it would not always take the whole day. (*Id.*).

In the four months of summer they make asphalt all day every day. (Ex. 54-7). When not making asphalt claimant would work on the equipment – welding, cutting, grinding and rebuilding. (*Id.*). He would have to "grind a plate of steel, stick it * * * where the rock's eaten a hole in the asphalt plant[,] and weld another plate onto it." (Ex. 54-8).

Claimant told the investigator that before he got sick he had not worn protective breathing equipment when operating the loader. (Ex. 54-8, and -9). He had brought some paper masks to work, but had not used them on a regular basis until September 2005, when he realized that he was getting sick. (Ex. 54-9). Claimant also told the investigator that he had not worn protective breathing equipment when welding or grinding – he wore only safety glasses, ear plugs and welding gloves. (*Id.*). Claimant never wore a respirator. (Ex. 54-15).

Claimant told the investigator that his symptoms came on gradually, starting in the summer of 2005 – he would wake up with a cough, usually a dry cough

often accompanied by vomiting. (Ex. 54-10). By September he was experiencing shortness of breath. (*Id.*).

Claimant told the investigator that he had to grind on large rusted metal bins, up to six hours per day, cutting steel plates out of them. (Ex. 54-14). He elaborated:

> "[T]hen you had to grind the welds down on 'em, the old welds before you put the new ones back, the new plates back in 'em. So you did a lot of torch work, a lot of grinding work." (*Id.*).

Claimant told the investigator that he had smoked for 10 years, from a half pack to a pack per day. (Ex. 54-15, and -16).

On March 9, 2006 Dr. Ordal saw claimant again. He noted that claimant was no longer working, and was feeling a bit better, "but still gets winded fairly easily." (Ex. 57). A new chest x-ray was obtained, showing no significant interval change since January 10, 2006. (*Id.*).

On April 4, 2006 Dr. Ordal saw claimant again, noting that he was about the same. (Ex. 60). Ordal told claimant that he would see him back in four weeks for follow-up chest x-ray and repeat pulmonary function testing. (*Id.*).

On April 21, 2006 Mr. Hutchinson, identified as an Industrial Hygiene Technician, wrote a memorandum to the adjuster regarding a site visit that had been conducted on March 22, 2006. The purpose of the visit was,

> "to measure air concentrations of contaminants that could have been found in the personal breathing zone of [claimant] when he worked as the Asphalt Plant Manager and Loader Operator for Mt. View Paving." (Ex. 64-1).

The report indicated that "[t]he air samples were collected during production of approximately 150 tons of asphalt mixing and subsequent truck loading." (*Id.*). According to the report air samples conducted in the areas of the plant manager and loader operator, as well as the pug mill operator, showed detectible concentrations of only two metals – manganese and iron oxide; and both of those were within OR-OSHA limits. (Ex. 64-10, and -11). Likewise, air concentrations of dust, respirable dust, and respirable crystalline silica measured at the workplace

were also found to be within OR-OSHA limits. (Ex. 64-11). No welding or abrasive grinding activity was performed on the day of the site visit. (Ex. 64-3).

On May 2, 2006 Dr. Barker performed an independent pulmonary evaluation, and reported the following findings and conclusions:

> "Overall, the bulk of evidence available to me suggests that the diagnosis of hard metal DIP is an appropriate diagnosis, and [claimant's] most recent workplace would be mainly implicated. Because of the strong association of personal cigarette smoking with DIP, I would be certain upon review with [claimant] that he has not been smoking cigarettes since the time of surgery." (Ex. 65-6).

> "At this time the evidence suggests that there are two major contributions. One is his personal cigarette smoking, and the other is an exposure at his most recent workplace, Mountain View Paving. I did review other possible workplace exposures." (Ex. 65-8).

> "I think there is a material relationship of his DIP/hard metal disease and his work at Mountain View Paving as well as his personal cigarette smoking." (*Id.*).

Dr. Barker concluded that claimant was not medically stationary, and could not yet return to work. (Ex. 65-9).

Dr. Ordal saw claimant again on May 4, 2006, noting that new pulmonary function tests had shown little change; and that a new chest x-ray still showed some infiltrates, primarily in both lung bases. (Ex. 68). Ordal told claimant that he would review Dr. Barker's IME report when it became available; and would see claimant again in four weeks. (*Id.*).

On May 22, 2006 SAIF denied compensability of an occupational disease, diagnosed as bilateral pulmonary infiltrates. (Ex. 69). Claimant timely requested a hearing.

On July 17, 2006 claimant sent his attorney a memo in response to the industrial hygiene report; and elaborated on the metal bin work:

> "These bins were in (*sic.*) unusable in the condition that they were present to me at the time of my employment. I would describe them to be 85% covered with rust with large gaping holes. * * *
>
> "The whole outer surface of the bins was covered in a thick gray paint of unknown origin.
>
> "My task was to grind with a metal grinder for large periods of time and then cut these damages areas with an oxyacetylene torch. This was in order to hopefully be able to weld in patches of steel to repair the holes. Then I was expected to replace these areas with new pieces of steel that we fabricated to fit from scratch.
>
> "While I worked on these 'rock bins' I at no time was told or offered any form of respirator or protective clothing or equipment, nor was I warned of potential safety or environmental risks." (Ex. 80-1).
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> * * * * *
>
> "At times the work would cause loud 'popping' sounds and metal splatter, that I received (*sic.*) significant burns to my skin and clothing. The smoke that was produced was at times overwhelming, and I (*sic.*) work would have to be stopped by myself because I would begin to feel as if I could not continue (ill, headachy, short of breath). It was then that I would find other tasks to do around the yard, such as driving the loader to move rock." (Ex. 80-2).
>
> * * * * *
>
> "As far as the face masks that Mr. Meyers claims to have provided for the safety of the employees, these were purchased by myself (surgical type masks) but this was not until months later when I began to have a severe cough and shortness of breath while working. This was at the request of my spouse who was beginning to worry about my possible exposure.

"I would also like to reiterate that I performed the work of grinding this metal from the day I was hired until the last day I worked for Mr. Meyers." (*Id.*).

On August 8, 2006 Dr. Barker performed another independent pulmonary evaluation, and reported the following:

"I had recommended a variety of studies as well as review of his pathology by other experts. I see no documentation that any of that has been done. Specifically, hard metal induced lung disease is unusual enough and I think a review by an expert such as Dr. Abraham in New York would be most helpful or any other expert that his personal physicians are aware. (*sic.*)

"I think this is particularly important as there are major implications regarding both the genesis and probable prognosis of his disease." (Ex. 88-3).

Barker felt that claimant might be a potential candidate for a lung transplant, but indicated that claimant would first have to lose a considerable amount of weight; and he recommended tapering of claimant's steroid medication. (*Id.*).

On August 24, 2006 Dr. Ordal saw claimant again, recording the following conclusions:

"I reiterated that I think [claimant's pulmonary disease] is likely related to his work exposure, whether due to metal exposure or simply the heavy smoke and dust to which he was exposed to, (*sic.*) When he would come in initially and see me in the office his clothes would smell very strongly of smoke and he says he had extensive exposure to burnt rock, oil and diesel as well as asphalt dust." (Ex. 90).

On November 22, 2006 Dr. Barker signed a supplemental report for the carrier. After observing a DVD of claimant smoking cigarettes in August 2006 Barker had changed his opinion:

> "After further review of descriptions of his workplace and of investigative reports of metals in the workplace as well as this video, I think the major, if not sole etiology is her personal cigarette smoking which is strongly linked to DIP. The main reason that I changed my thinking is review of the video." (Ex. 107-2).

Barker no longer believes that there is a material relationship between claimant's subject occupational activities and his DIP. (Ex. 107-3).

On November 27, 2006 Dr. Ordal signed a concurrence letter that had been prepared by claimant's attorney. He agreed that claimant has hard metal disease, caused by his employment; and that the worsening of disease since claimant had discontinued the work activity does not mitigate against a work relationship. (Ex. 114-1). Ordal indicated that claimant's exposure due to grinding and welding activities are not measurable by cement dust samples. (Ex. 114-2). He agreed that, although DIP can be due to cigarette smoking, the pathology findings in this case (including the giant cell pathology) are not consistent with a smoking-induced condition. (Ex. 114-3). Ordal endorsed the following summary:

> "In conclusion, you felt smoking to have been a contributor, but not the major contributing cause of this unique and particular disease given the pathological nuances as well as the known exposure to the grindings and the particulates you observed covering [claimant]'s clothing and skin at visits with you. You felt the nature of his illness is occupational caused by breathing in particulates, probably caused by breathing metal and particulates during the welding and grinding activities described." (*Id.*).

