WCAT Decision Number: WCAT-2005-00823-RB
WCAT Decision Date: February 16, 2005
Panel: Teresa White, Vice Chair

Introduction

The worker, who was employed in the employer's retail liquor stores, appeals a decision of the Workers' Compensation Board (Board) dated June 22, 2000. The Board denied the worker's claim for exacerbation of her pre-existing asthma. The worker appealed that decision to the former Workers' Compensation Review Board (Review Board).

The worker is represented by legal counsel. The employer is participating, represented by a consultant.

An oral hearing of the worker's appeal was originally scheduled for and commenced on April 14, 2004. The worker attended with her legal counsel, who requested an adjournment of the oral hearing on the basis that the worker had retained counsel only the day before. After hearing submissions from counsel for the worker, and the employer's representative, I decided to adjourn the oral hearing, which was recommenced on July 14, 2004.

There were two pre-hearing teleconferences held prior to the oral hearing, during which procedural issues relating to expert evidence and other matters were discussed. In addition, during the oral hearing, I requested production of additional medical records relating to the worker and additional reports relating to evidence provided by an expert on behalf of the employer. Counsel for the worker, and the employer's representative, were given opportunities to make submissions regarding this evidence, and to reply as appropriate.

During the pre-hearing processes, I disclosed to the parties three decisions of the former Review Board relating to other workers from the same workplace(s) who made workers' compensation claims relating to the glass crusher machines. Counsel for the worker objected to their production on the basis that they were irrelevant to the worker's appeal. After hearing all of the evidence and submissions in this appeal, I have concluded that it is unnecessary for me to have reference to those previous Review Board decisions. As such, it is unnecessary for me to address counsel's objection, and I have not reached any conclusion with regard to the previous decisions' admissibility or weight.

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Issue(s)

The issue is whether the worker is entitled to acceptance of her claim that her pre-existing asthma was significantly aggravated by the nature of her employment. Specifically, the worker alleges that she was exposed to contaminants, primarily mould, because of the introduction into the workplace of "glass crushing" machines used to crush returned glass bottles.

It should be noted that several others who were employed in the same workplace suggest that glass particles or dust caused illness or symptoms of various types. The worker's condition is alleged to be due to mould, and not glass.

During this appeal, counsel for the worker submitted medical reports respecting a sinus condition suffered by the worker. The Board has not adjudicated any sinus condition. During the pre-hearing procedures and at the oral hearing, I raised with counsel the issue of whether WCAT has jurisdiction with respect to an issue that had not been adjudicated by the Board. Counsel submitted that WCAT did have jurisdiction. He referred to WCAT Decision No. WCAT-2003-02677-RB. In that decision the panel found that it had jurisdiction to consider a worker's neck condition, in addition to bursitis/tendonitis, because the worker had initiated a claim for a "symptom complex" that could have been caused by either condition or both in combination, and the medical reports clearly identified both conditions.

While I agree that where a worker's claim is based on a symptom complex that could be caused by a number of conditions, WCAT may have jurisdiction with respect to those conditions, particularly where the medical reports identified those conditions, I do not agree that the facts of this case bring that principle in to play. A review of the medical reports on the worker's file at the time of the Board's decision under appeal did not suggest that the worker's condition related to her sinuses. It may be that sinus conditions and asthma can be related and/or can occur together, but in this case the Board did not have any information suggesting that the worker had a sinus condition alleged to be due to the nature of her employment. The medical reports addressing the worker's sinuses were tendered in this appeal proceeding. Those reports suggest that the worker has been suffering from a significant sinus condition which has required surgery. The Board has not had the opportunity to adjudicate the compensability of that condition.

To decide otherwise would deprive the worker of the opportunity to have her claim adjudicated by the Board, and if she is unhappy with the Board's conclusion, to appeal to the Review Division and then to WCAT. The medical reports respecting the worker's sinus condition will be scanned into the worker's Board file as part of the WCAT appeal documents.

Jurisdiction

This appeal was filed with the Review Board. On March 3, 2003, the Review Board and the Appeal Division of the Board were replaced by the Workers' Compensation Appeal Tribunal (WCAT). As this appeal had not been considered by a Review Board panel before that date, it has been decided as a WCAT appeal. (See the *Workers Compensation Amendment Act (No. 2), 2002*, section 38.)

