IN THE WORKERS' COMPENSATION COURT OF THE STATE OF MONTANA

2015 MTWCC 9

WCC No. 2014-3420

CHRIS HAINES

Petitioner

vs.

MONTANA UNIVERSITY SYSTEM SELF-FUNDED WORKERS' COMPENSATION PROGRAM

Respondent/Insurer.

FINDINGS OF FACT, CONCLUSIONS OF LAW, AND JUDGMENT

Summary: Petitioner was injured in an industrial accident in which he was exposed to chlorine gas while mixing swimming pool chemicals. He later developed peripheral neuropathy in his legs, which he attributes to either the industrial accident or as an occupational disease from exposure to various pool maintenance chemicals over the course of his employment. Respondent accepted liability for ocular chemosis and other acute injuries in the immediate aftermath of the industrial accident, but denied further liability for Petitioner's peripheral neuropathy, contending that it was not caused either by the industrial accident or his ongoing chemical exposures.

Held: Petitioner has not proven that his exposure to chemicals caused his peripheral neuropathy. The weight of the medical evidence indicates that neither chlorine gas nor calcium hypochlorite caused his peripheral neuropathy. Although Petitioner alternately contended that he suffered an occupational disease from exposure to a variety of chemicals in the course and scope of employment, the only physician who testified that these chemicals caused his peripheral neuropathy did not have sufficient foundation to offer such an opinion.

Topics:

> **Physicians: Conflicting Evidence.** Where a medical expert initially opined that chlorine gas caused Petitioner's peripheral neuropathy, but the expert's trial testimony revealed that he no longer held that opinion, the

Court was not persuaded by Petitioner's argument that the chlorine gas exposure caused his peripheral neuropathy.

Occupational Disease: Causation. Although the Court found it plausible that Petitioner's exposure to various chemicals in his workplace caused him to develop peripheral neuropathy, mere plausibility is insufficient. Where the only evidence Petitioner had in this regard was the opinion of an expert witness the Court found unreliable, Petitioner did not prove that he suffered from an occupational disease.

Proof: Causation. Although the Court found it plausible that Petitioner's exposure to various chemicals in his workplace caused him to develop peripheral neuropathy, mere plausibility is insufficient. Where the only evidence Petitioner had in this regard was the opinion of an expert witness the Court found unreliable, Petitioner did not prove that he suffered from an occupational disease.

Proof: Conflicting Evidence: Medical. Petitioner initially contended that a chlorine gas exposure at work caused him to develop peripheral neuropathy. He later argued that his peripheral neuropathy was an occupational disease due to chemical exposure in his workplace. The Court was unpersuaded by Petitioner's argument that Respondent's IME panel inadequately investigated his claim by focusing only on Petitioner's chlorine gas exposure; Petitioner changed his theory only shortly before trial, and the burden of proof lay with him, not Respondent.

Proof: Burden of Proof: Causation. Petitioner initially contended that a chlorine gas exposure at work caused him to develop peripheral neuropathy. He later argued that his peripheral neuropathy was an occupational disease due to chemical exposure in his workplace. The Court was unpersuaded by Petitioner's argument that Respondent's IME panel inadequately investigated his claim by focusing only on Petitioner's chlorine gas exposure; Petitioner changed his theory only shortly before trial, and the burden of proof lay with him, not Respondent.

¶ 1 The trial in this matter began on December 18, 2014, and concluded on December 19, 2014, at the Workers' Compensation Court. Petitioner Chris Haines attended and was represented by Michele Reinhart Levine and Richard J. Martin. Steven W. Jennings and Lucas Wallace represented Respondent Montana University System Self-Funded Workers' Compensation Program (MUS). Leah Tietz, Director for MUS, also attended on MUS's behalf.

¶ 2 Exhibits: The Court admitted Exhibits 1 through 6, 8 through 18, 20 through 29, and 31 through 38 without objection. The Court held that MUS's hearsay and foundation objections to Exhibits 7, 19, and 30 were untimely and admitted the exhibits into evidence. The Court admitted Exhibit 43 after Haines introduced it at trial.

¶ 3 Witnesses and Depositions: On December 18, 2014, Chris Haines, John C. Schumpert, MD, Patrick Aaron Ryan, and Melanie Stocks were sworn and testified at trial. On December 19, 2014, George Martin Zinkhan IV, MD, Connie Hoffman, and David J. Hewitt, MD, were sworn and testified.

¶ 4 Issues Presented: The Final Pretrial Order sets forth three issues, which this Court restates as follows:

> Issue One: Whether Petitioner's January 26, 2011, industrial accident caused his peripheral neuropathy;
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> Issue Two: Whether Petitioner's exposure to chemicals as part of his regular job duties from December 2010 through August 2012 caused his peripheral neuropathy; and
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> Issue Three: Whether Petitioner is entitled to reasonable costs, attorney fees, or a penalty in accordance with § 39-71-611, MCA, and/or § 39-71-2907, MCA.

FINDINGS OF FACT[1]

¶ 5 Chris Haines testified at trial. This Court found Haines to be a credible witness.

¶ 6 From December 2010 until August 2012, Haines worked for Montana State University (MSU) as a Stage and Production Coordinator. Haines' job duties included maintaining MSU's swimming pool. Haines testified that the pool pit area was poorly ventilated and hot, and the air had a strong chemical odor. Haines testified that he was exposed to many different chemicals which were used to clean and maintain the pool, deck, and facilities. In particular, Haines explained that he was exposed to calcium hypochlorite on a daily basis and a "shock oxidizer" once or twice a month, both of which irritated his nose and throat. Haines often used a product called "pH Down" which contains sodium bisulfate. Haines also used cleaners which contained 2-Butoxyethanol and petroleum distillates. Haines testified he frequently used an aluminum brightener containing ethylene glycol monobutyl ether, sulfuric acid, ethoxylated nonylphenol, and hydrogen chloride to clean metal surfaces such as the

[1] All findings herein are taken from trial testimony except where otherwise noted.

handrails, drinking fountains, and hanging lifts. Haines also testified that he was exposed to chloramines, which occur when chlorine breaks down organic molecules.

¶ 7 On the morning of January 26, 2011,[2] Haines was directed to clean the swimming pool's chlorinating machine. MSU had not properly trained Haines on how to clean the machine and had not purchased the correct type of chemicals to clean it. Thus, when Haines put calcium hypochlorite into the machine, a chemical reaction created chlorine gas. Haines was in the area for approximately 45 minutes before he realized that he was in danger from the chemical reaction. At first, he noticed a yellowish gas in the pit area. Once the chemicals reached more water, the entire room filled with chlorine gas. The fumes became overpowering and Haines left the area and removed his respirator. He was hit with an overwhelming odor of chlorine. He put his mask back on and returned to the area. He attempted to ventilate the room and turned off the water which was feeding the reaction. MSU evacuated the building because of the chlorine gas.