On February 7, 2007 Dr. Barker was deposed. He indicated that giant cells are "a fighter cell," or macrophage;[4] and they try to ingest foreign material. (Ex. 136-16). Barker testified that he does not see people with giant cells solely from smoking (Ex. 136-17); and agreed that "[g]iant cells are not part of the usual DIP." (Ex. 136-26). Barker also agreed that welding is a type of work that can cause respiratory disease, due to the risk of breathing particulates. (Ex. 136-21). He

[4] *Stedman's Medical Dictionary* (26th ed.) defines macrophage as: "Any mononuclear, actively phagocytic cell arising from monocytic stem cells in the bone marrow * * * [that, *inter alia*,] are involved in both the production of antibodies and in cell-mediated immune responses, participate in presenting antigens to lymphocytes, and secrete a variety of immunoregulatory molecules."

considers both claimant's work exposure and his smoking as contributing causes of his hard metal disease. (Ex. 136-29). Barker clarified:

> "Almost everything (*sic.*) that gets desquamative interstitial pneumonia, DIP, is a cigarette smoker. Whether it is a cause or a major contributing factor we don't know. There is clearly a very strong association." (Ex. 136-30).

When asked about the significance of the large cell and hard metal component elements of the instant case that distinguishes it from a "typical DIP situation," Dr. Barker responded as follows:

> "Well, the thing to distinguish it are (*sic.*) – first off, that he had been around dust, a variety of dust and fumes, that contained minerals; and that always raises a flag, number one. And second, it was the giant cells, which are a little bit unusual in DIP." (Ex. 136-32).

Barker testified that in claimant's case it was the giant cells that raised the suspicion of hard metal disease. (Ex. 136-33). The fact that claimant had continued to smoke was significant to Barker, and helped to explain why he continued to get worse, even after he had separated from the ostensibly offending work environment (inhalation of hard metal). (Ex. 136-34).

When asked whether claimant's smoking might simply have caused claimant to remain symptomatic with hard metal DIP, in contrast to having caused the DIP, Dr. Barker stated, "we don't know what causes DIP." (Ex. 136-36). When pressed further, Dr. Barker stated:

> "If this were hard metal disease, which sometimes can be difficult to distinguish from DIP, stopping the exposure to the hard metal, cobalt we're talking about, should slow down if not stop the disease. When I saw him the second time, as I said the only factors that I assume everybody would agree that he got rid of was the hard metal. And he progressed. So that was part of the reasoning that I used the second time." (Ex. 136-37).

Dr. Barker testified that once exposure to the hard metal is discontinued "ongoing progression and deterioration does not usually occur." (Ex. 136-38). Barker conceded, however, that he has not had much experience with hard metal disease, which is why he had recommended review of the case by someone with more experience. (*Id.*).

After reviewing a videotape showing that claimant was still smoking, Barker now believes that he had (smoking-related) DIP, but not hard metal disease. (Ex. 136-41). Barker does not, however, contend that claimant's smoking caused the giant cells. (Ex. 136-44). According to Barker, although the medical community does not know the cause of DIP, given the strong correlation between DIP sufferers and smokers it is likely that "personal cigarette smoking plays a role in the development of DIP." (Ex. 136-49).

Dr. Barker felt that the worsening of claimant's symptoms between May and August 2006 is consistent with worsening of the pathology. (Ex. 136-50). Even though a second lung biopsy was not done Barker is confident that claimant's pathology (DIP) did worsen. (Ex. 136-51). He did not, however, have an opinion as to whether the giant cells had also worsened. (*Id.*).

Dr. Barker does not believe that claimant's work exposure is the major contributing cause of his lung condition; but neither does he believe that the cigarette smoking was a cause of the giant cell pathology. (Ex. 136-54).

On February 14, 2007 Dr. Ordal wrote to claimant's attorney:

> "As you know, [claimant] is under my care. I believe that [claimant] has occupational lung disease. Lung biopsies done in January 2006 demonstrated desquamative interstitial pneumonia and multinucleated giant cells, suggestive of giant cell interstitial pneumonia, which is consistent with hard metal disease or extrinsic allergic alveolitis. Although [claimant] has smoked some, his cumulative smoking exposure is about eight pack-years and I do not think that that is the primary cause of his desquamative interstitial pneumonia or the giant cell interstitial pneumonia. I do not think that his initial worsening after leaving his place of employment argues against occupational exposure as the etiology and, in fact, it should be noted that his pulmonary function testing

did improve during the latter part of 2006. Certainly, the findings of giant cell interstitial pneumonia, which are consistent with hard metal disease, could only have been sustained from his work environment based upon the history I obtained from [claimant]. I should emphasize, however, that in addition to the metal dust exposure, [claimant] was exposed to other forms of smoke, dust, fumes and other pollutants at work, which could certainly be playing a role in his lung disease. I was impressed when he would come in to see me while still working at the plant that he reeked of industrial smoke and his clothing would be covered in material, which certainly was not tobacco smoke. I cannot say with precision how much of this exposure was related to cement versus ground rock versus metal and I frankly do not think the distinction is significant. I had stressed to him several times during the first part of 2006, after the lung biopsy, that he had to get away from this exposure and fortunately he subsequently did. (Ex. 137-1).

"As discussed above, I think the findings in his lungs are related to an acquired disease from his exposure at work and frankly do not consider smoking one pack a day of cigarettes for eight years as a heavy smoking history. I do not think his smoking has played a significant role in his lung condition. Although desquamative interstitial pneumonia is seen in heavy smokers, certainly the multinucleated giant cells are not associated with tobacco use. As we discussed, this is not a simple case of desquamative interstitial pneumonia and if smoking has been a contributing factor to his illness I believe it to be a very minor contributing factor at worst and that by far the major contributing factor to his lung disease is [claimant]'s exposure at work to not only metal dust but also other particulates, smoke, and fumes." (Ex. 137-2).

Claimant's attorney wrote to Ordal, asking him to explain why he did not believe that claimant's worsening condition following separation from the work environment suggested that there must be some other cause. (Exs. 142, 153).

In a June 7, 2007 letter to claimant's attorney Dr. Ordal wrote:

"What likely happened is that the inhalation of toxins into [claimant's] lungs set off a chain of events leading to the inflammation and damage to his lungs. This inflammatory process, of course, would not immediately stop once he was removed from exposure, thus leading to initial worsening after removal from the exposure. * * * For instance, a patient who develops lung cancer from cigarette smoking will have progression of the lung cancer if untreated, even after that person stops smoking." (Ex. 156-1).

Ordal explained that his opinion is not contingent upon a finding of hard metal in claimant's lung tissue:

"In regards to the chemical analysis being done on [claimant]'s lung tissue, I do not think that the issue of causality turns on whether or not hard metal is found in his lung tissue. If it is not hard metal, it is likely products of combustion or other toxins in the work environment which have caused his lung injury, and I do not think that the absence of hard metal in his lung tissue would disprove his work exposure being the cause of his lung condition." (Ex. 156-2).

On June 16, 2007 Dr. Beckett wrote to claimant's attorney, indicating that he agrees with Dr. Ordal's June 7, 2007 letter. Beckett elaborated:

"Specifically, it is common with many occupational dust diseases of the lung for the disease to progress after exposure has ceased. This can occur with hard metal lung disease, silicosis, asbestosis, chronic beryllium disease, and others." (Ex. 157).

On November 13, 2007 Dr. Abraham, who had examined the tissue samples from claimant's lung biopsy, wrote to Beckett and claimant. After describing the testing protocol, Abraham noted that,

"[b]ackground values in the general population * * * usually range up to 10 or 20 million total particles per cubic centimeter of tissue, the majority of which are aluminum silicates." (Ex. 163-1).

He then described the test results taken from claimant's lung biopsy:

"The analysis of the lung tissue sectioned from block 3A showed a total concentration of 24 million particles per cubic centimeter of tissue. The types of particles found included 2,000,000 silica, 1,000,000 aluminum silicates, .6,000,000 (*sic.*)[5] miscellaneous silicates and 20,000,000 metal particles. The types of metal particles detected included (in decreasing frequency) barium, titanium, iron, tin, nickel, chromium, lead, manganese and bismuth. These confirm exposure to welding or some metal working environments including particles consistent with stainless steel, paint materials, etc.