WCAT may consider all questions of fact and law arising in an appeal, but is not bound by legal precedent (section 250(1) of the *Workers Compensation Act* (Act)). WCAT must make its decision on the merits and justice of the case, but in so doing, must apply a policy of the Board's board of directors that is applicable in the case. WCAT has exclusive jurisdiction to inquire into, hear and determine all those matters and questions of fact and law arising or required to be determined in an appeal before it (section 254 of the Act).

The majority of the published policy applicable to this appeal is found in the *Rehabilitation Services and Claims Manual*, *Volume I* (RSCM I). In the event that any applicable policy is found in another source, such as Volume II of the RSCM (RSCM II), that fact will be specifically mentioned.

Background and Evidence

At the time she made her claim for compensation in February of 2000, the worker was working as an auxiliary clerk in the employer's retail stores. She was 24 years old. The worker stated on her application that she had asthma that had been worsening over a period of time.

The worker commenced work as an auxiliary clerk in June of 1995. She worked on call up to 35 hours per work, and her duties included working on the cash register, collecting empty bottles, working in the warehouse, and work as supervisor. The worker indicated on a questionnaire she completed for the Board that she wore a dust mask, gloves and goggles when working on empty bottles.

The worker said on the questionnaire that her asthma had worsened. She had a tight, heavy feeling in her chest, and fatigue. The worker also mentioned her "mouth bleeding", which she clarified at the oral hearing was "just a bit" inside her mouth when she brushed her teeth. She also said she had "blood spotting when eliminating waste," which she stated at the oral hearing went away "ages ago." She had trouble sleeping and had to pile up pillows.

I note that the worker's reports of blood spotting while brushing her teeth, and when "eliminating waste" were symptoms described by some other workers who made claims respecting alleged exposure and/or ingestion of glass from the glass crushing machines

in the workplace. The worker did not assert that the symptoms either continued, or that they were the result of glass ingestion. Neither did her counsel make any submissions in that respect. I consider the only issue to be whether the worker had a compensable exacerbation of her asthma.

On the questionnaire, the worker said that her symptoms were worse in the workplace, and she required the use of a bronchodilator at work.

The worker has a lengthy history of asthma. Her family physician's chart records commence in 1990, when the worker was 14 years old. The entries related to the worker's asthma are summarized following.

The worker was noted to have a history of asthma attacks, and an allergist had diagnosed bronchospasm (an abnormal contraction of the smooth muscle of the bronchi, resulting in an acute narrowing and obstruction of the respiratory airway). The worker was on Ventolin (a bronchodilator) puffs as needed.

Respiratory infections such as colds caused exacerbations of the worker's asthma. She often required antibiotics in these circumstances. There are chart entries in June 1990, January 1992, March 1993, December 1993, March 1996, April 1996, October 1996 and November 1996 reflecting exacerbations of her asthma in conjunction with respiratory infections.

The chart notes also reflect a number of emergency room visits, in December 1993, October 1995, and October 1996. In November 1996 the worker was hospitalized for one week due to asthma.

The chart notes generally reflect a fluctuating course, with exacerbations and remissions. The worker received a number of medications, including Ventolin, Beclovent (a type of inhaled steroid prescribed for the prevention of recurring asthma symptoms), Beconase (a type of inhaled steroid prescribed to relieve allergic symptoms in the nose), Pulmicort (another version of an inhaled steroid for asthma), prednisone (an oral corticosteroid), Severent (a long acting bronchodilator used to treat chronic asthma), Advair (an inhaler containing a combination of Fluticasone, a steroid, and Salmeterol, a bronchodilator). In April 2002 the worker was getting sudden asthma attacks which she said were like heart attacks.

The worker's evidence at the oral hearing was that her asthma was relatively mild before her exposure to the glass crushers. Given her three visits to the emergency room, and her hospitalization in November 1996, it seems apparent that although she had periods of time where she was better, her asthma was relatively severe, even before the introduction of the glass crushers in or about October 1998.

There are a number of consultation reports from specialist physicians on the worker's file. Several are from Dr. Keenan, respirologist.

