BEFORE THE INDUSTRIAL COMMISSION OF THE STATE OF IDAHO

JOSE MENDOZA-ZAPATA, Claimant, v. EAGLE RIDGE DAIRY, LLC, Employer, and STATE INSURANCE FUND, Surety, Defendants.	**IC 2015-002206** **FINDINGS OF FACT, CONCLUSIONS OF LAW, AND ORDER** **Filed February 20, 2020**

INTRODUCTION

Pursuant to Idaho Code § 72-506, the Idaho Industrial Commission assigned the above-entitled matter to Referee Alan Taylor, who conducted a hearing in Boise on May 23, 2018. Claimant, Jose Mendoza-Zapata, was present in person and represented by Justin P. Aylsworth, of Boise. Defendant Employer, Eagle Ridge Dairy, LLC (Eagle Ridge), and Defendant Surety, State Insurance Fund, were represented by Neil D. McFeeley, of Boise. The parties presented oral and documentary evidence. Post-hearing depositions were taken and briefs were later submitted. The matter came under advisement on May 1, 2019. The Commission has reviewed Referee Taylor's proposed findings of fact, conclusions of law, and recommendation, but declines to adopt the same. The Referee determined that Claimant's chemical exposure caused his GBS or GBS-like syndrome. As explained *infra*, the Commission disagrees that the record provides sufficient proof of this proposition, but supports the Referee's ultimate decision for other reasons.

ISSUES

The issues to be decided are:

1. Whether Claimant suffered an injury from an accident arising out of and in the course of employment.
2. Whether the condition for which Claimant seeks benefits was caused by the industrial accident.
3. Whether Claimant's condition is due in whole or in part to a pre-existing and/or subsequent injury/condition.
4. Whether, and to what extent, Claimant is entitled to medical care.

All other issues are reserved.

CONTENTIONS OF THE PARTIES

Claimant asserts he was exposed to hazardous chemicals while working at Employer's dairy resulting in respiratory injuries and/or causing Guillain-Barre syndrome requiring extensive medical treatment including prolonged hospitalization. He seeks medical benefits for treatment thereof. Defendants deny Claimant's exposure to chemicals at work caused him injury or Guillain-Barre syndrome. Defendants assert his Guillain-Barre syndrome is due to other factors for which they are not liable.

EVIDENCE CONSIDERED

The record in this matter consists of the following:

1. The Industrial Commission legal file;
2. The pre-hearing deposition testimony of Claimant and the pre-hearing deposition of Christopher Gale Rood;
3. Claimant's Exhibits 1 through 17 and Defendants' Exhibits A through I, admitted at the hearing;

4. Claimant's testimony taken at hearing;

5. The revised post-hearing deposition testimony of Robert H. Friedman, M.D., taken by Claimant on July 24, 2018;

6. The post-hearing deposition testimony of Robin Albert Dodson, B.S., B.S. Pharmacy, PH. D., taken by Claimant on July 31, 2018;

7. The post-hearing deposition testimony of Emil J. Bardana, M.D., taken by Defendants on October 24, 2018; and

8. The post-hearing deposition testimony of Gary Dawson, Ph.D., taken by Defendants on November 1, 2018.

All pending objections are overruled and motions to strike are denied, except for Defendant's objection to Dr. Dodson's deposition changes, treated *infra*. (See Defendant's reply brief at 17).

FINDINGS OF FACT

1. Claimant was born in 1977. He was 40 years old and lived in Nampa at the time of the hearing. He is a Mexican citizen and undocumented. Eagle Ridge is a dairy business with operations in Kuna.

2. **Background.** Claimant was born in Mexico and raised in Guadalupe. He does not read English and speaks very little English. He testified at hearing only through an interpreter.

3. Claimant worked on his father's fields in Guadalupe cultivating corn and beans. He was healthy and never required any hospitalization prior to 2015.

4. In 2014, Claimant lived and worked in Guadalupe. He does not recall being near or being bitten by any mosquitoes in 2014. He does not recall any friend or family member being bitten by any mosquitoes in 2014.

5. In December 2014, Claimant came to Idaho. He had never had a flu shot. He had no breathing problems, lung issues, or chemical sensitivities prior to January 5, 2015.

6. In late December 2014 or early January 2015, Claimant worked for three days at a dairy in Nampa. His work required no use of chemicals and he had no breathing problems or muscle weakness.

7. On January 3, 2015, Claimant began working at Eagle Ridge Dairy. His duties included milking cows and cleaning milking lines and other equipment.

8. **Industrial incident.** On January 5, 2015, Claimant's coworkers first tasked him with cleaning milking lines at work. To do so, he used a pitcher to repeatedly transfer soaps and acids from large containers into a vat of boiling water and then into the milking lines to flush and clean them. Thereafter, approximately every other day Claimant was assigned to clean the milking lines. The chemicals he used included T-Chlor 12.5% (containing Sodium Hydrochlorite, Clorox, and Sodium Hydroxide), U-2000 (a mixture of Phosphoric Acid, Nitric Acid, and Sulfuric Acid), Excel Final Oxy (a blend of Sulfuric Acid, Hydrogen Peroxide, and Methane Sulfonic Acid), and CC-5,000 (a mixture containing Sodium Hydroxide, Sodium Carbonate and Sodium Dichloroisocyanurate). The chemical mixtures splashed on his arms and he was extensively exposed to vapors from the hot vat into which he poured the cleaning chemicals. Claimant was not instructed to use protective gear, not provided protective gear, and used no breathing, facial, or other protective gear while cleaning the milking lines, with the exception of gloves. Claimant noticed that on the days he cleaned the milking lines, his face, hands and arms itched and he felt ill. On days he did not clean the milking lines, he had no such symptoms.