"The second tissue analysis used tissue from block 3D. This tissue revealed a total concentration of 40,000,000 particles per cubic centimeter of tissue. In this analysis the aluminum silicate particles measured 27,000,000 particles per cubic centimeter of tissue, talc was 1,000,000, gypsum was 5,000,000 and metal particles were 7,000,000. In this analysis the types of metals found included titanium followed by iron and barium. (*Id.*).

"It is not uncommon for there to be a variation in particle concentration ion the lung tissues from different sites. According to the surgical pathology report (RS06-781), the blocks labeled 3 came from the left upper lobe of the lung.

"The types of particles detected in [claimant]'s lung tissue can be compared with what is known about exposures he had at work. Considerable literature indicates that interstitial lung disease such as evident on [claimant's] biopsies can be produced by inhalation of various particulate materials. It is also part of standard [references omitted] criteria for diagnosis that such exposures must be excluded before considering an individual's interstitial lung disease to be 'idiopathic' pulmonary fibrosis. The pathology tissue samples confirm interstitial lung disease and the analysis of the tissue confirms [claimant]'s exposures to various inorganic

[5] It is assumed that the decimal before the numeral 6 is a typographic error or similar artifact, due both to the fact that less than one particle would not be considered significant here, and to the fact that there would be no need for the zeros if the author intended the decimal.

particulates, which represent a sampling of his exposures. These allow me to conclude to a reasonable degree of medical certainty that his dust exposures were a substantial contributing factor in the development of his lung disease." (Ex. 163-2). (Emphasis in original).

On December 20, 2007 Dr. Ordal wrote to claimant's attorney:

"In my opinion, Dr. Abraham's report confirms that [claimant]'s work exposure is the major, (greater than 51%), if not the only cause of his interstitial lung disease. The types of metal particles found in [claimant]'s lung correlate well with [his] work exposure history and the findings on his lung biopsies, as reported by Dr. Treger, who reported changes consistent with hard metal disease." (Ex. 170).

On April 21, 2008 Dr. Abraham was deposed. He testified that the general population acquires silicates, including aluminum silicate particles from dust, along with small amounts of iron oxide and titanium. (Ex. 176-9).

Abraham had received two of the four or five lung biopsy samples, which he indicated is standard protocol. (Ex. 176-12). Both samples came from the (left) upper lobe. (Ex. 176-13). Abraham testified that biopsy specimens will vary due to variations in concentration; and he considers the differential from 24 to 40 million parts per cubic centimeter to be relatively minor. (Ex. 176-14).

In reaching his conclusions Abraham relied upon the history provided by his friend and colleague Dr. Beckett, included in a March 8, 2007 letter. (Ex. 176-15). He also relied upon medical literature associating interstitial lung disease and inhalation of various particulate materials. (Ex. 176-17).

According to Abraham, "interstitial lung disease" describes the location of the pathology – *i.e.*, not in the airway itself (as in pneumonia), but in the structural supporting framework of the lung known as the interstitium.[6] (Ex. 176-18). In pulmonology the term interstitial refers to something "in between the air sacs, the

[6] The court reporter actually uses the word "interstition," which is not found in the medical dictionary. It is presumed that the witness used the word "interstitium," which means "[a] small area, space, or gap in the substance of an organ or tissue." *Stedman's Medical Dictionary* 884 (26th ed. 1995).

air spaces * * * [n]ot the air spaces, but it's the walls of the air spaces, the supporting framework of the lungs." (Ex. 176-42).

Abraham explained that interstitial non-cancer lung diseases include inflammatory types, which do not involve significant scarring, and fibrotic types, where fibrosis (or scarring) is the primary process. (Ex. 176-18). He testified that claimant has interstitial fibrosis, which means that there is scarring in his lungs, along with an accumulation of dust. (Ex. 176-19).

From what he saw in the biopsy here Dr. Abraham would not have diagnosed any of the three differentials proposed by Dr. Ordal – namely, DIP, hard metal lung disease, or extrinsic allergic alveolitis. (Ex. 176-22).

Abraham agrees that the industrial hygiene report makes no reference to tungsten carbide, or cobalt (Ex. 176-27); but testified that since cobalt is very soluble, and does not remain in the lung, it is rarely found in a biopsy. (*Id.*). Abraham indicated that cobalt is thought to be the main cause of hard metal disease (Ex. 176-28); and he agreed that no cobalt was found in the samples that he reviewed here. (*Id.*).

Abraham testified that a giant cell is,

> "a large cell, often one that has many nuclei instead of just one, so it's a large cell that can form from the fusion or sticking together or joining together of several individual cells such as macrophages." (*Id.*).

Dr. Abraham did not make a diagnosis of giant cell interstitial pneumonia here. (Ex. 176-29). He testified that the exact mechanism involved in the production of giant cells is poorly understood, but indicated that "[s]omething about the exposure to cobalt with or without the tungsten carbide seems to cause those cells to form." (*Id.*). Abraham agreed that there is probably a genetic component in development of giant cell disease. (Ex. 176-30).

The history that Abraham received from Dr. Beckett was not detailed – it merely indicated that claimant had performed manual welding on old steel bins, and had done grinding and welding of steel, along with oxyacetylene cutting of steel bins. (Ex. 176-31). He testified that "[s]ome people could have severe

reactions to a few months of exposure like that, some people might not be bothered by it." (*Id.*).

Asked whether claimant had a genetic predisposition to lung disease, Dr. Abraham answered as follows:

> "It's impossible to answer on an individual person's case unless you knew more about the genetics of the susceptibility. So I can't say that his susceptibility was more than somebody else's or less. All I can say is that he had some lung disease, and there is evidence of those exposures capable of causing lung injury documented in his lungs." (Ex. 176-32).

Dr. Abraham would not classify claimant's disease as hard metal lung disease, because he did not find "that particular exposure." (*Id.*). He testified, however, that even 32 days of exposure to the grinding of metals could have caused claimant's disease. (Ex. 176-33). Abraham was pressed by employer's attorney:

> Q [Mr. Harrell] "But given the genetic component at play, can you say the work exposure, if it's limited to 32 days, is the major cause of the condition?
>
> A [Dr. Abraham] "Sure. If he didn't have that exposure, he wouldn't have that condition." (Ex. 176-34).

According to Dr. Abraham, the work exposure causes the condition "[b]y causing a reaction in the lungs which leads to inflammation and fibrosis." (*Id.*). He explained:

> "[Claimant's] exposure to a variety of metals and other materials that we may not be able to detect such as gases that are involved in welding that are capable of producing fibrosis are the likely cause." (Ex. 176-39).

Dr. Abraham was not able to identify other causative factors, but added:

"I wouldn't call it idiopathic because there is proof looking at his lungs that he had exposures to agents capable of causing lung injury and his history is also part of that." (*Id.*).

When challenged with his lack of a detailed history, Dr. Abraham responded: "I have a very limited history, that is sufficient for me to say he had exposure to dust and fumes from grinding and welding a variety of metals." (*Id.*).

When asked about claimant's eight-year smoking history, Dr. Abraham answered, "That can contribute to lung disease of a certain type, [but] it doesn't cause fibrosis." (*Id.*). He elaborated:

"Lung disease, the smoking causes emphysema, which is the opposite of fibrosis, which is the destruction of lung rather than scarring. And, in fact, one of the papers that I published a year or two ago – Dr. Nasr * * * is the first author of that – actually looked at that issue of whether the scarring in the lungs of smokers was related to their smoking history or the dust we could demonstrate in the lungs, and it shows that the actual fibrosis correlated better with the amount of dust we could detect by analysis of the lungs than it did with their smoking history." (Ex. 176-40).

Asked whether he saw giant cells here, such as Dr. Treger described in his January 23, 2006 report, Abraham responded:

"I didn't see enough of the giant cells or the really peculiar giant cells that I have seen in hard metal disease or giant cell interstitial pneumonia. And I've probably seen more of those cases than most anybody." (Ex. 176-44, and -45).

Asked whether claimant's lung disease is acquired or congenital, Dr. Abraham responded:

"I don't think it's a genetic disease. I think it's a disease acquired by lung injury. Evidence of lung injury comes from his history and from the findings documenting some of those exposures by looking at his lungs the way we did." (Ex. 176-45).