A letter dated January 29, 2000 is from Dr. Keenan to the worker's union representative. Dr. Keenan noted that by history the worker told him that her asthma was well controlled until January of 1999, and at this time the glass crusher was introduced. She noticed a gradual worsening of her asthma. Dr. Keenan said that her asthma "did get worse from January 1999 to the time she left work in August 2000 according to her history and I am inclined to believe her." Dr. Keenan further said that the worker's asthma got worse coincident with the introduction of the glass crusher, and he suspected this had an effect on her asthma control, but it was difficult to prove. The worker took a long time to get better and continued to fluctuate for a number of months after she stopped work. Dr. Keenan said that he was not aware of evidence in the literature that supports the dust from glass crushers as a specific irritant for asthmatics. He said that he could see it acting as a non-specific irritant but his suspicion alone was not enough. The worker's pattern of worsening asthma control "does happen to asthmatics without any specific exposure. It will be difficult to prove the association."

On October 4, 2000 Dr. Keenan noted that the worker had a history of hospitalization "once at the age of 20." The worker was allergic to nuts, trees, glass, mould, weeds and cats. She had moved home which was less stressful, "but does add the exposure to the family cat." Dr. Keenan said it sounded like the worker's poor control over the past two years was related to occupational exposure to the glass crusher.

On October 23, 2000 Dr. Keenan noted that the worker had a history of "very stable asthma, until deterioration coincidence [sic] with a glass crusher at her job." The worker had been off work for about three weeks and felt subjectively improved. Dr. Keenan felt she may be developing some degree of fixed obstruction related to her work exposure.

On November 20, 2000, Dr. Keenan, respirologist wrote that he did not think the worker should return to her employment "at present and probably ever, as long as they have glass crushers." He said:

> It is still not being completely confirmed that this is a problem, but I suspect it is and is adding to her current difficulties. The concerning issue is the fact that she has been away from work now for over, I believe a couple of months and is not showing significant improvement.

On January 22, 2001 Dr. Keenan, respirologist, wrote that the worker was applying for workers' compensation for her asthma. He said:

> While I feel that it may well have been aggravated initially by the glass crusher it is going to be difficulty [sic] to prove that this initial exposure has led to ongoing problems with her asthma for a number of months after

> being out of the workplace. I have explained this to her. At this time her asthma is fairly well controlled and she is going to be faced with having to either return to work and seeing what happens or perhaps finding new employment. She is considering returning to work for shorter shifts if possible.

At the oral hearing, the worker said that she worked at 6 different stores, from 5 to 8.75 hours per day. She said that before she began working for the employer, she had asthma, but it was "not serious." The worker said she had medical attention for her asthma, but not very often. She had been in emergency for her asthma a "couple of times" but she was not sure if it was before she began working for the employer.

The worker's evidence was that her asthma had been in remission for a couple of years, and began to decline at the end of the summer in 1999. She had used Pulmicort, but only when she had a cold.

The worker's evidence was that she had some time loss from work between May and August of 1995. Just after she started with the employer she was in hospital for about one week. After that hospital stay, she had a remission of her asthma symptoms.

The worker said she noticed a change when she began working for the employer, and she began using her bronchodilator more and more. She said that the stores started using the glass crushers at the end of 1998.

The worker's evidence regarding protective equipment was that she wore gloves, eye goggles and was, at first, allowed to wear a dust mask but was later told not to do that by a manager.

The worker described the empty bottles that were returned to the store. She said that some were clean, but some were not. The wine bottles often had residue in them. "Corona" beer bottles often had lime pieces in them that were mouldy and rotten. There were often flies in them. "Bad" Corona bottles were put in a box and taped up. The worker's evidence was she was told to crush bottles that were not clean as it was "good customer service." Refusing bottles was considered to be bad customer service.

The worker said that her symptoms became worse after the bottle crushers were introduced, not before. "Raking" the broken glass in a bin was problematic, and the worker said the employer stopped bringing in masks.

The worker said that she became aware of her problem with nasal polyps in 2000. In February 2000 her peak flow rate had decreased and it had never been that low since she had been in hospital. She was wheezing and her nose was stuffed up. She was taking Advil every day for headaches.

The worker was well enough to return to work in early 2001, and she said that shortly after she returned to work the employer removed the glass crushers. The worker said she was advised by her physician not to work with the crushers, although she still worked on empty bottles. Someone else would crush them.

In 2001 the worker saw Dr. Javer, a specialist in ears/nose/throat. He felt she had allergic fungal sinusitis, and the worker said Dr. Javer had said to her that he had "seen a lot of it" in some other workers. Mould or fungus become lodged in the nose, and an allergic reaction causes swelling. The worker's condition became worse and worse.

At surgery, the worker was found to have "fungus balls" and polyps. She had surgery to remove them. After the surgery she did really well, but then gradually the stuffy nose feeling returned. The polyps started to grow again. The worker said that her breathing had still not returned to how it was before 1999.