¶ 8 Haines took a shower and put his same clothes back on. He then reported to Montana Occupational Health. MaryBeth Siewert, MSN, APRN, noted that Haines was exposed to chlorine gas and other chemicals from approximately 7:30 a.m. to 9:30 a.m.[3] Haines reported that his eyes were burning and that he was nauseous. On examination, Siewert noted:

> The patient presents ambulatory and appears anxious. Vital Signs: Pulse: 88. Respirations: 20. Blood Pressure: 120/68. Pulse Oximetry: 97%. Lungs are clear to auscultation. No wheezing. No adventitious breath sounds. Nasal pharynx is dark pink, and skin is intact on face. Conjunctivae are erythematous. Using a slit lamp and fluorescein, his corneas were examined and reveal bilateral chemosis. I do not appreciate any abrasions. Lab work was obtained and is normal . . . and included a CBC which is normal . . . and a complete metabolic panel was obtained, and is all within normal limits. . . . A pulmonary function test was also obtained and I was unable to get reproducible maneuvers, but the numbers were excellent and effort appeared maximum. . . .[4]

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[2] Although many of the medical records in this case represent that the date of Haines' chlorine gas exposure occurred on January 25, 2011, the parties agreed the industrial accident occurred on January 26, 2011. Therefore, the Court has used the date of January 26, 2011, throughout its findings regardless of the date in the document.

[3] Ex. 18 at 1.

[4] *Id*.

Siewert diagnosed Haines with chemical gas exposure and chemosis, meaning that the chemicals had irritated his eyes. Siewert did not note any redness or rash on Haines' skin.[5]

¶ 9 On January 27, 2011, Haines signed a First Report of Injury and Occupational Disease which stated that he suffered a burn injury to his eyes from exposure to chlorine gas on the previous day.[6] MUS accepted liability for the claim.[7]

¶ 10 On January 27, 2011, Haines returned to Montana Occupational Health. Bryan McDaniel, PA-C, examined Haines. Haines reported that he was doing "much better" and stated that he had no discomfort. McDaniel noted that Haines' chemosis had "significantly improved." McDaniel found no other complaints.[8] He released Haines to return to work without restriction.[9]

¶ 11 On February 2, 2011, Haines returned to Montana Occupational Health for a follow-up appointment. Haines reported that he was "100% better and in fact felt 100% better the night of the exposure." Siewert found him to have no residual problems and opined that he was at maximum medical improvement (MMI).[10]

¶ 12 On February 28, 2011, Jodi Borowick, Claims Assistant for Intermountain Claims, Inc., wrote to Haines and informed him that since he had not sought recent medical treatment, it appeared he had reached MMI. She advised him that he would need to seek pre-approval if he sought further treatment for his industrial injury.[11]

¶ 13 At the time of his industrial accident, Haines was an extremely active person. He skied, hiked, and participated in backpacking, shooting sports, whitewater rafting, and motocross. He described himself as "very, very fit and athletic."

¶ 14 On May 18, 2011, Haines went on a lengthy, rigorous hike in Hyalite Canyon. The next day, he had mild soreness in his Achilles tendons. Over the following weeks, his pain and soreness progressed to the point that he had trouble walking.[12]

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[5] Ex. 18 at 2.

[6] Ex. 2 at 1.

[7] *See* Ex. 5 at 1.

[8] Ex. 18 at 4.

[9] Ex. 2 at 4.

[10] Ex. 18 at 6.

[11] Ex. 5 at 1.

[12] *See* Ex. 15 at 1.

¶ 15 In June 2011, Haines presented at Bozeman Foot & Ankle Clinic, P.C.[13] He complained of a five-and-a-half-week history of acute onset persistent bilateral, equal, and symmetrical Achilles tendon pain which began after a hike and which worsened after physical therapy. William M. Wilshire, DPM, DABPS, found a severe cavus foot bilaterally. He opined that while Haines was at a high risk for Achilles tendonitis, the level of pain and progression to arch pain was uncommon for tendonitis.[14] Dr. Wilshire took Haines off work due to foot pain.[15]

¶ 16 Haines returned to work sometime prior to August 2, 2011.[16] When he returned, he worked limited hours at a "desk" job. Haines began to increase his hours and started working in the pool facilities. On August 16, 2011, Dr. Wilshire took Haines off work due to leg pain.[17]

¶ 17 Through the end of 2011, Haines saw several physicians and underwent physical therapy. Haines reported some intermittent improvement, but his symptoms spread to just below his knees. None of the physicians made a definitive diagnosis.[18]

¶ 18 On January 19, 2012, Haines saw Luke Omohundro, MD, to establish care. Dr. Omohundro took a history, noting that Haines reported feeling well until a vigorous hike on May 18, 2011, after which Haines developed mild soreness in his Achilles tendons which progressed over time until Haines had difficulty walking. The pain spread into his lower leg, mid foot, and anterior leg and at times was so severe that Haines used a wheelchair.[19] Dr. Omohundro examined Haines and noted, "Bilateral lower extremity pain: Etiology unclear. This sounds like some sort of reactive rheumatologic process to me, perhaps an enthesitis." Dr. Omohundro referred Haines to a rheumatologist.[20]

¶ 19 On February 7, 2012, Dr. Omohundro reported that when Haines saw him that day for a follow-up appointment, Haines asked whether his exposure to chlorine gas in January 2011 could account for his current leg symptoms. Dr. Omohundro noted:

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[13] The medical record for this appointment is dated January 31, 2011. At trial, Haines testified – and MUS's counsel agreed – that this appointment likely occurred in June 2011.

[14] Ex. 11 at 1-2.

[15] Ex. 11 at 5.

[16] Ex. 11 at 10.

[17] Ex. 11 at 11.

[18] *See generally* Exs. 6, 11, 12 & 31.

[19] Ex. 15 at 1.

[20] Ex. 15 at 2.

> I don't think that these 2 events are related. It was a significant exposure, but I am not aware of chlorine gas causing any sort of neuropathy or any mechanism by which it could cause leg pain. This also seems a bit remote that his symptoms began 3 months after the exposure. He does note that he has a daily exposure to these gases. The main injury I know from chlorine gas would [be] respiratory and he had really no significant respiratory symptoms or sequelae.[21]

¶ 20 On February 7, 2012, Haines saw rheumatologist John Waldron McCahan, MD. Dr. McCahan's nurse noted that Haines reported an exposure to high levels of chlorine gas in January 2011 with "severe eye burns," and that Haines developed bilateral leg pain approximately three months later.[22] After examination, Dr. McCahan found heel pain of unclear diagnosis. Since Haines reported that he was "improving" and was 70% better at that time, Dr. McCahan did not recommend any additional treatment.[23]

¶ 21 On February 17, 2012, Dr. Omohundro wrote a referral letter to Richard E. Popwell, Jr., MD, a neurologist. Dr. Omohundro asked Dr. Popwell to investigate Haines' chronic bilateral leg pain and to rule out peripheral neuropathy. He explained:

> Chris is a previously healthy avid outdoor athlete/mountain climber who has had debilitating lower extremity pain for the last 9-10 months. Chris recalls a significant chlorine gas exposure about 3 months prior to the onset of his symptoms. I think the significance of this is minimal. I have also discussed this with a toxicologist who agrees, but it is a notable exposure that occurred prior to the onset of his symptoms. His symptoms seemed to start after a very vigorous climb in May of 2011.[24]

¶ 22 On March 14, 2012, Dr. Popwell saw Haines for evaluation of his chronic refractory bilateral lower extremity pain. Dr. Popwell prompted Haines "for any clarification of his history" and reported that Haines recalled developing left Achilles pain after a strenuous hike in the spring of 2011. He developed similar symptoms on the right a few days later. The pain initially radiated into his heel and arch and eventually spread up his legs, but never above his knees. Haines reported that his symptoms had improved in the last several months, but that he had difficulty walking and standing. Haines reported redness and varying sensations of cold in his feet throughout the last year. Haines also told Dr. Popwell that he was exposed to chlorine gas approximately

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[21] Ex. 15 at 5.