9. On January 15, 2015, Claimant was again tasked with cleaning the milking lines. Over the course of his work day, he lost strength in his hands and arms such that his coworkers

had to complete his assigned duties and after his work shift a coworker took him to the hospital. Claimant described his symptoms at that time as: "pressure in my chest and that I was sort of choking." When asked by Defendants' counsel if Claimant felt like he had a cold or the flu, he responded: "No. …. I had a lot of loss of strength in my arms …. I was losing my speech. My nose was itching, I had a headache. My eyes were burning. And that's why I went to the hospital." Claimant's Depo., 32:18 33:2.

10. Claimant presented at St. Alphonsus Medical Center in Nampa at 10:55 p.m. on January 15, 2015, and was attended by LeAnna Rankin, R.N., who recorded: "Woke up this morning with sore throat, now c/o neck and head pain as well as shakiness in his hands." Exhibit 5, p. 40. Claimant's rapid strep test was negative and his temperature was 98.2. He was examined by Mark Burriesci, M.D., who recorded:

> The patient presents with sore throat and hurts to swallow, worse throughout today. Ears achy bilate [sic] resolved. No runny nose. The onset was gradual and today since this am. Course/duration of symptoms is constant. Location: Bilateral throat. The degree at present is moderate. Therapy today: Associated symptoms: denies fever. Additional history: nothing improves, worse with swallowing, eating/drinking. … Review of Symptoms … No fever, no chills … nausea … Muscle pain, upper arms, achy. Neurologic symptoms: Headache, dizziness ….
>
> Throat: Bilateral, moderate, pharynx, erythema, tiny amount of exudate on right tonsil. …. Lungs are clear to auscultation.
>
> Differential Diagnosis: Otitis media, upper respiratory infection, pharyngitis, viral illness.
> ….
>
> Diagnosis: Pharyngitis … Myalgias viral illness.

Exhibit 5, pp. 42-44. A slightly different note from the same visit provided: "denies fever. …. Respiratory symptoms: Shortness of breath, no cough." Exhibit 5, p. 45. Claimant was given Decadron and a prescription and released at approximately 12:19 a.m. on January 16, 2015.

11. It is unclear to what extent interpreter services were utilized. Medical records indicate patient history was obtained via an interpreter; however, Claimant testified no interpreter was utilized. The patient history contains no mention of any chemical exposure.

12. Claimant returned home and his symptoms worsened. After several hours of trying to sleep, Claimant awoke unable to eat or swallow and called his cousin who drove him back to the hospital.

13. On January 16, 2015, at approximately 2:13 p.m., Claimant presented at the emergency room where Amy Bowlin, R.N., recorded at triage: "Onset of chief complaint: sore throat with shortness of breath … possible allergic reaction, face swelling, tongue swelling, inhaled some acid at work, seen here yesterday dx pharyngitis, did not fill his meds." Exhibit 5, p. 50. Steven Von Flue, M.D., admitted Claimant to critical care: "Reason: Inhal. Injury, respiratory failure." Exhibit 5, p. 53.

14. The January 16, 2015 emergency record provides:

> CHIEF COMPLAINT:
> Difficulty breathing and speaking, recent chemical exposure.
>
> HISTORY OF PRESENT ILLNESS:
> Mr. Mendoza is a 37-year-old gentleman who works on a dairy farm. He presents to Emergency Department with complaints of discomfort and sensation of swelling in his tongue, lips, and his mouth, difficulty talking and speaking. He reports that these symptoms started 2 days ago after he was at the dairy farm where he works. He was using a chemical solution to clean the pipes, that the bucket dropped, and that a significant amount of fumes came up towards his face, that he breathed in a bunch of these fumes and afterwards he noticed that he was having some irritation to his throat and mouth and airway. He noticed some difficulties breathing and some swelling. He also had some rash and itching. He came in and was seen and evaluated yesterday in the emergency Department as the symptoms had been progressively worsening over the course of that evening. At that time, evaluation was done. The patient was not demonstrating any evidence of respiratory failure and was discharged home with return precautions. He returns now with some friends. He indicates that his symptoms have been progressively worsening and have increasingly worsened, particularly over the past couple of hours where he is now having quite a difficult time breathing. His voice is muffled. …. He … seems

> slightly confused. …. He feels like his lips and mouth and throat are all swollen. He rates this as severe and initiated or aggravated by breathing in the fumes from the acid substance that was involved in cleaning, which I was able to find was a combination of phosphoric nitric and hydrofluoric acid. …. He has not had a fever or any vomiting, he has been coughing slightly.
> ….
>
> Temperature 98.2. …. Patient does not appear toxic, but he does appear sleepy and intermittently in some distress with his breathing and with muffled attempts at speaking with significantly hoarse sounding voice. …. Upper and lower lips are edematous, as well as some erythema and edema of the bilateral facial mid facial regions, edema of the tongue. Upon pressing the tongue down significantly I am able to view his uvula, which appears only slightly edematous. …. Some mild erythema to the cheek regions ….
> ….
>
> He was given IM epinephrine as well as Benadryl, Pepcid, and Solu-Medrol. …. He was failing to demonstrate any significant improvement. …. It appeared that his voice was worsening and that he was having some increased edema rather than a decrease in edema. There was concern of impending airway compromise and failure.