The worker said that at the time of the oral hearing, she was still using Advair. She had a stuffy nose, and her sense of smell was not as good. She was still wheezing, particular if she got "going too much" or if she was laughing. She had headaches, but "not too many."

On questioning by the employer's representative, the worker said that she had owned a horse but did not any more. She said she had been living in a basement suite, but moved out shortly before she went off work in August 2000. The reason she moved out was because of her roommate's behaviour and not her asthma.

Dr. Javer provided a report dated June 10, 2004. He said he first saw the worker on April 24, 2001 with a chief complaint of bilateral nasal obstruction and significant worsening of her asthma over the previous six months. The worker told Dr. Javer that before she began working for the employer her asthma had been in remission and she had been "trying to regain her sense of smell" until the glass crusher machines were brought into the store. Since then, her asthma had gradually worsened and she was eventually required to stop work for a period of six months.

Dr. Javer reported that on rigid nasal endoscopy the worker had extensive polyposis completely obstructing her nasal cavities on both sides. She had what appeared to be consistent with allergic mucin (mucin is a substance found in mucous secretions.). Dr Javer felt she had bilateral extensive nasal polyposis and possibly chronic sinusitis with a "high likelihood of allergic fungal sinusitis."

On April 30, 2001 the worker had bilateral endoscopic polypectomy. The pathology results showed loose oedematous stroma infiltrated by eosinophils and "essentially an inflammatory polyp picture with no evidence of malignancy."

The worker had CT scans in August and September of 2001. These showed massive polyposis and Dr. Javer said the findings were consistent with possible allergic fungal sinusitis.

On August 27, 2003 the worker had computer assisted sinus surgery. She was noted to have extensive bilateral mucosal disease with large amounts of allergic mucin, which was "very consistent with allergic fungal sinusitis." However, fungal cultures returned negative. Dr. Javer said that difficulty growing fungus in the microbiology lab is a consistent problem, and "we therefore diagnose allergic fungal sinusitis when all other criteria are present despite a negative fungal culture."

Dr. Javer said that in his opinion the worker has allergic fungal sinusitis, which is extremely difficult to control. No fungi had been grown from her sinuses, but Dr. Javer said this was not inconsistent, as he had no formal mycology lab to work with. Dr. Javer said there was a very strong correlation between the worker's sinus condition worsening her asthma and the introduction of the glass crushers into the workplace. He said it was extremely likely that the "particulate matter from the glass crushers, plus or minus the mould that would be released from such a setup, would result in a worsening of her condition."

Dr. Javer also provided a "general opinion based on multiple patient consultations." He said that he had seen at least six patients who had been affected by the glass crushers. He opined that the "ventilation system for the glass crushers was ineffective." Most of his patients suffered from significant nasal and sinus irritation with a number requiring extensive surgical intervention for chronic sinus infection.

Dr. Javer provided an additional report dated September 13, 2004. He said that in August of 2004 a culture was done on the worker's mucus, and a significant amount of *Aspergillus* species had been grown from the mucus. He said he did not have any doubt that the worker had allergic fungal sinusitis. The growth of fungal cultures was the only one of five criteria the worker had not met, and this criteria had now been met. He said that it is felt that individuals such as the worker have significant hyper-reactivity to fungal spores in their environment, but when these patients are exposed to high levels of fungal spores, which may be present in the home or work environment, they become significantly worse.

Dr. Javer noted that reports about fungal levels in the employer's stores found that *Penicillium* and *Aspergillus* spores made up 1/3 of the total count near the glass crusher machine operator and more than half the concentration above the glass bin. There was an obvious source of fungal contamination in the Corona beer bottles. Dr. Javer said that his main concern was that the spores from the Dematiaceous (having brown or black conidia or hyphae) family of fungal pathogens are the most common ones known to cause allergic fungal sinusitis. Dr. Javer said that allergic rhinosinusitis is "an

extremely complex disease that experts in the field of rhinology continue to have a hard time grappling."

Dr. Javer said that even once a diagnosis has been made and a patient operated on, there is a high rate of recurrence, "mainly because of the risk of inoculating their sinuses with Aspergillus species from their environment." The patients continue to have a high level of hyperreactivity to the fungal spores as they did before surgery and treatment. Dr. Javer said that, "We also know that asthma is directly related to the sinus disease (AFS) and will get worse when their allergic fungal sinusitis worsens."