[22] Ex. 10 at 2.

[23] Ex. 10 at 4.

[24] Ex. 15 at 7.

two months prior to developing his leg pain. He also reported that approximately two or three months before the onset of his pain, he had unexplained sweating of his feet.[25] Dr. Popwell diagnosed Haines with complex regional pain syndrome (CRPS), type I. He recommended not pursuing any aggressive treatment until the Mayo Clinic had the opportunity to evaluate Haines.[26]

¶ 23 Haines testified that he may not have mentioned his sweating feet to any medical provider prior to Dr. Popwell because he only thought about it as a symptom retrospectively. Haines testified that he later concluded that the sweating was a symptom of his nervous system "breaking down."

¶ 24 On March 21, 2012, Haines returned to Dr. Omohundro. Dr. Omohundro noted that Dr. Popwell had recommended that Haines get a second opinion at the Mayo Clinic.[27] Dr. Omohundro wrote a referral letter to the Mayo Clinic at Rochester, Neuromuscular Clinic.[28] He summarized Haines' history and condition as follows:

> Thank you for seeing Mr. Haines. He is a pleasant 30-year-old male who I am referring to you for chronic refractory, bilateral lower extremity pain, possible reflex sympathetic dystrophy. Chris was previously a healthy, avid, outdoor mountain climber who has had debilitating lower extremity pain over the last 9-10 months. Chris recalls a significant chlorine gas exposure about 3 months prior to the onset of symptoms. I tend to think the significance of this exposure is minimal. I have discussed the case with a toxicologist who agrees, but it is a notable exposure that occurred prior to the onset of symptoms. His symptoms seemed to start after a very vigorous climb in May of 2011. He describes pain at the Achilles tendon bilaterally. There was a hot, tearing type pain that seemed to progress and involve most of his leg and foot. At times it was quite debilitating, requiring the use of a wheel chair. Over the last few months the pain seems to be slowly improving but he still has significant pain and disability.
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> Diagnostically his workup has been unrevealing. He has had 2 complete sets of blood work . . ., 24 hour urine for heavy metal and UPEP. Additionally he has had an MRI of the lumbar spine. All of these tests were fairly normal and did not reveal an etiology. Therapeutically he has

[25] Ex. 31 at 10.

[26] Ex. 31 at 12.

[27] Ex. 15 at 9.

[28] Ex. 15 at 10-11.

> seen multiple physicians, including orthopedics, neurology, rheumatology, several naturopaths, chiropractors and myself a family practice physician. He seems to have some limited benefit from Amitriptyline. More recently I started him on Cymbalta. That has provided some significant improvement in his symptoms. He is currently taking 90 mg a day.[29]

¶ 25 On May 30, 2012, Dr. Omohundro noted that Haines had decided against the Mayo Clinic and that he preferred to pursue an evaluation at the University of Kansas. However, Haines' health insurer refused to pay for an evaluation at the University of Kansas but agreed to pay for an evaluation with George Martin Zinkhan IV, MD, at the University of Utah.[30] Dr. Omohundro wrote a referral letter to Dr. Zinkhan.[31]

¶ 26 On June 6, 2012, Dr. Popwell saw Haines for a follow-up appointment and to evaluate a recent flare of Haines' symptoms. Dr. Popwell noted:

> Christopher recently experienced a flare of his presumed reflex sympathetic dystrophy (CRPS), such being temporally associated with another exposure to gas coming from a chlorine product used for pool maintenance at his workplace. Unfortunately an exacerbation of his condition by such does not necessarily establish causation of his illness. The real question is whether or not his more remote exposure was the provocative factor of onset of his symptoms, something that I would have to defer to investigation by an occupational medical specialist or toxicologist.[32]

¶ 27 On June 20, 2012, Haines saw Dr. Zinkhan for a neurological evaluation. Dr. Zinkhan is a board-certified neurologist and an assistant professor in general neurology at the University of Utah. Dr. Zinkhan estimated that he spends approximately 80% of his time diagnosing and treating patients and 20% of his time teaching residents and medical students. Dr. Zinkhan specializes in general neurology, including conditions such as neuropathy, headache, stroke, and seizure. His specialty also includes hereditary neuropathy, neuropathy due to toxic causes, post-viral neuropathy, and autoimmune neuropathy.

¶ 28 When Dr. Zinkhan first saw Haines, he took a history, noting that Haines reported that he suffered chemical burns of the eyes and a "rash all over the body" after his

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[29] Ex. 15 at 10.

[30] Ex. 15 at 15.

[31] Ex. 15 at 16-17.

[32] Ex. 31 at 16-18.

chlorine gas exposure on January 26, 2011.[33] Haines reported that he immediately began to experience heavy sweating of both feet, which lasted for two months. Haines also reported that he began to experience pain from his heels up into his mid calves, which eventually migrated into his feet and knee. Dr. Zinkhan further noted, "2 weeks ago, the patient was working with calcium hypochlorite . . . . He noticed headache and rash in the lower legs."[34]

¶ 29 Dr. Zinkhan diagnosed Haines with peripheral neuropathy.[35] Dr. Zinkhan also took a family history from Haines. He found it notable that Haines reported no family history of neuropathy, because it is unusual for someone to have a hereditary neuropathy without a family history of neuropathy. After reviewing Haines' records, Dr. Zinkhan concluded that Haines had had a fairly thorough workup to investigate possible nontoxic causes of neuropathy. He opined, "Given his above lab work-up and the temporal relationship between the start of his symptoms and the chlorine gas exposure, the patient's peripheral neuropathy is consistent with his chlorine gas exposure."[36]

¶ 30 On June 20, 2012, Dr. Zinkhan wrote a "To Whom It May Concern" letter stating, in relevant part, "The peripheral neuropathy is due to chlorine gas exposure that he experienced on January [26], 2011."[37]

¶ 31 On July 19, 2012, Dr. Omohundro saw Haines for a follow-up appointment. Dr. Omohundro then wrote a letter recommending that Haines no longer work with pool treatment chemicals and avoid exposure to chlorine, calcium hypochlorite, or other noxious fumes. He noted, "These agents have been implicated over the course of his workup, as potential sources for his peripheral neuropathy and persistent pharyngitis.[38] Dr. Omohundro recommended that Haines work three-quarters' time. He noted that Haines suffered from "chronic bilateral lower extremity pain of unclear etiology" which might be CRPS. He further noted that Dr. Zinkhan had recently evaluated Haines and opined that his condition was a peripheral neuropathy caused by chlorine gas exposure.[39]

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[33] Contemporary medical records do not indicate that Haines presented with a rash on January 26, 2011.