Exhibit 5, pp. 66-67.

15. Luke Morris, D.O., removed Claimant to the resuscitation bay and Claimant agreed to intubation. Dr. Morris recorded:

> it was difficult for him to breathe and … he felt like he was having much swelling. …. Poison Control Centers … agreed with intubating the patient, managing the airway …. They indicated that the substance can cause some pulmonary edema. Additionally, concern[s] are for burns to the trachea airway as well as edema and swelling and airway compromise.
> ….
>
> airway edema, upper airway edema, and hoarse and muffled with respiratory distress and failure secondary to upper airway edema. After an exposure to inhaled fumes from acidic cleaning chemicals I suspect that this may be the cause. It is somewhat atypical that the progression was somewhat gradual and delayed over the couple of day[s].

Exhibit 5, p. 58.

16. During intubation under direct visualization Dr. Morris reported "edema along the vocal cords." Exhibit 5, p. 58. He recorded his diagnostic impression: "Acute respiratory failure,

upper airway edema, occupational exposure to inhaled acid fumes." Exhibit 5, p. 68. Claimant was admitted to the intensive care unit in critical but stable condition.

17. On January 16, 2015, Cary Jackson, M.D., performed bronchoscopy and recorded:

> The distal trachea was normal down to the carina. The posterior aspect of the carina was erythematous and indurated. There was a large purulent mucous plug in the left main stem bronchus. …. [T]he left upper lobe … were within normal limits. The left lower lobe orifice was diffusely indurated and erythematous with tenacious secretions. In the right bronchial tree, the right upper lobe orifice was normal. The right middle lobe was normal as well. There were focal areas of induration and erythema around the segmental orifices of the anterolateral posterior segments. …. There is evidence of lower airway injury as well as upper airway injury which by history occurred after an inhalational injury associated with cleaning chemicals.
> ….
>
> Acute respiratory failure. Upper airway edema appeared to be the primary cause. History is given that there was an occupational exposure which may have been a factor. However, it is unusual that induration did not occur until 2 days later. There is also evidence of lower airway inflammation. This may be related to airway injury from a caustic inhalation, but also atypical is that the findings are not uniform.

Exhibit 5, pp. 63, 65.

18. On the morning of January 17, 2015, Dr. Von Flue examined Claimant and assessed: "Acute respiratory failure, apparently secondary to upper airway edema due to occupational acid exposure." Exhibit 5, p. 74.

19. The Nampa Saint Alphonsus Medical Center discharge summary dated January 20, 2015, noted Claimant's use of cleaning chemicals while working at a dairy, his subsequent respiratory distress, emergent intubation, bronchoscopy revealing "left lower and upper airway injury consistent with inhalational injury associated with cleaning chemicals," and supportive ventilator care. Exhibit 5, p. 71. The summary recounted Claimant's improvement and extubation, then subsequent deteriorating condition and reintubation:

> On the morning of January 18, 2015, he passed a spontaneous breathing trial and a cuff leak test and was subsequently extubated without difficulty. He did relatively well the remainder of the day; however, during the night and the next morning, he

> developed increased respiratory distress and difficulty clearing secretions. …. It then became apparent that patient had significant proximal upper extremity muscle weakness with minimal to no lower extremity muscle weakness. This was symmetrical, with perhaps decreased reflexes. …. He developed worsening respiratory failure with evidence of aspiration the morning of 01/20/2015 … was intubated and sedated and transferred to St. Luke's Regional Medical Center for Neurology consultation as this is unavailable at this facility.
>
> With further consideration, it appears that the patient's presentation is most consistent with probable neuromuscular disorder, perhaps Guillain-Barre variant or acute inflammatory demyelinating polyneuropathy. …. In hindsight, the patient's reported possible viral illness in the week or so prior to presentation would be consistent with Guillain-Barre variant.

Exhibit 5, p. 72.

20. The discharge summary noted: "Acute hypoxic respiratory failure, possibly multifactorial secondary to apparent occupational acid inhalation, now with aspiration pneumonia/atelectasis/mucous plugging and neuromuscular weakness." Exhibit 5, p. 70.

21. On January 20, 2015, Claimant was admitted to St. Lukes Regional Medical Center in Boise where David Fonseca, M.D., examined him and assessed:

> 1. Progressive neuromuscular weakness of the upper extremities, unclear etiology. Query possible atypical Guillain-Barre syndrome. Of note, he did receive an influenza vaccination on January 16, 2015. He also had a rash that resolved approximately three or four days before onset of these neuromuscular weakness symptoms.
>
> 2. Respiratory failure secondary to progressive neuromuscular weakness of the upper extremities.

Exhibit 7, p. 128.

22. On January 21, 2015, Anna Irwin, M.D., examined Claimant. She noted Claimant's tracheal edema, respiratory distress following suspected inhalation injury, and diffuse neuromuscular weakness and assessed aspiration pneumonia and myasthenia gravis. Exhibit 7, pp. 135, 137. She commenced Claimant on IVIG treatment, and his upper extremity strength improved over the next five days. On January 24, 2015, Claimant was extubated, but then

developed hypoxia and was reintubated the following day. He continued to be alimented through a nasogastric tube. Antibody testing for myasthenia gravis was negative.