There was evidence of metal in claimant's lungs:

> "[T]he predominant and most unusual type of particulate material found in his lung was the metal and you can see there is a list of, I don't know how many, six or eight different metal elements that were found which support the fact that he's had exposure to sources containing those metals." (Ex. 176-46).

The level of metal in claimant's lungs was greater than would be expected in the general population. (*Id.*). Abraham did not find in significant quantities of silicon material in claimant's tissues. (Ex. 176-47).

Dr. Abraham explained the risks involved in welding activities:

> "The toxic gases that are generated in welding or torch cutting or something like that involve ozone or nitrogen oxide, those aren't retained, they can cause injury but are no longer demonstrable in the lungs. They're just gases and they do their thing and are gone.
>
> "But in welding sometimes the metal fumes are actual particles that are generated in welding and that is sometimes a source of some of the oxyacetylene. If someone goes (*sic.*) steel welding, for example, you can see particles that contain iron together with chrome or iron together with chrome and nickel that can even match the composition of different types of steel that someone might be grinding or welding." (Ex. 176-47, and -48).

Abraham was asked whether he could identify specific metals from the biopsied material:

> "I can say from what we found that some of his exposure came from material consistent with stainless steel because we found single metal particles that contained iron together with chromium, nickel, and manganese, or iron together with titanium and tin that could have come from a metal or solder kind of material." (Ex. 176-48).

Dr. Abraham testified that these findings are consistent with the grinding of metal, as well as with welding or soldering:

> "Because when something is ground sometimes that activity releases very small particles that could be inhaled that represent the welding seam that is ground off to polish it or just the metal itself, and I can't distinguish between those two from looking at the particles in the lungs." (Ex. 176-49).

Dr. Abraham indicated that cigarette smoking is not a source of the metals found in claimant's lung biopsy. (*Id.*). He explained:

> "Finding these various metal particles in the lungs means somebody inhaled them. And it didn't come from smoking and it didn't come from the ambient air, it came from some operation that resulted in release of those particular individual and combinations of metals into the air that the person breathes." (Ex. 176-50).

Abraham felt that claimant's description of his work (Ex. 89-1) is consistent with exposure to the material found in his lungs. (Ex. 176-52). He testified that if claimant had done welding or grinding on prior jobs that could have been a source of some of the matter found in his lungs; but he indicated that there is usually a temporal relationship between an offending exposure and the onset of symptoms. (Ex. 176-53).

Dr. Abraham believes that claimant's work exposure is the major contributing cause of the (interstitial fibrosis) condition diagnosed in his November 13, 2007 report.[7] (Ex. 176-55). He testified that Dr. Ordal "says it really well," in describing claimant's condition as "severe restrictive disease," (Ex. 176-56). Abraham elaborated:

> "That means his lung is very stiff, and that is a sign of scarring or fibrosis. Then he goes on to say, this is Dr. Ordal's words, he says: 'It's likely related to work exposure, whether due to metal exposure or simply the heavy smoking (*sic.*) dust to which he was exposed." (Ex. 176-57).

[7] This is a reference to Dr. Abraham's letter to Dr. Beckett. (Ex. 163).

Abraham explained that he would not characterize claimant's condition as hard metal disease because he did not have evidence of cobalt and/or tungsten carbide in his lung tissues. (Ex. 176-58). He agreed that claimant's occupational exposure, as described both by claimant himself and by Dr. Beckett, is consistent with the biopsy findings. (*Id.*). Abraham feels that the choice in this case is between idiopathic and occupational cause(s); and he does not believe that the idiopathic choice fits here because of the known occupational exposures that claimant had. (Ex. 176-59, and -60). He agrees with Dr. Ordal that claimant's occupational exposure is the major contributing cause of his lung disease. (Ex. 176-61).

Dr. Abraham reviewed each of the 27 slides that were sent to him; and he selected two of them (3A and 3D) to slice and examine with an electron microscope. (Ex. 176-62). He believes, based upon the history that he has received, that all of the findings from the biopsy tissues that he examined were from the occupational exposure from December 2004 forward, although it is possible that some of the matter could have come from other sources. (Ex. 176-64).

When asked about whether he had a specific diagnosis, Dr. Abraham stated:

> "[G]iven the information of [claimant's] exposure and the findings, I can say that his exposures to metals and other stuff, fumes, contributed to that lung injury that ended up with fibrosis." (Ex. 176-65).

When pressed to explain whether it was the metals or the dust that caused the condition, Dr. Abraham elaborated:

> "It's not one or the other, it's the totality. Metals are dust. What I'm looking at are dust particles of metals and nonmetals. The predominant unusual finding in his lung was the metal." (*Id.*).

Dr. Abraham felt that since the welding and grinding activities were not being performed on the day of the industrial hygiene visit, the environment was not representative of some of claimant's work, and the report is therefore irrelevant. (Ex. 176-68, and -74). He also pointed out that NIOSH or OSHA standards do not

mean that there is no (harmful) exposure: "The regulatory levels don't prevent disease, they're just designed to reduce them below a certain level." (Ex. 176-70).

When pressed once again by employer's attorney to explain the basis of his opinion, Dr. Abraham patiently reiterated:

> "I'm saying what is found in his lungs is evidence that he has exposures to certain things that are capable of causing lung injury, and they're consistent with the history I've been provided." (Ex. 176-71).

When counsel tried to interject, Dr. Abraham continued,

> "Can I finish, please. If he has some other history of hobbies, someone should tell me and I would consider that. If it says in the records here, lead me to it." (Ex. 176-71, and -72).

If claimant had performed some welding and grinding in his earlier jobs, Abraham would agree that all of them together were probably the major contributing cause of the disease; and that the latest exposure had contributed. (Ex. 176-75).

In his January 24, 2009 response to a letter from employer's attorney, Dr. Lewis indicated that he had reviewed lung biopsy slides with fellow pathologists, and agrees with Dr. Abraham that giant cells seen here are incidental findings, and do not support a diagnosis of hard metal lung disease. (Ex. 189-1). Lewis believes that claimant's condition represents the integration of at least two separate conditions or disease processes, including DIP, and some associated fibrotic changes in the lungs, diagnosed by Abraham as pulmonary fibrosis. (*Id.*).

Lewis attributes claimant's DIP to his cigarette smoking, and not to his occupational exposure. (*Id.*). He also disagrees with Abraham's assessment that the fibrotic changes in claimant's lungs are unrelated to his smoking. Although he believes that some of the fibrosis is due to silicates to which claimant was exposed at work, Lewis feels that some of it is due to the fact that claimant's DIP went undetected for so long. (*Id.*). Lewis also felt that genetics was a likely contributing and predisposing factor, but he was not willing to express an opinion regarding the major contributing cause of claimant's pulmonary condition. (Ex. 189-2).

On March 10, 2009 Dr. Burton performed a records review. He believes that claimant has a confirmed diagnosis of DIP, which is associated with cigarette smoking, but not with occupational exposures. (Ex. 193-18). Burton explained:

> "[Claimant] does not have clinical or pathological findings that support diagnoses of extrinsic allergic alveolitis, hard metal lung disease, or pulmonary fibrosis. Prior to confirmation of DIP by pathological examination from a lung biopsy sample, extrinsic allergic alveolitis, hard metal lung disease, and pulmonary fibrosis were diagnoses to consider in the formulation of a differential diagnosis." (*Id.*).
>
> "I relied upon the interpretation of the microscopic evaluation described by both pathologists, Dr. Abraham and Dr. Treger. The interpretation of the pathological findings yields a diagnosis of DIP." (Ex. 193-19).
>
> "The only exposure known to be associated with the development of DIP is to cigarette smoke. There are not medical data that support a conclusion that work exposures resulted in the development of DIP." (*Id.*).

On April 29, 2009 Dr. Burton performed an IME. He provided a detailed description of the work environment, and an overview of operations at employer's worksite. (Ex. 194-3). Burton described claimant's normal daily work activities, as follows:

> "At approximately 0730 hours * * * [claimant] began operation of a three-yard loader and began scooping up rock and dumping it into the receiving bin. After making approximately five trips to fill the bin, he would then stand next to the drum as the asphalt was produced. Within 15-20 minutes, it became necessary for him to resume moving crushed rock into the bin. Depending upon the asphalt orders for the day, [claimant] could spend the remainder of the day operating the loader and have no time for any other task." (Ex. 194-4).