[34] Ex. 21 at 1. Haines testified that he has had three flare-ups of his condition, and that each flare-up occurred after he worked with calcium hypochlorite.

[35] Ex. 21 at 4.

[36] *Id*.

[37] Ex. 21 at 5.

[38] Ex. 15 at 19.

[39] Ex. 15 at 20.

¶ 32 Connie J. Hoffman, Senior Claims Examiner for Intermountain Claims, Inc., received Dr. Zinkhan's June 20, 2012, letter via MSU. She then contacted Haines because she had not received any information regarding Haines' claim since February 2011. Haines informed Hoffman that he was having neurological problems with his feet, he had been unable to work full-time, and multiple doctors were unable to determine what was wrong with him. Haines asked MUS to pay for his medical treatment and wage loss. Hoffman obtained and reviewed Haines' medical records and found conflicting information about Haines' condition. She denied liability for Haines' leg problems pending an independent medical examination (IME).

¶ 33 MUS hired John C. Schumpert, MD, MPH, FACOEM, and David J. Hewitt, MD, MPH, to conduct the IME, which took place on August 16, 2012.[40] Drs. Schumpert and Hewitt consulted with Charles B. Anderson, MD, FAAN, a neurologist. The IME panel ruled out CRPS via a three-phase bone scan. The IME panel concluded that Haines suffered from ocular chemosis due to the chlorine gas exposure. The IME panel also concluded that Haines had peripheral neuropathy, based in part on Dr. Anderson's impression that Haines' nerve conduction studies suggested polyneuropathy.[41] Dr. Anderson noted that Haines was exposed to chlorine gas and stated, "I can't make [a] connection, but can't rule it out. Looks like mild neuropathy . . . . I would wonder about post-viral, idiopathic, autoimmune, etc."[42] Drs. Schumpert and Hewitt opined that neither Haines' chlorine gas exposure nor his calcium hypochlorite exposure caused the peripheral neuropathy. They explained that while chlorine gas causes eye, skin, and respiratory injuries, it does not cause "persistent neurological effects." They also stated, "[T]he onset of symptoms 4-5 months after the exposure incident is temporally inconsistent with a causal relationship and indicates other etiologies."[43] They suggested that the potential causes of Haines' peripheral neuropathy could be early Charcot-Marie-Tooth (CMT) syndrome, post-viral polyneuropathy, or early multiple sclerosis.[44]

¶ 34 In his report, Dr. Hewitt opined that Haines' symptoms are not secondary to exposure to chlorine gas.[45] He further noted:

> In this case, several factors indicate the individual's exposure was relatively mild: 1) the individual was wearing a respirator during the 45-minute procedure and did not notice a strong chlorine odor while wearing

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[40] Ex. 22.

[41] Ex. 23 at 4.

[42] Ex. 23 at 1.

[43] Ex. 22 at 16.

[44] Ex. 22 at 13-14.

[45] Ex. 22 at 14-15.

> the respirator; 2) the individual had eye irritation consistent with chlorine gas exposure which quickly resolved; 3) the individual had no respiratory complaints indicating minimal inhalation exposure; and 4) there was no evidence of chemical burns or other significant skin injury.[46]

¶ 35 Dr. Hewitt also opined no causal connection exists between Haines' neurological complaints and his exposure to chlorine gas and calcium hypochlorite:

> 1) neurological systemic effects are not known health effects of chlorine or calcium hypochlorite exposure; 2) chlorine exposure during the incident was relatively mild with minimal post-incident symptoms and no evidence of adverse respiratory effects; 3) the onset of symptoms 4-5 months after the exposure incident is temporally inconsistent with a causal relationship and indicates other etiologies; and 5) there are other more plausible explanations for the individual[']s neurological symptoms which have not been ruled[]out including possible Charcot-Marie-Tooth syndrome, a post-viral syndrome, or multiple sclerosis.[47]

¶ 36 At trial, Dr. Hewitt explained the process he used in evaluating Haines' claim. When he investigates causation for a possible chemical exposure, Dr. Hewitt first considers whether the chemical at issue is capable of causing the reported health effect. He then investigates whether an exposure occurred, and if so, if the exposure was sufficient to cause the reported health effect. Next, he considers whether the reported health effect occurred within the time frame expected for such an effect after the exposure, and finally he considers whether a better alternate explanation exists for the reported health effect.

¶ 37 Dr. Hewitt stated that he did not doubt that Haines experienced a chlorine gas exposure on January 26, 2011. However, Dr. Hewitt explained that he has conducted extensive research on the health effects of chlorine because of his involvement with the evaluation of several chlorine gas exposure cases, and the primary health effect of chlorine gas exposure is irritation of the eyes or mucus membranes of the throat and respiratory tract. He stated that most people recover from chlorine gas exposure unless they have a severe exposure. Dr. Hewitt stated that long-term health effects from a severe exposure are rare and are usually respiratory in nature. Dr. Hewitt could not find any support in medical literature for Haines' contention that chlorine gas caused his peripheral neuropathy. Dr. Haines testified that peripheral neuropathy has not been

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[46] Ex. 22 at 16.

[47] Ex. 22 at 16-17.

reported as a health effect from chlorine gas exposure, in spite of a significant body of research which has examined the effects of chlorine gas exposure.

¶ 38 After Hoffman reviewed the IME report, she denied Haines' claim because she believed that the report, in conjunction with the records from the various medical providers Haines had treated with in Bozeman, did not establish that chlorine gas exposure caused Haines' peripheral neuropathy.

¶ 39 On September 20, 2012, Dr. Zinkhan saw Haines for a follow-up appointment. Dr. Zinkhan reported, "No high arches or peripheral neuropathy in the family."[48] Dr. Zinkhan further testified that high arches are common in people who have hereditary neuropathy, so he asked about Haines' arches to determine if he had that symptom. Dr. Zinkhan testified that the reason why he noted that Haines had no history of high arches or peripheral neuropathy in his family was because he wanted to confirm this information. He noted that sometimes, a patient will talk to family members after an appointment and will learn something new.