23. By January 27, 2019, Sogol Nowbar, M.D., assessed neuromuscular weakness of the eyes and mouth but improved in upper and lower extremities most consistent with atypical Guillain-Barre, acute respiratory failure secondary to a bulbar pharyngeal muscle weakness, and aspiration pneumonia. Exhibit 7, p. 158. On January 31, 2019, Eric Garner, M.D., performed a tracheostomy tube placement due to Claimant's Guillain-Barre syndrome with aspiration secondary to bulbar palsy and ventilator dependence. Exhibit 7, p. 166. By February 1, 2015, Dr. Nowbar diagnosed atypical Guillain-Barre syndrome with continued oropharyngeal and facial muscle weakness. Due to Claimant's persisting neuromuscular weakness and dysphagia, on February 12, 2015, a gastrostomy feeding tube was surgically placed. His tracheotomy tube was removed on February 19, 2015.

24. On February 23, 2015, Claimant was discharged from the hospital. His discharge summary provided in part:

> Acute respiratory failure in the setting of atypical Guillain-Barre syndrome. Upon his initial presentation to the outside facility, there was a report of sore throat, odynophagia, otalgia, and a pruritic rash with progressive respiratory failure, and concern for possible myasthenia gravis. He received empiric five days IVIG on admission to our facility at the recommendation of Neurology; however, acetylcholine antibody studies have resulted negative. Upon further consideration, it was felt that his clinical course more likely represented an atypical Guillain-Barre syndrome of questionable etiology (possible chemical exposure at work, recent influenza vaccination on 01/16/2015, versus viral or other cause).

Exhibit 7, p. 189.

25. On March 27, 2015, Dr. Irwin authored a neurology progress note summarizing Claimant's history which provides in part:

> Patient is known to me from his recent prolonged hospitalization with probable atypical Guillain-Barre syndrome with bulbar-oropharyngeal muscular weakness.

> Patient presented with symptoms of aspiration pneumonia, after chemical exposure at work (inhalation of a cleaning solution). Within a few days following inhalation, patient developed rapidly progressive weakness involving his face, bulbar muscles, respiratory muscles, upper extremities and to a lesser degree lower extremities.

Exhibit 7, p. 192.

26. **Condition at the time of hearing.** At the time of hearing on May 23, 2018, Claimant continued to experience some upper extremity weakness and reported hypersensitivity to fumes from several household cleaners.

27. **Credibility.** Having observed Claimant at hearing and compared his testimony with other evidence in the record, the Referee finds that Claimant has displayed memory lapses, but is generally a credible witness and testified credibly of his chemical exposure at work. The Commission finds no reason to disturb the Referee's findings and observations on Claimant's presentation or credibility.

DISCUSSION AND FURTHER FINDINGS

28. The provisions of the Idaho Workers' Compensation Law are to be liberally construed in favor of the employee. Haldiman v. American Fine Foods, 117 Idaho 955, 956, 793 P.2d 187, 188 (1990). The humane purposes which it serves leave no room for narrow, technical construction. Ogden v. Thompson, 128 Idaho 87, 88, 910 P.2d 759, 760 (1996). Facts, however, need not be construed liberally in favor of the worker when evidence is conflicting. Aldrich v. Lamb-Weston, Inc., 122 Idaho 361, 363, 834 P.2d 878, 880 (1992).

29. **Causation.** The crux of the issues herein is whether Claimant suffered an industrial accident on or about January 15, 2015, causing personal injury and resulting in his need for medical care including extensive hospitalization. Claimant asserts his exposure to chemicals while cleaning milking lines at Eagle Ridge on and about that time caused him respiratory and other injuries and/or Guillain-Barre syndrome. The fact that Claimant suffered personal injury is not

disputed. However, Defendants deny that Claimant suffered injury from any alleged chemical exposure at work. While acknowledging that Claimant has Guillain-Barre syndrome (GBS), Defendants assert it was caused by a non-industrial cause.

30. Idaho Code § 72-102(18)(b) defines accident as "an unexpected, undesigned, and unlooked for mishap, or untoward event, connected with the industry in which it occurs, and which can be reasonably located as to time when and place where it occurred, causing an injury." A claimant must provide medical testimony that supports a claim for compensation to a reasonable degree of medical probability. Langley v. State, Industrial Special Indemnity Fund, 126 Idaho 781, 785, 890 P.2d 732, 736 (1995).

31. Claimant testified that he had no respiratory problems, fatigue, or muscle weakness and was healthy prior to January 3, 2015. He testified that he never received any immunizations or vaccinations prior to January 15, 2015.

32. Consideration of the reports and depositions of Drs. Bardana, Dawson, Friedman, and Dodson, leads the Commission to conclude that Claimant suffered an inhalation injury, how significant it is difficult to tell, as a result of the exposures of early January 2015. These injuries may very well have been compounded by aspiration of stomach contents connected to his neurological injury. The real difficulty in this case lies in answering the question of whether Claimant's neurological injury is causally related to the aforementioned exposure. Before discussing the specific facts relating to this issue, it is worth reiterating that it is Claimant who bears the burden of proving medical causation to a reasonable degree of medical probability. The Commission must be persuaded that there is more medical evidence supporting causation than not, or Claimant fails of his burden of proof. Langley v. State, Industrial Special Indemnity Fund, *supra*. The assertion, which must be proven by Claimant, is that Claimant's nerve damage is

causally related to his chemical exposure. It is the testimony of Drs. Friedman and Dodson upon which Claimant principally relies in support of demonstrating medical causation.