Burton included the following specific history:

"[Claimant] cited a particular project that he feels consumed an average of approximately 20 hours per week during December 2004 through March 2005. This project involved cutting and then welding metal panels on interconnected bins in the shape of cubes measuring 15 [feet] x 15 [feet] x 15 [feet], with an open top. This project typically involved cutting out a 5 [feet] x 5 [feet] steel panel with an oxyacetylene torch. [Claimant] states that he did this cutting while standing inside the bin. He specifically recalls 'lots of rust,' which tended to pop when he applied the cutting torch. Otherwise, he proceeded without noticing any adverse symptoms and was unconcerned about performing this task. [Claimant] reports that he did not utilize any respiratory protection while working on the bins." (*Id.*).

Dr. Burton recorded claimant's smoking history as follows:

"[Claimant] reports that he began smoking cigarettes at the age of 20. He smoked approximately one-half to one pack per day until December 2005. At that time, he tried to quit but found it difficult, but finally quit smoking during March 2006, when he smoked his last cigarette." (Ex. 194-6).

Burton diagnosed DIP, which he attributed to claimant's smoking. (Ex. 194-7). He explained the reasons for his conclusion:

"[Claimant]'s reported history indicates that he engages in activities at the paving company in the outdoor air that has been assessed for potential dust exposure. There are no indicators that he was exposed to excessive respirable dust components while performing his duties that involve loading and unloading rock or operating the machinery at the asphalt plant. The welding activities described by [claimant] are characteristic tasks utilizing mild steel and welding rod components. These exposures did not result in any noticeable symptoms or the part of [claimant] and did not prompt medical evaluation or treatment, nor did they produce any acute symptoms. Accordingly, the work history and industrial hygiene data do not

document evidence of any exposures of sufficient intensity or duration to result in any diagnosable occupational respiratory condition." (Ex. 194-8).

On July 23, 2009 Dr. Keppel performed a worker-requested medical examination (WRME), and diagnosed interstitial lung disease with DIP and giant cells. (Ex. 204). On August 18, 2009 Keppel performed a record review, and reported the following:

> "Extensive records were reviewed and will only be summarized as there is excellent summary on March 10, 2009 by Dr. Brent Burton, as well as earlier clinical reviews by Dr. Alan Barker and ongoing clinical care by Dr. John Ordal." (Ex. 205-1).

On August 25, 2009 Keppel completed his WRME report, offering the following conclusions:

> "In summary, I believe that [claimant] is an unfortunate gentleman of only 34 years with rather advanced lung disease with the potential for both bariatric surgery secondary to his use of steroids and potential lung transplant. In medical literature, this is unusual at such a young age and I think represents situations that would not be frequently seen. The first issue is that his workplace at Mountain View Paving had multiple potential exposures, which as pointed out by several observers were outside rather than in an inside closed space. He was manufacturing asphalt and described to me in some detail the process of heating the rock as well as his having to crawl under the machine, which had several baffles that had to be opened with a lot of dust and irritants. In general, the exposures he described, however, were dusty. At his visits to doctors, he was noted to be covered with dust and irritants; I would expect those to be of a size, with cement and rock larger than what frequently penetrates the lung; in silicosis less than 1 micron particles are much more of a concern than the larger particles. He had exposures to combustion and other fumes involved with the process. However, it seems to me the significant exposure and the one that we should focus on is the period of time he was sanding with abrasive containing cobalt. This was in a more sheltered space in

that there were large rock containers in which he would be down in corners in order to prepare the site for welding. He describes no protection in those spaces as well as being fairly closed in. He also for periods of time, when the other asphalt was less busy, he was doing this for many days of exposure. Industrial hygienists who went to the site did sampling that did show some metals, but I do not think re-created his particular activity with the exposure to cobalt and hard metal as described above. I believe that the rapid onset is unusual, although I recall a situation in Portland when Mount St. Helens erupted. We reviewed the silica data and acute silicosis was possible. I was aware of one case in which sand blasting for less than six months created significant acute silicosis with pulmonary disability. Nevertheless, although it is unusual over the one year period of time that his exposure would result in this abnormality; I do think it is medically feasible. In addition, the exposure is documented by Dr. Abraham in terms of both metals and particularly higher than his normal background for the amount of silica in the specimens." (Ex. 206-1, and -2).

* * * * *

"Given all of these factors, I believe that the preponderance of greater than 51% of the evidence suggests to me that this is indeed a case of hard metal disease that has progressed. I note that there is a report of hard metal disease in which a lung transplant was done and a similar process recurred in the transplanted lung despite the worker having no further exposure to cobalt. This suggests a process or sensitization that is ongoing as seen in [claimant]'s case where he has severe restrictive disease with a forced vital capacity of 32% of predicted with the potential that he may need a lung transplant. I have explained my thought process and the reason for the conclusions. If you have further questions or concerns, I will be available for discussion." (Ex. 206-3).

On October 20, 2009 Dr. Keppel signed a supplemental WRME report. He summarized his opinion and conclusions as follows:

"It is my impression after reviewing all the pathologic data, including second bronchoscopy with bronchoalveolar lavage, that

> [claimant] had giant-cell interstitial pneumonia with multinucleated giant cells consistent with inhalation of hard metals, specifically cobalt. This pathology can occasionally be seen similar to desquamative interstitial pneumonia. Subsequently, however, I believe finding giant cells again in the lavage after his biopsy reiterates that this is hard metal – a result of his work history, specifically grinding with materials that are known to have hard metal or cobalt. His lack of improvement after stopping smoking would also help support the fact that desquamative interstitial pneumonia was unlikely. Pathophysiology (*sic.*), it is felt that the giant cells are phagocytes that have ingested histiocytes as part of the process and is fairly distinctly related to inhalation disease from cobalt." (Ex. 209-1).

Dr. Keppel does not believe that claimant's smoking was a preexisting condition for DIP. (*Id.*).

On May 12, 2011 Dr. Burton was deposed. He testified that the presence of macrophages is one of the diagnostic criteria for DIP. (Ex. 220-10). According to Burton, giant cells are composed of multiple macrophages, and "are basically some of the macrophages that have eaten each other." (*Id.*). Burton agreed that hard metal disease may have features of giant cell macrophages (Ex. 220-11); but he did not make that diagnosis here:

> "I don't think there's really any dispute about what the pathologist sees in the [lung biopsy] slide,[8] it's really the interpretation of that. And the interpretation that is consistent is that there are numerous macrophages that line the alveolar spaces. That's diagnostic of DIP.
>
> "Now, the classification of DIP is not perfect so we expect that seeing a few giant cells doesn't really change that diagnosis, but it does enter into consideration other forms of pneumonia. So it should be considered, but there's nothing in the record that indicates that that consideration should be taken beyond DIP to some other diagnosis." (Ex. 220-13).

[8] The reference is to the January 23, 2006 surgical pathology report of Dr. Treger. (Ex. 41).

When asked whether claimant's lung condition would be associated with an occupational exposure, Dr. Burton responded as follows:

> "No. Since there's nothing in the record to indicate that he had an occupational exposure to cobalt, tungsten or any other material capable of causing the kind of pulmonary disease that he has, that connection cannot be made.
>
> "But if we look at the alternative * * * the only connection that can be drawn from an issue of association or a potential causality would be his cigarette smoking, because it's been clearly and repeatedly demonstrated that cigarette smokers in, by far and away, the majority of DIP cases are those that develop DIP." (Ex. 220-21).

Burton assumed that claimant was welding in the open air (Ex. 220-36, and -37); and he considered this assumption to be a "major factor" in his causation analysis. (Ex. 220-40). He agreed that DIP is not a condition that typically results in significant fibrosis. (Ex. 220-47). Burton cannot state that claimant's cigarette smoking caused DIP, only that smoking is "the only factor that we know of that is connected by association to DIP." (Ex. 220-49). According to Burton, "DIP is considered an idiopathic lung disease with a strong association to cigarette smoking." (Ex. 220-52).

When asked to explain where the metals in claimant's lungs came from, as described by Dr. Abraham, Burton responded as follows:

> "The test Dr. Abraham describes is that he basically takes a piece of lung and dissolves it and tests it for metal concentrations, and he finds – basically what he finds can be summarized into people (*sic.*) who work in an office have less particulate in their lung than somebody who is a cigarette smoker and/or is engaged in an occupation that is more likely to expose one to particulates than the indoor air.
>
> "Beyond that, it's not possible to draw any conclusions because simply measuring the amount of particulate in the lung is not something that we can rely upon to establish either the presence of a disease or its causation." (Ex. 220-54).