¶ 40 Dr. Zinkhan further noted that an EMG/NCS showed evidence of sensorimotor demyelinating peripheral neuropathy, but that the slower conduction velocity could be due to Haines' height. He stated:

> I looked up chlorine gas and calcium hypochlorite today in Pubmed and could find reports of peripheral neuropathy associated with these toxins. The patient's neuropathic pain however started after exposure to these chemicals and has improved since exposure stopped. This suggests a correlation between the neuropathic pain and chemicals.[49]

However, Dr. Zinkhan testified that this record contains a typographical error, as he actually did ***not*** find reports of peripheral neuropathy associated with chlorine gas or calcium hypochlorite.

¶ 41 On October 4, 2012, Haines entered into a separation agreement with MSU, ending his employment because his physical limitations prevented him from performing the essential functions of his job.[50]

¶ 42 On October 29, 2012, Drs. Schumpert and Hewitt wrote to Hoffman in response to her letter and in response to Dr. Zinkhan's September 20, 2012, medical records. They stated that they could not locate any articles supporting Dr. Zinkhan's statement

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[48] Ex. 21 at 8.

[49] Ex. 21 at 10.

[50] Ex. 27 at 1.

that peripheral neuropathy has been associated with chlorine gas or calcium hypochlorite.[51]

¶ 43 On November 12, 2012, Haines saw Maryam Tahmasbi Sohi, MD, and Mazen Dimachkie, MD, FAAN, neurologists at the University of Kansas Medical Center, for a second opinion. In the report, Dr. Dimachkie noted that "both his parents have high arches and when I inspected his parents, [e]specially [h]is mother does strike me as someone with high arches and patient has lifelong history of high arches."[52] Dr. Tahmasbi Sohi noted, "History also significant for high arches in family members: mother, father and brother – everybody in family has remained asymptomatic."[53] Dr. Dimachkie also noted that Haines exhibited bilateral high arches and mild hammering of the left big toe.[54]

¶ 44 Dr. Dimachkie's impression was that Haines had two distinct issues: (1) a longstanding genetic neuropathy that accounts for his lifelong high arches, alternatively due to orthopedic bony changes involving peripheral neuropathy of genetic type; and (2) acute pain affecting his heels and lower Achilles area which "sounds musculoskeletal." Dr. Dimachkie suggested MRIs of Haines' ankles and provided him with educational information on peripheral neuropathy and on CMT.[55] In the diagnosis section of their report, Drs. Tahmasbi Sohi and Dimachkie wrote, "HEREDITARY PERIPHERAL NEUROPATHY."[56] In an addendum note, Dr. Dimachkie reported that an EMG nerve conduction study showed no electrophysiologic evidence for CMT.[57]

¶ 45 While at the University of Kansas, Haines asked about "the role of the about three months[']] exposure to chlorine and calcium chlorite at work."[58] Dr. Tahmasbi Sohi's report further stated: "A brief review of available articles in Pub med and Washington University Neuromuscular home page did not reveal any reported cases of neuropathy caused by chlorine and calcium hypochlorite. This is was [sic] communicated with the patient and his family."[59]

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[51] Ex. 22 at 26-27.

[52] Ex. 20 at 10.

[53] Ex. 20 at 5.

[54] Ex. 20 at 10.

[55] *Id.*

[56] Ex. 20 at 14 (capitalization in original).

[57] Ex. 20 at 10.

[58] Ex. 20 at 5.

[59] Ex. 20 at 9.

¶ 46 On November 16, 2012, Dr. Omohundro noted that Haines had undergone a "very extensive" workup at the University of Kansas and that physicians there had diagnosed Haines with a hereditary polyneuropathy. Dr. Omohundro explained, "The consensus was he had some genetic vulnerabilities including high arch, which led to an overuse injury."[60] Dr. Omohundro also noted, "They did not think it was [due] to the chlorine or calcium hyperchloride [sic] exposure." Dr. Omohundro indicated that he intended to follow the recommendations of the University of Kansas physicians and would refer Haines to a physical medicine and rehabilitation physician.[61]

¶ 47 On January 4, 2013, Dr. Dimachkie wrote to Haines regarding "corrections" Haines had sent to the University of Kansas physicians. Dr. Dimachkie noted that Haines added that he had been "hospitalized after a 19-month exposure to chlorine and calcium chloride at work,"[62] and that Haines had suffered a chlorine gas accident, and had also been exposed to hydrochloric acid. Dr. Dimachkie further stated, "Surprisingly, the EMG/nerve conduction study did not show any evidence of hereditary neuropathy. One step that could be worthwhile pursuing to further evaluate you for the possibility of genetic neuropathy would be to obtain the test through Athena Diagnostics."[63] Haines testified that he has not undergone this test due to its cost.

¶ 48 Dr. Zinkhan also reviewed the medical records from the University of Kansas. Dr. Zinkhan noted that the physicians saw no electrophysiologic evidence for CMT from an EMG nerve conduction study, and that he considered this study an appropriate way to rule out CMT. He opined that genetic testing would be expensive and unnecessary to further rule out CMT, because Haines had almost no chance of having the condition, based on his understanding of Haines' family history and the EMG test results.

¶ 49 Drs. Hewitt and Schumpert reviewed the medical records from the University of Kansas. At trial, Dr. Schumpert testified that although the University of Kansas physicians found no evidence of CMT from the EMG testing, they recommended further genetic testing. Dr. Schumpert observed that this recommendation means the University of Kansas physicians did not rule out CMT as a possible diagnosis. Dr. Hewitt also testified that while the EMG nerve conduction study did not show evidence of CMT, this test alone does not rule out the possibility that Haines has CMT.

¶ 50 On August 1, 2013, Haines filed a First Report of Injury or Occupational Disease in which he described the accident as "EXPOSURE TO CHLORINE GAS ON

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[60] Ex. 15 at 25.

[61] *Id.*

[62] The Court has not seen any medical records which corroborate this statement.

[63] Ex. 20 at 15-16.

1/25/2011, OR IN THE ALTERNATIVE, DAILY EXPOSURE TO CHLORINE GAS FROM 12/7/2010 THROUGH 8/7/2012." He indicated that the part of body affected was "MULTIPLE BODY PARTS (LEGS, ARMS, MIND)" and that the nature of the disease was bilateral polyneuropathy of the legs and post-traumatic stress disorder.[64]

¶ 51 On August 20, 2013, Dr. Zinkhan wrote a letter stating he had reviewed Drs. Schumpert's and Hewitt's August 16, 2012, IME report and that he disagreed with the finding of a lack of temporal relationship between exposure and symptoms. Dr. Zinkhan explained:

> The patient had initial exposure on January 25, 2011. He continued to have exposure to the chlorine gas through July 2012. At his appointment on June 12, 2012, the patient reported that 2 weeks prior, he was working with calcium hypochlorite from chlorinator pump. He was sitting next to it 2-4 hours per day a few days per week. He noticed headache and rash in the lower legs when working with it. At the time he was working 35% less. I think the headache and rash would indicate that he was having toxic exposure at that time. I think that given the temporal relationship between symptom onset with chronic exposure and the onset of the pain in his legs that it would be reasonable that the chronic exposure to chlorine was related to the onset of his symptoms.[65]

¶ 52 On September 3, 2013, Haines' counsel sent a letter demanding that MUS accept liability for his condition as either an injury or an occupational disease claim. Haines' counsel wrote, in relevant part, "Attached please find Dr. Zinkhan's response (dated 8/20/13) to the IME report dated 8/16/12. Dr. Zinkhan notes that Mr. Haines' chronic exposure to chlorine gas while working at MSU's pool facility was related to the onset of Mr. Haines' symptoms."[66]

¶ 53 By letter dated September 4, 2013, Hoffman advised Haines that MUS would not cover additional medical treatment for his injury claim since Drs. Schumpert and Hewitt had determined that his symptoms were not related to his industrial injury.[67]

¶ 54 On September 20, 2013, Hoffman wrote to Haines and stated that MUS was denying his occupational disease claim, as MUS had been "unable to substantiate compensability."[68]

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[64] Ex. 4.