33. In framing the causation inquiry, it is helpful to understand the diagnosis which attaches to Claimant's neurological condition; if a conclusion about Claimant's diagnosis cannot be reached, it is much more difficult to speak intelligently about what caused the condition. For example, if a worker's presenting symptom is chest pain, to understand whether overexertion at work has anything to do with this complaint, it would be helpful to know whether the worker carries the diagnosis of acid reflux v. myocardial infarction.

34. The medical evidence demonstrates that Claimant suffers from injury to neurological structures involving his oropharyngeal, neck, and upper extremity musculature. At least three of the experts whose testimony was taken in this matter concur that Claimant suffers from GBS. Only Dr. Dodson equivocated, but even he acknowledged that GBS, whether caused by toxic chemical exposure or antecedent infection is in the differential. (Dodson Depo. at 39, 44-45). The evidence persuades the Commission that Claimant's neurological diagnosis is best described as GBS.

35. As described by Dr. Bardana, GBS was historically thought to be a single disease, but is now well known to be a heterogeneous disease with many variants, possibly up to 10 or 11. Dr. Dodson testified that there are five subgroups of GBS, three associated with antibodies and two without an etiology related to antibodies. (Dodson Depo. at 35-36). Dr. Bardana testified that 70 percent of GBS patients present with a history of antecedent infection of some type prior to the development of neurological symptoms. (Bardana Depo. at 22-23). Having said that, Drs. Friedman, Dodson, Bardana, and Dawson all agree that the etiology of the syndrome is poorly understood. Many causes or associations have been entertained. Per Dr. Friedman, GBS is thought

to be an autoimmune disease (Friedman Depo. at 18) which may be triggered in a variety of ways. As noted, antecedent infections are associated with GBS. Vaccinations as well have been associated with the syndrome. Certain types of surgery, in particular, heart surgery and bone marrow transplantation surgery, have known associations with GBS. (Bardana Depo. at 24). Certain cancers are associated with GBS. Finally, because of the peculiar facts of this case, a good deal of discussion was devoted to the question of whether or not exposure to toxic "chemicals" can trigger GBS.

36. Dr. Dawson testified that in medical literature there has "never" been a case reporting an association between chemical exposure and GBS. Dr. Friedman acknowledged that he is aware of no medical literature supporting a direct cause between toxic chemical exposure and GBS. Dr. Bardana was unaware of medical literature linking GBS to chemical exposure, noting that chemical exposures are very common, while GBS is very rare. (Bardana Depo. at 27). Specifically, while acknowledging that Claimant may have suffered some minor irritation as the result of his chemical exposure, Dr. Bardana testified that there is no medical evidence to support the conclusion that the chemicals in question caused Claimant to develop GBS.

37. Dr. Dodson appears to believe that Claimant suffered a sensory and motor neuron injury as a result of his chemical exposures:

> Q: [By Mr. Alysworth] Would you be able to describe for us - - upon going back to Saint Al's for the second time, what were these toxic, caustic, hazardous chemicals doing to Jose's body at that point?
>
> A: All of those signs and symptoms that he presented were evidence that neurons have been damaged - - and there are various kinds of neurons that are involved. Most of these are sensory or motor neurons, and part of that - - we've known for a long time that motor neurons have what's called a "Node of Ranvier," and it has been known for some - - many, many years that the Node of Ranvier is a major site of pharmacological agents like local anesthetics and toxic chemicals that can damage then the Node of Ranvier, which results then in changes in conduction

> to motor neurons in responding to surroundings, to movement, to their space and time.
>
> Secondly, those sheaths are contained in a myelin sheath. Myelin is made up of protein and made up of fatty substances, which then also can be damaged.
>
> The myelin sheath is an insulation of neurons allowing for the neurons then to have a very speedy conduction and will actually protect the neuron, but those myelin sheaths could also be damaged by toxins and by antibodies.
>
> So all of those signs and symptoms that Jose is presenting are evidence that motor neurons have been impacted at either the Node of Ranvier or the architecture around the Node of Ranvier.

Dodson Depo. 29:9-30:11. Therefore, while Dr. Dodson appears to endorse the proposition that the chemicals in question directly attacked Claimant's neurons at the "Nodes of Ranvier," he also appears to acknowledge the possibility that the myelin sheaths of Claimant's nerves could have sustained damage by an antibody response to the chemical exposure. (See Dodson Depo. at 45-46).

38. It is important to point out that Dr. Dodson reached his conclusion concerning the cause of Claimant's neurological injury not because he identified evidence associating those injuries with the toxic chemicals in question, but because he ruled out other known causes/associations. (Dodson Depo., 35:12-39:5). It is difficult to accept that ruling out other possible causes of Claimant's neurological injury satisfies Claimant's burden of ruling-in (and proving) the cause of his injury by exposure to toxic chemicals. Where the etiology and mechanism of GBS is poorly understood, ruling out other causes is not a substitute for affirmative medical proof establishing that Claimant's condition is causally related to the subject exposures.