Dr. Javer said that it was very likely that environmental exposure worsened the worker's condition because of "her exposure to the high levels of fungal spores in the liquor stores."

With respect to the relationship between allergic fungal sinusitis to asthma, Dr. Javer said it was "present and significant." He said, however, that

> …not all allergic fungal sinusitis patients suffer from asthma. Adult onset asthma on the other hand is very often closely associated with allergic fungal sinusitis. There is strong evidence that sinuses and lungs constitute what we refer to as "united airways" and disease in one area will affect the other. There is a strong sense that sinus inflammation can trigger asthma in patients who previously did not suffer from asthma.

Dr. Javer said that in the worker's situation, it is hard to predict whether the asthma, which began when she was in Grade 8, triggered the sinus inflammation because of environmental irritants, or whether there was a low grade sinus condition since the worker was a teenager that caused her asthma and was exacerbated when she was exposed to the environmental irritants.

Dr. Javer provided copies of two scientific articles (Ferguson, B.J. Eosinophilic Mucin Rhinosinusitis: A Distinct Clinicopathological Entity. *The Laryngoscope* 110: 799-813, May 2000*;* Marple, B.F. Allergic Fungal Rhinosinusitis: Current Theories and Management Strategies. *The Laryngoscope* 111: 1006, June 2001).

The Ferguson paper reviewed the available literature, and reported on a study of forty-two cases. The author noted that there were two different conditions. The first is allergic fungal sinusitis (AFS), and the second eosinophilic mucin rhinosinusitis (EMRS). These overlap on clinical and immunologic examination, but are "significantly and statistically different." EMRS patients are "usually older" and have adult onset asthma. They are more frequently aspirin sensitive. There are immunological differences, but the significance of them is unknown.

The Marple paper notes that the combination of nasal polyposis, crust formation and sinus cultures yielding *Aspergillus* was first noted in 1976. Eventually this became known as AFS. The abstract notes that fungi associated with the development of AFS are ubiquitous and predominately of the dematiaceous family. Exposure alone appears to be insufficient to initiate the disease. It is likely that the initiation of the inflammatory cascade leading to AFS is a multifactor event which includes exposure to fungi.

The section of the paper titled "epidemiology" states that AFS is most common among adolescents and young adults, and is invariably associated with nasal polyposis and the presence of allergic fungal mucin. Atopy is characteristic of the disease, with 2/3 patients reporting a history of allergic rhinitis. In one study, approximately 50% of the patients with AFS had asthma.

The conclusion section of the Marple paper notes that treatment techniques for AFS are changing, and that a comprehensive management plan incorporating both medical and surgical care remains the most likely way to provide long term disease control.

Mr. G. A. Clark gave evidence on behalf of the employer. Mr. Clark has a Masters degree in Biology, and is a certified industrial hygienist, registered occupational hygienist and registered professional biologist. He has attended a number of courses and conferences respecting indoor air contamination and in particular the issue of fungal contamination. He has also given evidence in many court proceedings in the United States, in most instances for the plaintiff.

Mr. Clark was involved in testing the employer's stores in mid-1999. His evidence at the oral hearing was that the filtration system attached to the glass crushing machines was adequate. He took samples in the stores using spore trap samplers and samples of glass dust.

Mr. Clark said that the highest incidence of exposure to mould spores occurred while raking the crushed glass. The highest level of spores was near the machine. The spore levels "faded away to background" five feet away from the machines. The handling of bottles themselves, and in particular bottles with visible fungal growth, would spread the spores around. He said that Corona bottles often were colonized by mould because of the lime pieces in them, and that fungal growth could occur in other bottles.

Mr. Clark gave extensive evidence about the types of spores found. He said that *Aspergillus* and *Penicillium* spores looked very similar, but his opinion was that *Penicillium* was predominant. He based that opinion on the spore producing structure seen in the samples (a conidiophore, which *Penicillium* produces but not *Aspergillus*), and on the fact that a culture from another store on Vancouver Island had grown *Penicillium*.

Mr. Clark said that *Aspergillus* grows in the soil, and "all over." There are 150 different species recorded. They do better in a warmer, wetter environment. *Aspergillus fumigatus* is a common variety, and it occurs outdoors and in water damaged areas. Mr. Clark said it was probably present in the hearing room. It is not the most common, with *Aspergillus niger* and *Aspergillus flavus* more common. Mr. Clark's opinion based on the data he had on the employer's stores was that *Aspergillus fumigatus* was not the predominant organism. However, he could not be certain.