[65] Ex. 21 at 14.

[66] Ex. 5 at 2.

[67] Ex. 5 at 3.

¶ 55 On November 5, 2013, in response to questions posed by Haines' counsel, Dr. Zinkhan again opined that the January 26, 2011, chlorine gas exposure caused Haines' peripheral neuropathy.[69] Dr. Zinkhan explained:

> By history his symptoms started on the day that he was exposed to chlorine gas. He had chemical burns of the eyes and rash all over the body. He started to have heavy sweating in both feet. This lasted 4 months. He then developed severe pain in the legs that has limited his ability to work.[70]

¶ 56 In response to a question as to whether Haines had an occupational disease under Montana law, Dr. Zinkhan opined that Haines' ongoing exposure to chlorine gas at work from January 2011 through August 2012 was the major contributing cause of his neuropathy. He wrote:

> Work contributed 40-100%. The symptoms started and worsened during his exposure. He may also have Charcot-Marie Toothe [sic] neuropathy which is hereditary. I would estimate this may contribute 0-30%. He had high vitamin B which may contribute 30% although I think the B supplement was started after onset.
>
> . . . .
>
> B complex was started after symptom onset. He had a high vitamin B6 which may contribute to neuropathy. I think the other causes of neuropathy were ruled out in labs. . . . There were no significant abnormalities. MRI Lumbar spine showed no significant abnormality. EMG and nerve conduction studies showed mild distal slowing which is indicative of neuropathy.[71]

Although Dr. Zinkhan attributed Haines' neuropathy to his exposure to chlorine gas, Dr. Zinkhan answered a question asking for "citations to the medical studies which connect chlorine gas exposure or similar toxins with peripheral neuropathy," by writing, "There are none."[72] At that time, Dr. Zinkhan attributed Haines' peripheral neuropathy to chlorine gas and no other chemical.

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[68] Ex. 5 at 4.

[69] Ex. 21 at 15.

[70] Ex. 21 at 16.

[71] *Id.*

[72] *Id.*

¶ 57 On February 11, 2014, in response to an inquiry from Haines' counsel requesting scientific literature connecting Haines' condition to chlorine exposure, Dr. Zinkhan replied that he found two studies describing neurological deficits in connection with chlorine inhalation exposure. Dr. Zinkhan acknowledged that he did not read the full articles for either study; rather, he only reviewed the abstracts.[73] Dr. Zinkhan also cited several references which he believed might support his opinions.[74] Dr. Zinkhan testified that these studies do not specifically discuss peripheral neuropathy, but show that the patients examined had an "imbalance" which could be due either to peripheral neuropathy or a central nervous system cause.

¶ 58 On March 11, 2014, Drs. Schumpert and Hewitt wrote to Hoffman in response to her recent communication regarding Dr. Zinkhan's February 11, 2014, correspondence. Drs. Schumpert and Hewitt advised Hoffman that they had obtained complete copies of the articles of which Dr. Zinkhan had relied upon only the abstracts. After reading the articles, they concluded that none were relevant to Haines' situation. Three of the articles found no peripheral neuropathies as a result of chlorine gas exposure, and a fourth described peripheral neuropathy in individuals who had been exposed to trichloropropane and dichlorohydrin, which are industrial solvents. Drs. Schumpert and Hewitt indicated that the articles and Dr. Zinkhan's opinions did not change their opinions as expressed in the IME report.[75]

¶ 59 On October 3, 2014, Haines' counsel wrote to Dr. Zinkhan and asked his opinion about whether chemicals other than chlorine and calcium hypochlorite could have caused Haines' neuropathy. Haines' counsel stated:

> In addition to his severe chemical exposure, including chlorine gas, on January 26, 2011, Mr. Haines had cumulative daily chemical exposures to numerous chemicals in a MSU swimming pool basement maintenance room with poor ventilation and high temperatures often over 100 degrees Fahrenheit from January 2011 through August 2012.[76]

Haines' counsel then set forth a list of over 40 chemicals Haines alleged he was exposed to in his work environment, including a phenol and anti-freeze (propylene or ethylene glycol).[77] Haines' counsel also provided Material Safety Data Sheets for many of the chemicals and set forth additional information about the frequency and means by

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[73] Ex. 21 at 24.

[74] Ex. 21 at 25.

[75] Ex. 22 at 28-30.

[76] Ex. 21 at 100.

[77] *Id.*

which Haines was allegedly exposed to several of the chemicals listed.[78] Haines' counsel asked Dr. Zinkhan to opine whether Haines had toxic neuropathy and "whether his occupational exposure to chemicals at MSU is the 'major contributing cause' of his neuropathy."[79]

¶ 60 Dr. Zinkhan responded on October 20, 2014, and opined that non-occupational factors unlikely played a major role in Haines' condition. Dr. Zinkhan reiterated that it was his opinion that chlorine gas exposure caused Haines' peripheral neuropathy:

> I think that the fact that his symptoms started immediately after [exposure] with autonomic symptoms of sweating in the feet followed by pain in the feet 4 months later would be most consistent with the chlorine gas causing his symptoms.[80]

Dr. Zinkhan did not comment about the other chemicals listed in the October 3, 2014, letter.

¶ 61 However, less than 60 days later, Dr. Zinkhan testified at trial that he no longer believes that chlorine gas or calcium hypochlorite exposure caused Haines' peripheral neuropathy. On cross-examination, he agreed that "there's no clear evidence of chlorine gas being associated with peripheral neuropathy." He also agreed that he found no studies linking calcium hypochlorite to peripheral neuropathy. He testified that it was "[his] position today that Chris' peripheral neuropathy is caused by a chemical other than chlorine gas or calcium hypochlorite." Dr. Zinkhan testified that he now believes, on a more probable than not basis, "That Chris had developed toxic peripheral neuropathy due to the toxins that he was exposed to and the cleaning agents, and then the chemicals he was using with the pool." He testified that he found it significant that Haines reported improvement in his symptoms when his exposure to chemicals in his workplace stopped.