39. Dr. Dodson's theory that the toxins in question directly attacked Claimant's nerve cells raises more questions than it answers. Dr. Dodson testified to his "professional opinion that this is a textbook case for chem exposure, i.e., very straightforward chemical exposure." (Dodson

Depo. at 47). The statement implies that neurological symptoms of the type exhibited by Claimant are a well-known consequence of exposure to the chemicals at issue. However, no supporting evidence was offered in the form of peer reviewed studies or learned treatises, much less textbooks. As noted, Dr. Dawson testified that there is a dearth of medical literature supporting a relationship between toxic chemical exposure and GBS. In rebuttal, Claimant offered a number of medical abstracts suggesting that there may be some relationship between exposure to certain specific chemicals (organophosphates and mustard gas are the ones referenced in the abstracts) and the development of GBS. This does not controvert the testimony of Drs. Dawson, Bardana, and Friedman that there is no medical literature which documents a relationship between GBS and the chemicals to which Claimant was exposed. Nor do the Material Safety Data Sheets (MSDS) for the chemicals in question suggest that exposure to the substances may result in neurological injury. (Clt. Exhibits at 10). If the chemicals to which Claimant was exposed are neurotoxins, it is reasonable to believe that literature and warnings documenting these effects would exist. As Dr. Bardana has noted, in an industrial society, exposure to chemicals, both benign and toxic, is frequent, affording many opportunities to catalogue effects. Moreover, Dr. Bardana rejected Dr. Dodson's theory of direct neural damage by the chemicals at issue:

> A: Okay. Dr. Dodson believes that the chemical exposure that might have occurred, and I went through it in sort of a brief terminology, affects the tissue, it damages the airway, and damages nerves, and it finally accesses a part of the axonal tissue and the myelin tissue of the nerves, some of which is referred to as the node of Ranvier and impacts this particular setting and causes the entire process of Guillain-Barre to take place. Now, I have explained this in other terms which are published in the literature and reproduced in rabbits and to a certain extent in mice. What does happen, not what - - I don't believe any of what Dr. Dodson believes happened, which I believe is unsubstantiated speculation is ever published anywhere or proven anywhere. What does happen and what I have explained before in some detail is that there are antigens that identify one with another on the covering of organisms - - Ecscherichia coli, campylobacter, Klebsiella. There are many organisms that have a form of polysaccharide material that identifies with epitopes on the nervous tissue. And when this infection occurs, the body is making

> antibodies against the organism, and in doing so, these antibodies not only kill the infections but they recognize as part of their kill zone the nervous section of the axons and of, if you wish, the nodes of Ranvier and affect them adversely. This is the mechanism that operates. It is an immunological mechanism and autoimmunity as a result of immunity gone wrong. There is no literature that would support Dr. Dodson's belief that this simply happens by way of the chemical traversing through the tissues at concentrations high enough to impact the nerves directly. That simply is not plausible biologically in my view and it simply has never been supported by any literature that I have read by any animal models that have been tried or by literature that Dr. Dodson put forward to support his theory.

Bardana Depo. 47:8-48:20.

40. Dr. Dodson's hypothesis on a direct causal link between the chemical exposure and nerve damage is further called into question by Dr. Friedman's testimony. As developed in more detail, *infra*, Dr. Friedman endorses a more indirect link by which chemicals cause cell damage to Claimant's respiratory system, and that it is this cell damage which provokes an immune response which, in turn, damages nerve cell.

41. As above noted, all four experts have testified that it is generally accepted that certain antecedent infections can cause the development of GBS. Drs. Friedman, Bardana, and Dodson described the mechanism by which an antecedent infection by a bacteria or virus causes GBS. Dr. Bardana succinctly described how the body's response to pathogens causes GBS:

> The infections are quite varied, and there is an association between many infections and this particular condition. The most common infection that is cited is an infection called - - an infection called campylo bacteria, which is an infection of the bowel and can really cause havoc with the immunological system since the bacteria has - - it has antigens that are similar to antigens on the Schwann cell of the nerve. And as a result of these antigens in the bacterial covering there is a massive production of antibodies against the bacteria to get rid of it, but in doing that, the antibodies that are created, these antibodies turn out to be antiganglioside antibodies that affect the nerve, particularly the nerves on the axons of the nervous system in the spinal cord and in the brain, depending upon the form of Guillain-Barre you have.
>
> So if you have this camplyo bacteria, this campylo bacteria jejuni infection, then you have what is called molecular mimicry. Because you have identical antigens on the bacterial surface as you have on the nervous system, the antibodies that are

> produced in an attempt to destroy the bacterium also react in a way with the - - to destroy the nervous system. So it is called molecular mimicry.

Bardana Depo. 22:22-23:21. (See also Friedman Depo., 17:10-18:24). Therefore, central to the "molecular mimicry" theory is the existence of antigens on the surface of an invading pathogen which bear some similarity to antigens on the outside of the human nerve cell. The antibodies which are generated as part of the body's response to infection erroneously target antigens on the nerve cells, as opposed to antigens on the surface of the invading pathogen. Therefore, it is possible that Claimant's GBS resulted from an antecedent infection unrelated, and coincidental, to his occupational exposure.