According to Dr. Burton cigarettes typically include many metallic particulates, probably including most or all of the following: chromium, nickel, manganese, iron, titanium and tin (Ex. 220-55), which may derive from the soil where the tobacco was grown. (Ex. 220-56). He testified that the lungs of smokers are anthracotic[9] due to all the particulates that accumulate there; and said that "among those particulates are metals, because they tend to be more persistent in the lung tissue than a lot of other substances." (*Id.*).

Dr. Burton agreed that a pathologist (including Dr. Abraham) would "have greater expertise in evaluating a section of lung tissue than probably most of us." (Ex. 220-58). Burton himself specializes in occupational medicine, medical toxicology and emergency medicine, and holds board certifications in those disciplines. (Ex. 220-6).

Burton testified that hard metal lung disease is a very rare disorder; and that workers around here are not exposed to concentrations of cobalt or tungsten that would lead to this disease. (Ex. 220-59). He has, however, seen numerous cases of fibrotic lung disease as a result of exposure to substances such as asbestos and silica. (*Id.*).

On cross-examination Dr. Burton was pressed to admit that his diagnosis of DIP rendered the question of occupational exposure moot, since he does not associate DIP with occupational exposure. He answered as follows:

> "[T]he process in evaluating a patient or worker in any case is always to evaluate exposure. And I might add, since nobody else has, that the only examiner here, other than myself, that attempted to characterize the exposure was really Dr. Barker. And if we take Dr. Barker's history from [claimant] and the history that [claimant] gave to me and also how it was elaborated at hearing, nonetheless, that exposure is not a significant exposure that one would be concerned about developing any medical condition, let alone a pulmonary condition.

[9] According to *Stedman's* anthracosis means: "Pneumonoconiosis from accumulation of carbon from inhaled smoke or coal dust in the lungs."

"I see welders day in and day out welding not only mild steel but stainless and sometimes exotic metals utilizing typical protective practices and they do not develop pulmonary disease as a result of that exposure.

"The one caveat is working in a closed-space environment where some respiratory protection may be required, but that's a highly monitored situation. Ordinarily welders do not have to use protective equipment to weld unless it's an exotic metal." (Ex. 220-67, and -68).

Dr. Burton noted that welders as a group are not known to develop DIP or other forms of interstitial pneumonia; nor do they typically develop fibrotic lung disease. (Ex. 220-69). After considering claimant's work exposure, Burton concluded that it did not place claimant at risk of developing pulmonary disease. (Ex. 220-70).

On May 16, 2012 Dr. Ordal responded to a letter from claimant's attorney. He indicated that claimant's biopsy reveals exposure to metals; and believes that the exposure was likely occupational, due to the fact that significant quantities of these metals are not found in the ambient Rogue Valley atmosphere. (Ex. 221-2). Ordal agreed that claimant had presented to his office with industrial dust on his face and clothing, and that the presence of industrial dust on his face suggests exposure to respirable particulates. (*Id.*).

Dr. Ordal believes that the finding of low levels of respirable dust or respirable particulates reported in the industrial hygiene report is inconsistent with the quantity of particulate material he observed on claimant, and notes that the report fails to accurately represent claimant's actual exposure, due to the fact that the welding activities that claimant performed were not tested. (Ex. 221-3). He agreed that claimant's disease likely arose out of activities to which he was not ordinarily subjected or exposed other than during a period of work activity, rather than from his smoking, or from activities of daily living in the Rogue Valley. (*Id.*).

Dr. Ordal indicated that, although a long period of smoking could be associated with simple DIP, it would not cause "giant cell interstitial pneumonia," such as claimant had; and would not have produced metal particulates and giant

cells. (Ex. 221-3). In any event, because he does not have simple DIP, Ordal concludes that claimant's disease was not caused by smoking. (*Id.*).

Ordal believes that the major contributing cause of claimant's lung disease is his industrial exposure, probably the last exposure – *i.e.*, the job at injury here. (Ex. 221-3). He believes that claimant's disease is exclusively an acquired disease from external exposure, and is not idiopathic (*i.e.*, one with no available medical explanation). (Ex. 221-4). Ordal cited an article from the *American Journal of Surgical Pathology*, to the effect that a finding of giant-cell interstitial pneumonia strongly suggests hard-metal disease and may implicate occupational exposure. (*Id.*).

Dr. Ordal agrees that claimant's disease is of the type that one would expect to become symptomatic at or near the period of exposure, rather than surfacing only after a lengthy delay. (Ex. 221-4). According to Ordal this fact, together with evidence of occupational metals in claimant's lungs, points to the subject exposure as the major cause of claimant's disease. (Ex. 221-5).

CONCLUSIONS OF LAW AND OPINION

Procedure

In a post-hearing motion claimant requested that I take "judicial notice" of certain DEQ records, apparently relating to environmental conditions at employer's asphalt production facility, which records claimant proffered. Employer argued that it would be improper to admit the records. For the reasons stated in my Interim Order of March 21, 1011, which order is hereby incorporated by reference into this Opinion and Order, claimant's motion was denied.

Compensability

Claimant bears the burden of proving a compensable occupational disease. ORS 656.266(1). An occupational disease is any disease or infection arising out of and in the course of employment caused by activities to which an employee is not ordinarily subjected or exposed other than during a period of regular actual employment. ORS 656.802(1)(a)(A).

To establish a compensable occupational disease, the worker's

employment conditions must be the major contributing cause of the disease. ORS 656.802(2)(a), which means a cause that contributes more than all other causes combined. *See Smothers v. Gresham Transfer, Inc.*, 332 Or 83, 133-34 (2001). Determination of the major contributing cause is a complex medical question that must be resolved on the basis of expert medical opinion. *Uris v. Compensation Department*, 247 Or 420, 426 (1967); *Deborah L. Lee*, 61 Van Natta 235, 236-237 (2009).

To determine the major cause of claimant's disease the relative contribution of all material contributing causes must be weighed. *Dietz v. Ramuda*, 130 Or App 397 (1994), *rev dismissed* 320 Or 416 (1995). When there is a dispute between medical experts more weight is given to those medical opinions which are well reasoned and based on complete information. *Somers v. SAIF*, 77 Or App 259, 263 (1986).

Claimant must prove both legal and medical causation by a preponderance of the evidence. To establish legal causation claimant must show that he engaged in potentially causative work activity; and he must prove by medical evidence that the work activities caused the injury. *See Harris v. Farmers' Co-op Creamery*, 53 Or App 618, 621 (1981). To be persuasive a medical opinion must be based upon a complete and accurate history. *Miller v. Granite Construction Co.*, 28 Or App 473, 476 (1977) (medical opinion not based on complete and accurate history is less persuasive). *See also Jackson County v. Wehren*, 186 Or App 555, 561 (2002) (a history is complete if it includes sufficient information on which to base the opinion and does not exclude information that would make the opinion less credible).

Because the issue presented involves a complex medical question it must be resolved with expert medical opinion. *Uris v. Comp. Dep't*, 247 Or 420 (1967); *Barnett v. SAIF*, 122 Or App 279, 283 (1993). More weight is given to those medical opinions which are well reasoned and based on complete information. *Somers v. SAIF*, 77 Or App 259, 263 (1986).

ANALYSIS

Claimant relies upon Dr. Abraham, who diagnosed pulmonary interstitial fibrosis, and upon Drs. Ordal and Keppel, both of whom believe that claimant has

giant cell interstitial pneumonia. All three physicians attribute claimant's pulmonary disease to the subject occupational exposure.

Employer contends that the correct diagnosis for claimant's pulmonary condition is DIP; and relies on Drs. Burton and Barker, who believe that the condition was caused by cigarette smoking.

For the reasons that follow, I find that claimant has met his burden of proof. This is based primarily upon the persuasive opinion of Dr. Abraham, who believes that claimant has pulmonary fibrosis, and that it was caused by his occupational exposure.

Dr. Abraham was initially recommended to assist in evaluation of the case by IME physician Barker, due to Abraham's national reputation as an expert in hard metal lung disease. (Ex. 88-3). Abraham is a board certified anatomic pathologist (Ex. 176, Exhibit E-2), and is a widely-published author of many medical journal articles and abstracts. (Ex. 176, Exhibits B, E). After examining claimant's biopsy slides, Dr. Abraham wrote to Dr. Beckett, indicating that the metals found in claimant's lung biopsy had confirmed "exposure to welding or some metal working environments[.]" (Ex. 163-1).