¶ 62 Dr. Zinkhan also testified that when he compared a list of the chemicals Haines used in maintaining the swimming pool to a list of chemicals associated with peripheral neuropathy in an article from the *Journal of American College of Occupational and Environmental Medicine*, he found ethylene glycol and a phenol on both lists. Dr. Zinkhan noted that Haines used an aluminum brightener that contained ethylene glycol monobutyl ether and ethoxylated nonylphenol.[81] When asked on cross-

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[78] Ex. 21 at 101-02.

[79] Ex. 21 at 102.

[80] Ex. 21 at 99-103.

[81] *See* Ex. 25 at 82.

examination to identify which chemical caused Haines' peripheral neuropathy, Dr. Zinkhan responded, "I think any one of the chemicals we discussed earlier." Dr. Zinkhan was then asked, "Specifically which chemical, Doctor?" He responded that it was either the phenol, the ethylene glycol or a "couple others." Dr. Zinkhan then testified that trichloropropane or dichlorohydrin "might be" the chemicals that caused Haines' peripheral neuropathy. Notwithstanding, he admitted that he did not know how often or to what extent Haines was exposed to these chemicals.

¶ 63 Irrespective of whether Dr. Zinkhan is Haines' treating physician, this Court does not find Dr. Zinkhan's initial opinion that Haines' January 26, 2011, chlorine gas and calcium hypochlorite exposure caused Haines' peripheral neuropathy credible or persuasive because there is insufficient foundation to support it. Neither Dr. Zinkhan nor the IME panel found any literature which indicates that chlorine gas or calcium hypochlorite exposure can cause peripheral neuropathy. Although physicians do not need to support every causation opinion with a medical study,[82] this Court was persuaded by Dr. Hewitt's testimony that the health effects of chlorine gas and calcium hypochlorite have been thoroughly studied, are well-known, and that these chemicals do not cause peripheral neuropathy. Dr. Tahmasbi Sohi's and Dr. Dimachkie's records indicate that they did not believe Haines' exposure to chlorine gas or calcium hypochlorite was of any consequence to his peripheral neuropathy. Dr. Omohundro's records reflect that he did not believe Haines' exposure to chlorine gas or calcium hypochlorite caused his peripheral neuropathy. The journal article on which Haines relies contains a list of nearly 100 chemicals that are associated with toxic peripheral neuropathy, but neither chlorine gas nor calcium hypochlorite are on the list.[83] It appears that Dr. Zinkhan abandoned his initial opinion that Haines' chlorine gas exposure caused his peripheral neuropathy in the weeks before trial because it was unsupportable.

¶ 64 Dr. Zinkhan based his initial opinion largely on the temporal relationship he saw between Haines' chlorine gas exposure of January 26, 2011, and his symptoms, including Haines' sweaty feet. However, a temporal relationship is insufficient by itself to prove causation.[84] Moreover, if the chlorine gas exposure on January 26, 2011, did not cause Haines' peripheral neuropathy, then the alleged temporal relationship between the industrial accident and the onset of Haines' sweating is meaningless.

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[82] *See Montana State Fund v. Grande*, 2012 MT 67, ¶¶ 41-46, 364 Mont. 333, 274 P.3d 728 (holding that while a rheumatologist's opinion that truck driving contributed to claimant's arthritis was not supported by any "literature," her opinion was persuasive because it was based upon her experience treating arthritis and her knowledge that truck driving involves repetitive hand use).

[83] Ex. 30 at 7.

[84] *See Pasha v. Nat'l Union Fire of Pittsburgh*, 1997 MTWCC 5, ¶¶ 56-63.

¶ 65 Since there was little to no basis for Dr. Zinkhan's initial opinion that Haines' January 26, 2011, exposure to chlorine gas caused his peripheral neuropathy, and since Dr. Zinkhan disavowed this opinion at trial, this Court ascribes it no weight.

¶ 66 Furthermore, this Court did not find Dr. Zinkhan's opinion that Haines' exposure to other chemicals, including chloramines and other disinfection byproducts, caused his peripheral neuropathy credible or persuasive because this opinion lacks a sufficient foundation and because he did not offer sufficient explanation as to why his opinion changed in the weeks before trial. Dr. Zinkhan did not have any specific knowledge about the dose or duration of Haines' exposures to chemicals that have been associated with peripheral neuropathy other than the list of chemicals provided by Haines' counsel and Haines' reports that he used products containing some of these chemicals as part of his job duties. Although Haines testified that he used the aluminum brightener that contained a phenol and ethylene glycol "quite a bit," this does not quantify Haines' exposure in any meaningful way.

¶ 67 Dr. Zinkhan also testified that trichloropropane or dichlorohydrin "might be" the chemicals that caused Haines's peripheral neuropathy, but there is no evidence as to the amounts of these chemicals in Haines' workplace. Furthermore, speculation that something "might be" the cause of a claimant's condition is a mere possibility; it does not mean that it is more probable than not that these chemicals caused Haines' peripheral neuropathy.

¶ 68 Although this Court recognizes that toxic neuropathy can be a diagnosis of exclusion,[85] it is not persuaded by Dr. Zinkhan's opinion that all other potential causes of Haines' peripheral neuropathy have been ruled out. Dr. Zinkhan relied on the EMG results and Haines' alleged lack of high arches to rule out hereditary neuropathy. However, the Court heard no explanation as to why Dr. Zinkhan did not find Haines to have high arches when Drs. Wilshire, Dimachkie, and Tahmasbi Sohi reached the opposite conclusion upon examination. Although Dr. Zinkhan relied upon Haines' increased sweating, he testified that while sweating of the feet can be present in toxic neuropathy, it is typically more common in patients with hereditary neuropathy. While Dr. Dimachkie said that the EMG/nerve conduction studies did not show evidence of hereditary neuropathy, he offered additional testing "to further evaluate [Haines] for the possibility of genetic neuropathy,"[86] which, as noted by Drs. Schumpert and Hewitt, indicates that Dr. Dimachkie had not conclusively ruled out hereditary neuropathy. Faced both with evidence indicating that Haines' peripheral neuropathy may be hereditary, that neuropathy can sometimes be idiopathic, and a lack of foundation for

[85] Ex. 30 at 6.

[86] Ex. 20 at 15.

Dr. Zinkhan's opinion that chemical exposures caused Haines' peripheral neuropathy, the Court cannot find it more probable than not that Haines' peripheral neuropathy is a toxic neuropathy.

CONCLUSIONS OF LAW

¶ 69 Haines' injury claim is governed by the 2009 version of the Workers' Compensation Act (WCA) since that was the law in effect at the time of Haines' industrial accident.[87] His occupational disease claim is governed by the 2011 version of the WCA since that was the law in effect on his last day of employment and, consequently, his alleged last injurious exposure.[88]

¶ 70 Haines bears the burden of proving by a preponderance of the evidence that he is entitled to the benefits he seeks.[89]

Issue One: Whether Petitioner's January 26, 2011, industrial accident caused his peripheral neuropathy.