42. Dr. Friedman did not endorse Dr. Dodson's theory that Claimant's neurological injuries were directly caused by the toxic chemicals to which he was exposed. Rather, Dr. Friedman testified that there is a more indirect mechanism by which toxic chemicals of the type to which Claimant was exposed can cause GBS. Per Dr. Friedman, the toxic chemicals Claimant inhaled caused injury to the cells of Claimant's mouth, trachea, and lungs. The damage to these tissues, in turn, provoked an immune response, i.e., an inflammatory response to localized tissue damage. (Friedman Depo., 19:21-20:19; 39:18-40:3; 52:18-53:10; 59:23-60:13). What is left unexplained by Dr. Friedman is how the immune response to dead or damaged lung tissue, for example, impacts Claimant's sensory and motor neurons. The molecular mimicry theory involving the body's erroneous response to a foreign pathogen does not explain how damage to the body's own tissues somehow causes the immune system to attack the body's sensory and motor neurons. (Bardana Depo., 40:1-9). Dr. Friedman offered no elaboration on this critical piece of the mechanism of injury. GBS occurs with very low frequency in the population at large. However, people suffer cell death and damage everyday in accidents large and small. If such tissue damage puts people at risk for developing GBS, then it is not unreasonable to expect that the incidence of

GBS would be greater than it is, and that this mechanism of GBS would be better reported in the literature. (Dawson Depo., 33:18-34:19).[1]

43. There is yet another problem with Dr. Friedman's opinion, a problem which was noted by the Referee. At one point in his testimony, Dr. Friedman acknowledged that it is just as likely that Claimant's condition is due to an infection as it is to the consequences of toxic chemical exposure. Of course, if this is Dr. Friedman's view, then on Dr. Friedman's testimony Claimant's claim would fail since this testimony does not support an industrial cause to a reasonable degree of medical probability. However, the Referee downplayed this testimony, explaining that in other portions of his testimony, Dr. Friedman amplified an opinion that Claimant's condition has an industrial cause to a reasonable degree of medical probability. The Commission questions whether Dr. Friedman's equivocation can be so easily rejected. Consider this testimony:

> Q: [By Mr. Alysworth] Doctor, based on your knowledge and professional medical experience, is there any dispute that exposure to toxic, caustic, hazardous chemicals such as those in Jose's case are known to cause a human immune response?
>
> A: Well, there's always controversy no matter what. I'm just going to out that people still believe the Earth is flat - - okay - - despite physical evidence.
>
> Nonetheless, the answer is - - that is my opinion that the toxic, destructive chemicals caused tissue damage and initiated an immune response, which is normal.
>
> Q: So you set forth in your October 9, 2017 letter, quote, "It is my medical opinion that it is equally likely that Mr. Mendoza-Zapata's neurological condition was as a result of his exposure to chemicals as it is to the speculated non-confirmed viral infections"; is that correct?

[1] It is noted that both Dr. Bardana and Dr. Dodson recognize that surgery (according to Dr. Bardana, heart surgery and bone marrow transplantation surgery, in particular) has been associated with GBS. The argument might be that since surgery necessarily involves incisions which result in damage to the skin, muscle, and other tissues, the fact that certain surgeries are associated with GBS lends support to Dr. Friedman's theory that tissue damage provokes an immune response which, in turn, causes GBS. At first blush this argument has some appeal, but starts to fall apart on closer examination. Why only certain surgeries (according to Dr. Bardana) and not every surgery involving the use of a scalpel? Maybe surgery is a risk for developing GBS because of something else that attends surgery, such as use of anesthesia or other medications commonly used in surgery.

A: That is correct - - and that is an accurate statement as read from my report of October 9, 2017.

Q: And if I read the medical records correct, with respect to your opinions as to the role of Jose's exposure to the toxic chemicals at work, you weren't alone in that opinion, were you?

A: That is correct.

Q: And it looks like Jose's neurologist at St. Luke's, Dr. Anna Irwin, among others, actually considered chemical causation by providing or stating in some of the reports in pertinent part, quote, "Upon further consideration, it was felt that his clinical course more likely represented Atypical Guillain-Barre Syndrome of questionable etiology, possible chemical exposure at work"?

A: That is correct.

Q: So the opinions again that you provided on October 9th, 2017, were those given to a reasonable degree of medical probability?

A: Yes.

Q: And are those still your opinions as you sit here testifying today?

A: Yes.

Friedman Depo. 39:18-41:7. It is hard to know what to make of this. On the one hand, Dr. Friedman appears to affirmatively express an opinion that Claimant's exposure to toxic chemicals caused tissue damage, which initiated an immune response. However, in the very next breath, he concedes that it is just as likely that Claimant's neurological symptoms are the result of Claimant's exposure to a pathogen. From this testimony, it is unclear whether Dr. Friedman actually believes that it is more probable than not that Claimant's neurological injury is mediated by an immune response provoked by tissue damage resulting from his exposure to toxic chemicals.

44. Even if this last objection is set aside, it is clear that Drs. Dodson and Friedman have come to different conclusions concerning the etiology of Claimant's neurological injury; Dr. Dodson appears to believe that Claimant's neurological injury represents direct injury to

Claimant's nerves by the chemical agents in question. Dr. Friedman appears to believe that the toxic chemicals caused damage to other structures, and that the body's immune response to that damage somehow triggered an attack on Claimant's nerve cells. No one has suggested that both theories operate to explain Claimant's neurological symptoms. Moreover, as discussed above, each theory comes with its own set of problems. Dr. Dodson believes that the chemicals in question caused direct injury to Claimant's nervous system, yet there is no evidence that the chemicals in question are known to damage nerves. (Dawson Depo., 34). With respect to Dr. Friedman's theory, the critical link between the hypothesized immune response and damage to Claimant's nerve cells was not provided. In both cases, if the proponent of a particular theory is correct, one might reasonably expect to see proof in the medical literature supporting that theory. Nothing of the type was adduced in a case that fairly screams for a detailed literature search.