Mr. Clark said that both types of fungus utilize similar food sources, such as gypsum board, fruit and cheese. They are common soil fungi. *Aspergillus* prefers warmer temperatures than *Penicillium*.

Mr. Clark's opinion is that the worker was not exposed to anything other than elevated concentrations of *Penicillium*, and not *Aspergillus*, although he acknowledged that it was not possible to be certain. He said that his microscopic analysis usually could not distinguish between the two because the spore types are similar.

Mr. Clark acknowledged that if workers in the stores with glass breaking machines were not wearing masks, they would be exposed to higher concentrations of spores. He agreed that the risk was "significantly higher" than the population at large if there was no mask.

With respect to the finding of *Aspergillus* in the worker's nasal cavities, Mr. Clark stated that the fungus would be found in many people's noses because it is ubiquitous. He stated that if it got into an individual's nose, it would stay there until it was blown out.

The claims adjudicator sought advice from a Board medical advisor. In March 2000 he noted that because of her history of asthma, the worker was someone who would likely be adversely affected by airborne contaminants which would not affect an individual with no history of asthma. He noted that the worker was to undertake peak flow monitoring. However, at the time, the Board only had the worker's personal opinion that there was something at work aggravating her asthma. The glass crushing machine was only a speculative cause since there was no objective evidence to support this possibility.

The worker's peak flow monitoring results were reviewed by the Board medical advisor in June 2000.

The peak flow results are in the worker's file. They reflect monitoring from March 29, 2000 to April 30, 2000 with measurements in the morning and evening on most days. On March 15 the worker went to emergency, and was given prednisone.

The peak flow results show a 10 and 40 drop between mornings and evenings on days the worker went to work.

The Board medical advisor said that the peak flow results could not be described as showing the worker's work as the cause of a significant aggravation of her asthma. Overall, there was a tendency for her peak flow readings to drop between 5% and 10% on days she went to work, with no drop on days when she did not work. This evidence was compatible with "something at work causing a minimal aggravation of her asthma." In his view, a significant aggravation would result in a drop of at least 15%. He said he still did not know the precise cause of this small but fairly consistent drop in her peak flow monitoring since, "all Occupational Hygiene investigations of the glass cutting machinery have been negative."

The claims adjudicator recorded her decision in a June 16, 2000 claim log memo. She noted that the worker had problems that were not consistent with any one store, and it would be difficult to conclude that each store would have the same factors that could affect the worker's asthma. She noted that a "significant aggravation" to the worker's pre-existing asthma had not been established, referring to policy item #29.20 in the RSCM I.

In this appeal, the employer's representative provided June 29, 2004 and July 4, 20004 opinions from Dr. Jeffries, a medical doctor who practices in the areas of occupational and psychological medicine. Dr. Jeffries said that the attribution of causation by clinical physicians is less reliable than their opinions respecting evidence based diagnosis. This is because few physicians have any graduate level training in epidemiology or occupational medicine. In the case of the worker, Dr. Jeffries pointed out that she had a pre-existing disease and there was "no evidence" that any occupational exposure aggravated, accelerated or activated her asthma. Dr. Jeffries also noted that the introduction of the glass crushing machines was unwelcome and that there was apprehension about possible health effects. Asthma is subject to seasonal variations and to exacerbations over time.

In his second letter, Dr. Jeffries focused mainly on the question of causation of the worker's sinus problems, which are, as set out above, not before WCAT in this appeal.

Findings and Reasons

The evidence clearly establishes that the worker had a long-standing and significant history of asthma before the work place exposure to the glass crushers. That history included at least three emergency room admissions for asthma/shortness of breath, and, in November 1996 a week long hospital stay. I consider the evidence to strongly support a conclusion that the worker had pre-existing asthma that was subject to exacerbations. The worker's asthma had been significant enough to require ongoing medical intervention, emergency room and hospital admissions, and oral prednisone. She had significant aggravations relating to viral infections.

The worker had an aggravation of her asthma in November 1998, which was soon after the introduction of the glass crushers, although it should be noted that Dr. Keenan was under the impression that the exacerbation occurred in early 1999. There is a distinct absence of medical documentation relating to the worker's asthma between November 1996, when she was hospitalized, and the note in November 1998 regarding an exacerbation. On that basis, it appears that the worker's asthma was relatively less problematic during the period after her hospitalization in November 1996 and her November 1988 exacerbation. In that regard it should be noted that there may have been other reasons for the lack of records. However, the weight of evidence supports a conclusion that the worker did feel better for a period of time.