¶ 71 Causation is an essential element of an entitlement to workers' compensation benefits. The claimant has the burden of proving a causal connection by a preponderance of the evidence.[90] Under § 39-71-407(2), MCA, an insurer is liable for an injury if the injury is established by objective medical findings and the claimant establishes that it is more probable than not that the claimed injury occurred or aggravated a pre-existing condition. A worker is required to prove causation through medical expertise or opinion.[91]

¶ 72 Although Haines argues that the January 26, 2011, industrial accident caused his peripheral neuropathy, the medical evidence presented to the Court established that chlorine gas does not cause peripheral neuropathy. Although Dr. Zinkhan initially opined that Haines' January 26, 2011, chlorine gas exposure caused the peripheral neuropathy, this Court did not find his initial opinion credible or persuasive. From Dr. Zinkhan's trial testimony, it is apparent that Dr. Zinkhan no longer holds this opinion.

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[87] *Ford v. Sentry Cas. Co.*, 2012 MT 156, ¶ 32, 365 Mont. 405, 282 P.3d 687; § 1-2-201, MCA.

[88] *Hardgrove v. Transp. Ins. Co.*, 2004 MT 340, ¶ 2, 324 Mont. 238, 103 P.3d 999 (citing *Grenz v. Fire & Cas. of Conn.*, 278 Mont. 268, 272, 924 P.2d 264, 267 (1996)); *Nelson v. Cenex, Inc*., 2008 MT 108, ¶ 33, 342 Mont. 371, 181 P.3d 619.

[89] *Ricks v. Teslow Consol.*, 162 Mont. 469, 483-84, 512 P.2d 1304, 1312-13 (1973); *Dumont v. Wickens Bros. Constr. Co*., 183 Mont. 190, 201, 598 P.2d 1099, 1105-06 (1979).

[90] *Taylor v. Montana State Fund*, 2012 MTWCC 17, ¶ 70 (citing *Grenz v. Fire & Cas. of Conn.*, 250 Mont. 373, 380, 820 P.2d 742, 746 (1991)).

[91] *Ford*, 2012 MT 156, ¶¶ 44-49.

No other physician remains of the opinion that Haines' January 26, 2011, chlorine gas exposure caused his peripheral neuropathy. Likewise, although calcium hypochlorite irritated Haines, no reliable medical evidence indicates that it causes peripheral neuropathy. Haines has therefore not proven that it is more probable than not that the January 26, 2011, industrial accident caused his peripheral neuropathy.

¶ 73 From the evidence presented, the Court found that Haines suffered from chemosis as a result of the January 26, 2011, industrial accident, that he reached MMI with no permanent impairment, and that, at this time, he does not need any additional treatment. Therefore, at this time, MUS bears no further liability for Haines' industrial accident.

Issue Two: Whether Petitioner's exposure to chemicals as part of his regular job duties from December 2010 through August 2012 caused his peripheral neuropathy.

¶ 74 Section 39-71-116(23)(a), MCA, defines "occupational disease" as "harm, damage, or death arising out of or contracted in the course and scope of employment caused by events occurring on more than a single day or work shift." Section 39-71-407(12), MCA, states:

> An insurer is liable for an occupational disease only if the occupational disease:
>
> (a) is established by objective medical findings; and
>
> (b) arises out of or is contracted in the course and scope of employment. An occupational disease is considered to arise out of or be contracted in the course and scope of employment if the events occurring on more than a single day or work shift are the major contributing cause of the occupational disease in relation to other factors contributing to the occupational disease.

¶ 75 Section 39-71-407(16), MCA, defines "major contributing cause" as "a cause that is the leading cause contributing to the result when compared to all other contributing causes."

¶ 76 In *Grande v. Montana State Fund*, this Court held that for a condition to be compensable as an occupational disease, the worker's job duties need not be the only contributing factor, but per § 39-71-407(16), MCA, must be the cause that ranks first among all causes.[92] This Court explained, "a 'leading cause' under the statute is that

[92] *Grande*, 2011 MTWCC 15, ¶¶ 29-31 (citing § 39-71-407(13), MCA (2009)), *aff'd*, 2012 MT 67, 364 Mont. 333, 274 P.3d 728.

cause which ranks first among all causes 'contributing to the result' – i.e., the condition for which benefits are sought – regardless of the respective percentages of the multiple contributing causes."[93]

¶ 77 Haines contends that his workplace exposure to chemicals was the leading cause, if not the only cause, of his peripheral neuropathy. Although Haines' contention is plausible since he used some products containing chemicals associated with peripheral neuropathy, mere plausibility is insufficient. Haines must prove that it is more probable than not that his workplace exposure was the leading cause of his peripheral neuropathy. Haines' only evidence in this regard is the testimony of Dr. Zinkhan, which the Court found unreliable and unpersuasive.

¶ 78 Although Haines argued that MUS inadequately investigated his claim by not asking Drs. Schumpert and Hewitt to explore the possibility that a workplace exposure to chemicals other than chlorine gas or calcium hypochlorite caused his peripheral neuropathy, the burden of proof lies with Haines. In a previous case with three potential causes of a claimant's condition, this Court explained, "It is not the insurer's burden to affirmatively establish a cause other than the work as the more likely of the three possibilities: it is the claimant's burden to affirmatively establish that her work was likely the cause of her condition."[94] Moreover, this Court cannot fault MUS for limiting its investigation to chlorine gas and calcium hypochlorite, as Haines' position until shortly before trial was that these chemicals caused his peripheral neuropathy.

¶ 79 Haines has failed to meet his burden of proving that his exposure to chemicals as part of his job duties is the major contributing cause of his peripheral neuropathy. Therefore, the Court concludes that MUS is not liable for his alleged occupational disease.

Issue Three: Whether Petitioner is entitled to reasonable costs, attorney fees, or a penalty in accordance with § 39-71-611, MCA, and/or § 39-71-2907, MCA.

¶ 80 Since Haines is not the prevailing party, he is not entitled to his costs, attorney fees, or a penalty under §§ 39-71-611, and -2907, MCA.

JUDGMENT

¶ 81 Petitioner's January 26, 2011, industrial accident did not cause his peripheral neuropathy.

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[93] *Grande*, 2011 MTWCC 15, ¶ 31.

[94] *Christensen v. Rosauer's Supermarkets*, *Inc*., 2003 MTWCC 62, ¶ 26.

¶ 82 Petitioner's exposure to chemicals as part of his regular job duties from December 2010 through August 2012 did not cause his peripheral neuropathy.

¶ 83 Petitioner is not entitled to reasonable costs, attorney fees, or a penalty in accordance with § 39-71-611, MCA, and/or § 39-71-2907, MCA.

¶ 84 Pursuant to ARM 24.5.348(2), this Judgment is certified as final and, for purposes of appeal, shall be considered as a notice of entry of judgment.

DATED this 9th day of June, 2015.

(SEAL)

/s/ DAVID M. SANDLER
JUDGE

c: Michele Reinhart Levine
Steven W. Jennings

Submitted: January 27, 2015