Dr. Abraham believes that claimant has pulmonary fibrosis, caused by his inhalation of material associated with grinding, welding and cutting steel bins. (Ex. 176-31). He explained that this exposure caused a reaction in the lung which led to inflammation and fibrosis. (Ex. 176-34). Abraham believes that claimant's pulmonary fibrosis was caused by "his exposure to a variety of metals and other materials that we may not be able to detect such as gases that are involved in welding that are capable of producing fibrosis[.]" (Ex. 176-39).

Employer contends that Abraham is not persuasive, because he does not address Dr. Treger's finding of limited interstitial fibrosis. I disagree. Dr. Abraham reviewed each of the 27 pathology slides that were sent to him; and he examined two of them (that had been sent in paraffin blocks) with an electron microscope. (Ex. 176-62). Abraham's findings and conclusions regarding what the pathology slides reveal was not rebutted by Treger. As there is no other pathologist in this record, I find that Dr. Abraham's interpretation is more persuasive than Dr. Treger's. *See Julio Garcia,* 59 Van Natta 802, 803 (2007) (failure to rebut a contrary opinion rendered a physician's opinion unpersuasive).

Dr. Abraham explained the difference between inflammatory and fibrotic conditions (Ex. 176-18); and testified that claimant has interstitial (pulmonary) fibrosis – he has scarring in the lungs, along with an accumulation of dust. (Ex. 176-19). Abraham believes that claimant's work exposure is the major contributing cause of the (interstitial fibrosis) condition he diagnosed. (Ex. 176-55, and -61). He does not believe that there was any other significant contributing cause. (Ex. 176-33, -34, -39).

Employer argues that Abraham is not persuasive, because he does not rebut Dr. Lewis, who attributes claimant's pulmonary fibrosis to cigarette smoking. Again, I disagree. Abraham does rebut Lewis – he testified that the kind of industrial exposure claimant experienced provokes a reaction in the lungs, which leads to inflammation and fibrosis. (Ex. 176-34). By contrast cigarette smoking, while it can contribute to lung disease, does not cause fibrosis. (Ex. 176-39, and -40). According to Abraham, smoking causes emphysema (destruction of the lung), which is the opposite of fibrosis. (Ex. 176-40).

Abraham concluded that claimant's pulmonary fibrosis was acquired by lung injury, based upon his history and pathological findings. (Ex. 176-45). He explained that,

> "the predominant and most unusual type of particulate material found in his lung was the metal * * * which support[s] the fact that he's had exposure to sources containing those metals." (Ex. 176-46).

Abraham explained that toxic gases from welding are not detectible on biopsy – "they do their thing and are gone." (Ex. 176-47, and -48). Sometimes, however, the metal fumes do contain actual particles. (Ex. 176-48). From claimant's biopsy Abraham can say that some of his exposure came from material consistent with stainless steel (*Id.*); and could have come from grinding as well as soldering or welding. (Ex. 176-49). Cigarette smoking, however, is not a source of the metals found in claimant's biopsy. (*Id.*). Abraham explained that the metal in claimant's lungs,

> "didn't come from smoking and it didn't come from the ambient air, it came from some operation that resulted in release of those

particular individual and combinations of metals into the air that the person breathes." (Ex. 176-50).

Abraham does not consider claimant's disease idiopathic, due to the fact that he was exposed to agents capable of causing lung injury – *i.e*, to dust and fumes from grinding and welding a variety of metals. (Ex. 176-39). Nor does he believe that claimant's genetics played a role. (Ex. 176-32, and -45).

Employer contends that Abraham is not persuasive because he did not have a complete understanding of claimant's work activities, and has not reviewed all the relevant medical records. I disagree. I find that, among other things, Abraham had read claimant's own description of his work activities for employer, along with his relevant employment history. (Ex. 176-51, and -52).[10]

I specifically find support in this record for Abraham's assumptions (Ex. 176-39) that claimant's work had exposed him to various metals and other potentially harmful materials including gases, and to dust and fumes from grinding and welding a variety of metals. Although he initially knew only that claimant "did manual welding on old steel bins, and grinding and welding of steel, oxyacetylene cutting of steel bins" (Ex. 176-31), Abraham testified that even 32 days of grinding metals was enough to have caused claimant's disease. (Ex. 176-33). He is also aware of claimant's smoking history. (Ex. 176-40). Employer, in fact, fails to cite specific history or significant information that Dr. Abraham lacked, even after being invited to do so. (Ex. 176-71, and -72). Accordingly, I find that he had a complete history. *See Jackson County v. Wehren, supra.*

In particular, I do not find persuasive Dr. Burton's suggestion that the metals in claimant's lungs might have come from the soil where the tobacco that claimant smoked was grown. (Ex. 220-55, and -56). Dr. Ordal stated that claimant's smoking would not have produced the metal particulates found in his lungs (Ex. 221-3); and Dr. Keppel also rejected the theory that claimant's disease was caused by smoking. (Ex. 206-2). Further, Dr. Burton fails to explain how the metal particles found in claimant's lungs – even assuming that they could have gotten from the soil into the cigarettes – could have bypassed the filter in claimant's cigarettes.[11]

[10] This two-page work history prepared by claimant is referred to in Abraham's deposition as Exhibit 83; but was thereafter renumbered, and appears in this record as Exhibit 89.
[11] Claimant told the SAIF investigator that he smoked filtered Newport 100s. (Ex. 54-16).

I also find that Dr. Abraham was justified in rejecting the findings of the industrial hygiene report. (Ex. 176-68, -70, -73 and -74). I do not consider that report helpful in evaluating claimant's exposure, due to the fact that the welding and abrasive grinding activities were not performed on the day of the site visit. (Ex. 64-3). Further, although the testing was apparently done on a "typical production day" (Ex. 64-2), air monitoring consisted of only four hours during the morning. (Ex. 64-9, and -10). In light of the varied nature of claimant's work environment, including the seasonal changes described both by claimant and Mr. Meyers, I find that the industrial hygiene report has little probative value.

Finally, employer urges me to reject Dr. Abraham's causation analysis on the grounds that it "is impermissibly based on deductive reasoning," citing *Bronco Cleaners v. Velasquez*, 141 Or 295 (1996). Again I disagree. In *Bronco* the court simply explains that under ORS 656.266 a claimant cannot meet his burden of proving compensability merely by disproving other possible explanations:

> "The rationale forbidden by the statute depends on the absence of specific evidence of causation. Among other things, the statute provides that a claimant may not solely rely on the deductive reasoning that, because the condition did not occur until after the exposure to the work environment and cannot be proven to have been caused by another causative agent, it must have been caused by the work environment." *Id.*, at 299.

Deductive reasoning, however, is not what Dr. Abraham uses here. I find that he persuasively explains why he rejects competing opinions regarding the nature of claimant's pulmonary disease. Abraham testified that he could not, from the tissue samples that he saw, have diagnosed, *inter alia*, DIP. (Ex. 176-22). He agreed that DIP is mostly associated with smoking, and that claimant's smoking could have contributed to DIP, "if that was the main pattern." (Ex. 176-24). Abraham explained, however, that cigarette smoking does not cause fibrosis – it causes emphysema, citing a study by Dr. Nasr. (Ex. 176-39, and -40).

Dr. Burton, who is not board certified in either pathology or pulmonology (Ex. 220-32), concedes that Abraham has superior expertise when it comes to evaluating a section of lung tissue. (Ex. 220-58). And Burton agrees that "DIP is differentiated from other forms of interstitial lung disease on the basis of

pathological findings." (Ex. 193-17). Burton himself has never treated a patient with hard metal lung disease. (Ex. 220-33). Nonetheless, Burton stubbornly rejects the possibility that the evidence here might lead to a diagnosis other than DIP (Ex. 220-13); and summarily dismisses the possibility that claimant's grinding and welding activities could have contributed to his pulmonary condition. Even though he acknowledges that the industrial hygienist had not sampled those activities, Burton contends:

> "The grinding operations later described by [claimant], though not specifically addressed in the industrial hygiene survey, are unlikely factors that led to the development of any pulmonary disease. Most welders will utilize a hand grinder to prepare the metal surface or to revise or shape a weld joint. When operating a hand grinder, the wheel is always directed in a manner to spin the metal fragments and grinding debris away from the operator. In the outdoor air, there is little opportunity for an airborne exposure while operating a grinder in this manner." (Ex. 193-16, and -17).