The question is whether the exacerbation in November 1998, and the ongoing problems experienced by the worker after that date, are due to the nature of her employment.

Policy item #29.20 in the RSCM I addresses asthma as an occupational disease.

"Asthma" is listed in Schedule B to the Act. Schedule B to the Act and section 6(3) of the Act provide workers with the benefit of a presumption that certain occupational disease is due to the nature of employment, unless the contrary is proven. In the case of asthma, the processes or industries that give rise to the presumption are exposure to western red cedar dust, isocyanate vapours or gases, or the "dust, fume or vapours of other chemicals or organic material known to cause asthma."

Policy states that there are many substances which are either known to cause asthma in a previously healthy individual or are capable of aggravating or activating an asthmatic reaction in an individual with a pre-existing asthma condition. The policy states that the significance of occupational exposures to these substances may be complicated by evidence that the worker is exposed to such substances in both occupational and non-occupational settings.

In the investigation of the claim, the adjudicator should seek evidence of whether the worker is exposed to any sensitizing substances (obtaining where available any material safety data sheets), on the nature and extent of occupational and non-occupational exposure to such substances, and on whether there is any correlation between apparent changes in airflow obstruction/responsiveness and exposure to such substances. Additional medical evidence may be available in the form of airflow monitoring, expiratory spirometry, inhalation challenge tests, and skin testing for sensitization.

Policy item #29.20 states that a pre-existing asthma condition is not compensable unless such underlying condition has been significantly aggravated, activated, or accelerated by an occupational exposure. A worker is not entitled to compensation where his or her pre-existing asthma condition is triggered or aggravated by substances which are present in both occupational and non-occupational settings unless the

workplace exposure can be shown to have been a significant cause of an aggravation of the condition. A speculative possibility that a workplace exposure to such a substance has caused an aggravation of the pre-existing asthma is insufficient for the acceptance of a claim.

Policy specifies that compensation is not payable because a worker develops an allergy or sensitivity to a substance or substances as a result of their employment. Compensation may be paid where a workplace exposure to the allergen or substance results in an asthmatic reaction.

Policy item #26.55 is also relevant to this appeal. It states that where a worker has a pre-existing disease which is aggravated by work activities to the point where the worker is thereby disabled, and where such pre-existing disease would not have been disabling in the absence of that work activity, the Board will accept that it was the work activity that rendered the disease disabling and pay compensation.

Evidence that the pre-existing disease has been significantly accelerated, activated, or advanced more quickly than would have occurred in the absence of the work activity, is confirmation that a compensable aggravation has resulted from the work.

Policy specifies that this must be distinguished from the situation where work activities have the effect of drawing to the attention of the worker the existence of the pre-existing disease without significantly affecting the course of such disease.

To be compensable as a work-related aggravation of a disease, the evidence must establish that the employment activated or accelerated the pre-existing disease to the point of disability in circumstances where such disability would not have occurred but for the employment.

Although the Board recognized that the worker had an aggravation of her asthma, this was considered to be a “minor effect,” that did not amount to a significant aggravation. It was considered a “minimal aggravation,” because a “significant aggravation” would require a drop in peak flow of at least 15%. Although the drop in peak flow was small, it was “fairly consistent.”

A review of the peak flow results from March 29 to April 30, 2000 show that there were reductions in the worker’s peak flow on seven of sixteen work days. The reductions range from approximately 4% to 20%. The reductions were over 15% on two of the work days. There are incomplete records for three of the sixteen days, and on three of the work days there was no change between morning and evening. On March 15 the worker had an emergency room visit and was put on oral prednisone. She worked on March 17, 18 and 19, while taking prednisone. On those days her peak flows were the same or improved as the day went on.

The Board medical advisor's opinion was based on his conclusion that there was a "tendency" for the worker's peak flow to drop between 5% and 10%, with no drop on days she did not work. Based on my analysis of the peak flow records, this conclusion was not completely correct. There were days with reductions over 15%. Further, the records are not very complete.

I am also cognizant of the fact that by March 29, 2000 when the worker began her peak flow record, she had already begun experiencing an exacerbation in her asthma. The chart notes show an exacerbation in November 1999, although there are no peak flow results to assist in determining the extent of that exacerbation. It seems likely that if the worker's condition did get worse in November 1999, her peak flows would have dropped then, so the baseline peak flow readings were already low.

Policy does not specify what amounts to a "significant" exacerbation. The Board medical advisor's opinion was that a 15% drop in peak flow would be significant, but a drop of 5% to 10% would not.

The problem with adjudicating whether the exacerbation of the worker's asthma was "significant" is that, as noted above, the peak flow results are neither complete, nor reflective of the worker's peak flows immediately upon onset of the exacerbation. Further, some of the results do show what would be considered by the Board medical advisor to be a significant drop.

I accept that the worker had an exacerbation of her pre-existing asthma, which began in or around November 1999. By April 2000 the worker's condition was such that she required emergency room treatment and oral prednisone.

Dr. Keenan is a respirologist, and was directly involved in the worker's care. In his very frank opinions, he states that although the worker's asthma control worsened, it would be difficult to establish that her condition months after ceasing work was still the result of the work exposures. It is also apparent that Dr. Keenan did not have a complete picture of the worker's history of asthma, which included periods of time long before the workplace exposure where she was having serious problems with her asthma which required hospitalization.

I accept, based on the whole of the evidence and in particular Mr. Clark's evidence, that it is likely that the worker was exposed to an increased level of airborne mould spores because of the empty bottles returned to the store, and the operation of the glass crusher. I do not accept that mould spore concentrations were elevated throughout the store or that those elevations were consistent over time.

I also accept that the worker, because of her pre-existing asthma, was more likely to experience problems due to elevated mould spore levels. In that respect, it do not consider it necessary to resolve the question of whether the higher levels were

Aspergillus or *Penicillium* species. I accept that both were likely present in the returned bottles, and that either or both were likely to aggravate the worker's pre-existing asthma.

On that basis, I have concluded that the worker suffered a temporary exacerbation of her pre-existing asthma as a result of exposure to mould from the empty bottles and the operation of the glass crusher. I do not consider the evidence to support a conclusion that the exacerbation was permanent

Based on Dr. Javer's evidence, it seems possible that the worker's sinus condition, which has not been adjudicated by the Board, prolonged the exacerbation of her asthma. However, the compensability of the sinus condition remains to be adjudicated.

On that basis, I have concluded that the worker's pre-existing asthma was temporarily aggravated by exposure to mould due to the nature of her employment.

Policy requires that the exacerbation be to the point of disability, and that the pre-existing disease would not have been disabling in the absence of that work activity. I am satisfied on the evidence that the worker's asthma was exacerbated to the point of disability. In that respect, I note her emergency visit on April 14, 2000 and the fact that she did not work that day. The evidence also establishes that the worker became disabled from work in August 2000, and the weight of evidence supports a conclusion that her disability from work commencing in August 2000 was due to the compensable exacerbation.

Although I consider the evidence to support a conclusion that the worker's cessation of work in August 2000 was due to the compensable exacerbation, I am unable on the evidence before me to determine how long the compensable aggravation persisted. I am satisfied that it was not permanent, given the extent of the worker's pre-existing disease and Dr. Keenan's opinions in that respect.

The question of compensability of the worker's sinus condition is referred back to the Board for adjudication, as is the question of the duration of the temporary exacerbation of her asthma caused by the nature of her employment.

Counsel requested reimbursement for the costs of Dr. Javer's report(s). Those reports were useful and the evidence was useful or helpful to the consideration of the appeal. It was reasonable for the worker to have sought such evidence in connection with the appeal. As such, they should be reimbursed in accordance with the current tariff.

I also consider Dr. Jeffries' report to have been useful, and it was reasonable for the employer's representative to have sought his report. I direct that those costs be reimbursed in accordance with the current tariff.

Finally, the costs associated with counsel for the worker obtaining copies of Dr. Jones' chart notes should be reimbursed in accordance with the current tariff.

Conclusion

The worker's appeal is allowed, in part, and the Board's decision varied. The worker had a compensable temporary aggravation of her pre-existing asthma due to the nature of her work, and specifically due to exposure to elevated levels of mould spores in the work environment because of mould contaminated empty bottles and the operation of the glass crusher. The extent of disability due to that temporary aggravation is referred back to the Board for determination.

The compensability of the worker's sinus condition has not been adjudicated by the Board and was not before WCAT in this appeal.

Teresa White

Vice Chair

TW/pm