Burton considered his assumption that claimant was welding in the open air to be a "major factor" in his causation analysis. (Ex. 220-40). His assumption of a basically risk-free grinding environment, however, is not supported by the record. Claimant credibly testified that he would have to work at times in confined spaces while grinding, and would breathe particulate matter and welding fumes while doing so. He told the SAIF investigator that he was cutting metal seven hours per day for the first three weeks; then cutting and grinding two or three hours per day for six months. (Ex. 54-7). Claimant told the investigator that he had not used breathing protection until he had already started to get sick. (Ex. 54-9). Dr. Burton, however, having decided that claimant has a smoking-related disease, denies that the occupational exposure is even relevant:

> "Because [claimant's] diagnosed condition is unrelated to workplace exposures, it is a moot point whether or not he actually experienced any measurable exposure to airborne dust or metals at his workplace since the type of condition diagnosed in this case is causally unrelated to occupational exposures." (Ex. 193-16).

Dr. Burton completely ignores the considerable evidence of claimant's occupational exposure, including the very conditions which fit the "one caveat"

that Burton had appended to his statement about the relative safety of welding activity – namely, "working in a closed-space environment where some respiratory protection may be required[.]" (Ex. 220-68). For these reasons I find Burton unpersuasive. *See Moe v. Ceiling Sys.*, 44 Or App 429, 433 (1980) (rejecting unexplained or conclusory opinions).

Employer suggests that Dr. Barker, who had also settled on smoking as the cause of claimant's condition, is persuasive. I disagree. Barker initially felt that claimant's work activities had contributed to his disease (Ex. 65-8), but he did not want to offer an opinion regarding the major cause "until further review of the pathology is made." (Ex. 88-4). He suggested that claimant's condition was unusual enough that "a review by an expert such as Dr. Abraham in New York would be most helpful." (Ex. 88-3).

Rather than wait for Abraham's report, however, Barker later changed his mind, based on two factors: (1) the video of claimant smoking (after his biopsy procedure); and (2) "further review of descriptions of [claimant's] workplace and of investigative reports of metals in the workplace." (Ex. 107-2). Barker does not describe his "further review" of claimant's workplace, or identify the "investigative reports" that he cites.

In his February 7, 2007 deposition, in fact, Barker was asked whether using a grinder to grind aggressively on rusty steel items and breathing in that environment might be potentially hazardous. He responded, "Well, it is possible. I would have to review in detail, you know, the exact components and things because I actually haven't seen those reports[.]" (Ex. 136-24). Later in the same deposition he was asked whether the biopsy tissue might be available to be analyzed by a pathologist. Barker responded,

> "Well, let me put it this way. The tissue, itself, again, I'm not there. But the tissue and the slides can't be reviewed, or maybe they have been reviewed, I actually don't know. As far as analyzing them for minerals, I don't – you would have to talk to the pathologist to see whether it is in a form that can be analyzed for any of the metals or minerals that we are talking about." (Ex. 136-27).

Dr. Barker does not offer an explanation for the presence of metal in claimant's lungs; nor does he rebut Dr. Abraham's opinion (Ex. 176-49, and -50)

that cigarette smoking is not a source of those metals. Accordingly, I find him unpersuasive. *Julio Garcia, supra*. Further, because Barker ultimately reached his conclusions without evaluating the very evidence that he had indicated he would need, and without explaining the "further review" of claimant's workplace, or the "investigative reports" upon which he based his opinion, I find him unpersuasive. *Moe v. Ceiling Sys., supra.*

Finally, Dr. Abraham does not diagnose giant cell interstitial pneumonia, as did Drs. Ordal, Keppel and Treger. Abraham testified that claimant's lung tissue did have "a few nuclei, but it doesn't approach what is seen in the hard metal disease of giant cell interstitial pneumonia." (Ex. 176-41). Nobody here has challenged Dr. Abraham's statement that he had "probably seen more of those cases than most anybody." (Ex. 176-44, and -45). Because I find that this case turns to a significant degree upon the interpretation of claimant's lung pathology, I find that Abraham is the most persuasive.

In sum, I find that Dr. Abraham's analysis is actually very straightforward: (1) the objective medical evidence showed that claimant had abnormally high levels of a variety of industrial metals in his lungs; (2) the presence of those metals could be explained by the proven occupational exposure, and could not be explained by claimant's smoking; and (3) the presence of metal in claimant's lungs caused pulmonary fibrosis, which requires medical treatment.

I find that the persuasiveness of Dr. Abraham's opinion is enhanced by his consideration of competing opinions, as discussed above. *See Pennie McAdams*, 47 Van Natta 258 (1995) (consideration of contrary opinions further heightens the logical force of an opinion). I also find that Abraham's factual assumptions are supported by the record, and that his reasoning is logical. I find that his opinion meets claimant's burden of proof.

Summary of Findings and Conclusions

I find, based upon the expert opinions of Drs. Abraham, Ordal, Keppel, and Lewis, that claimant's subject work activity, including the described metal-grinding, metal-cutting and welding activities, exposed him to potentially harmful inorganic airborne materials, including dust.

I find that, as a result of his exposure to industrial dust, claimant suffered an accumulation of various metal particles in his lungs, as demonstrated in Dr. Abraham's November 13, 2007 letter/report. (Ex. 163).

I find that, as a result of the accumulation of metal particles in his lungs, claimant contracted pulmonary interstitial fibrosis, as described by Dr. Abraham.

I find, based upon the persuasive opinion of Dr. Abraham, that claimant's described work activity here was the major contributing cause of his pulmonary interstitial fibrosis condition; and of the disability resulting from, and the need for medical treatment of, that condition.

In light of these findings, employer's denial must be set aside.

Attorney Fee

Claimant's attorney is entitled to an assessed attorney fee for services at hearing under ORS 656.386(1). After considering the factors set forth in OAR 438-015-0010(4) and applying them to this case, I find that a reasonable fee for claimant's counsel's services is $32,000, payable by employer. In reaching this conclusion, I have particularly considered the time devoted to the case (as represented by the size and content of the record, including three lengthy physician depositions); the complexity of the issues; the skill and extensive experience of the two lawyers involved; the value of the interest involved; and the significant risk that claimant's counsel could go uncompensated. In particular, I find that the case was of greater than ordinary medical complexity; and that claimant may face substantial medical expenses in the future, given his relatively young age, and the fact that he faces the prospect of significant future medical treatment, including the possibility of a lung transplant.

Litigation Costs

Because this order establishes compensability of the claim, and issues after the effective date of *amended* ORS 656.386(2) and OAR 438-015-0019, it is appropriate to award reasonable litigation costs. *See Sean Mecham*, 61 Van Natta 259 (2009). Accordingly, claimant is awarded reasonable expenses and costs for records, expert opinions, and witness fees, if any, incurred in finally prevailing

over the denial, to be paid by the employer. The procedure for recovering an award of costs is prescribed in OAR 438-015-0019(3).

ORDER

IT IS THEREFORE ORDERED that employer's May 22, 2006 denial is set aside, and employer is ordered to accept and process the claim according to law.

IT IS FURTHER ORDERED that, in accordance with ORS 656.386, claimant's attorney Arthur W. Stevens III is awarded $32,000 as a reasonable assessed attorney fee, together with his litigation costs, to be paid by employer.

Notice to all parties: If you are dissatisfied with this Order, you may, within thirty (30) days after the mailing date on this Order, request a review by the Workers' Compensation Board, 2601 25th St. SE, Suite 150, Salem, Oregon 97302-1280. Any such request for review shall be mailed to the Board at the above address, with copies of such request mailed to all other parties to this proceeding. **Both of these actions must be taken within 30 days of the Administrative Law Judge's Order.**

When one party requests a review by the Board, the other party or parties shall have the remainder of the 30-day period, and in no case less than 10 days, in which to request Board review in the same manner. **The 10-day minimum is provided even if it extends the time allowed to request Board review beyond 30 days.**

Failure to mail the request for review to the Board and mail copies of the request to all other parties within the time allowed will result in the loss of your right to appeal this order and the Board will be unable to review the Administrative Law Judge's decision.

Entered at Medford, Oregon on October 4, 2012, with copies mailed to:

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Workers' Compensation Board

BRUCE D SMITH
Administrative Law Judge