45. Although it is not Defendants' burden to prove the negative, Dr. Bardana persuasively testified about what seems to be the most accepted and understood cause of GBS, i.e., exposure to a pathogen of some type. The parties have devoted a good deal of unnecessary effort to arguing about the likelihood that a particular pathogen could be implicated. The Commission believes that this is a somewhat pointless exercise since it appears that GBS may be triggered by one of any number of infectious agents, only a few of which were ruled-out during Claimant's hospital stay. The point is that aside from possibly ruling-out the Zika virus as the culprit, Claimant did not successfully denigrate the possibility that Claimant's condition was initiated or triggered by some other infectious agent.

46. Based on the foregoing, the Commission concludes that the Claimant has failed to establish that his GBS was initiated by his exposure to the toxic chemicals in question. On this

point, the Commission concludes that the evidence is, as Defendants have suggested, in equipoise, with no theory of what caused Claimant to develop GBS entitled to greater weight than any other.

47. Having said that, it is hard not to be struck by the fact that Claimant's neurological symptoms followed so closely on the heels of his industrial injury. Certainly, a temporal relationship between the chemical exposure and Claimant's injuries is required before a causal relationship can be established; cause must precede effect. However, a temporal relationship between the industrial accident and Claimant's neurological injury is not, in and of itself, sufficient to establish causation. In at least one recent case we have come close to departing from that rule, but that case was decided on the basis of its own compelling facts. Tenny v. Loomis Armored US, IC 2014-032378 (Issued December 10, 2019). Here, it is tempting, as well, to be swayed by the existence of a close temporal relationship. Claimant had an injurious exposure to toxic chemicals which, at the very least, caused irritation to his respiratory system and mucus membranes. It is no great leap to want to believe that the same exposures must have caused his neurological injury because of the temporal proximity of his chemical exposure to his neurological injury, especially where the cause of GBS is not well understood. However, as explained above, the Commission is unpersuaded by the evidence that has been adduced that the chemical exposures did anything to trigger/cause Claimant's GBS. It is just as likely that the triggering cause of Claimant's GBS is entirely unrelated to the subject accident.

48. However, it is equally axiomatic that the Employer takes the injured worker as it finds him. Wynn v. J.R. Simplot Co., 105 Idaho 102, 104, 666 P.2d 629, 631 (1983). Even though Claimant's proof is insufficient to prove that his GBS was initiated, i.e. caused, by his industrial exposure, Employer may nevertheless have some responsibility for this neurological injury if it is demonstrated that this contemporaneous, but unrelated, condition was aggravated or accelerated

by the work accident. Claimant has asserted this as a fallback position, and in support thereof, relies on the following testimony from Dr. Friedman:

> Q: [By Mr. Alysworth] So I'm going to present you with another hypothetical - - if you can please try and follow me. Let's presume that Dr. Dawson and Dr. Bardana are somehow correct, and Jose is statistically one of the - - it would appear to be one of the unluckiest people on the planet in that he somehow contracted unverified Zika virus, which coincidentally manifested itself via GBS at the exact same time he suffered injuries from toxic, caustic, hazardous worksite chemical exposures on January 14th or 15th of 2015. Okay?
>
> A: Okay.
>
> Q: Did those workplace chemical exposure injuries hasten, aggravate, or otherwise contribute to Jose's need for medical treatment he received on January 15th, 2015 and his January 16th, 2015 hospitalization?
>
> Mr. McFeeley: Object to the form of the question.
>
> A: With your hypothetical, the assumption is he's had the infection, and he's having the Guillian-Barre response to this infection - -
>
> Q: And he's having the chemical exposures.
>
> A: - - and he's having the chemical exposures, which they document in his throat, and his airways, and into his left lung - - would likely make him worse faster - - would aggravate his condition and make it highly likely he would require hospitalization and intubation.

Friedman Depo. 46:19-47:18.[2]

[2] Claimant asked a similar question of Dr. Dodson. At the time of his July 31, 2018 deposition, the question and response were as follows:

> Q: [By Mr. Alysworth] Doctor, I'm going to provide you with a hypothetical, so please try to follow me here. Let's presume that Dr. Gary Dawson and Dr. Bardana are somehow correct - - and Jose is a really, really unfortunate person in that he somehow contracted the unverified Zika virus which coincidentally manifested itself via GBS at the exact same time he suffered injuries from toxic, caustic, hazard worksite chemical exposure on January 14th and 15th of 2015. Okay? Did those workplace chemical expose [sic] injuries hasten, aggravate, or otherwise contribute to Jose's need for medical treatment on January 15, 2015, and his subsequent hospitalization?
>
> A: Possibly.

Dodson Depo. 48:14-49:2. This testimony is not helpful to Claimant's aggravation theory. However, within the time allowed by IRCP 30(e) Dr. Dodson offered some substantive changes to his response. He changed his answer from "possibly" to "yes definitely," offering the explanation that he misheard this, and another, question due to service-

49. Therefore, Dr. Friedman's opinion is that if Claimant coincidentally developed non-work related GBS at around the time of his chemical exposure, that exposure aggravated the underlying syndrome and is responsible for Claimant's course of medical treatment.

50. Although the testimony appears to have been adduced as somewhat of an afterthought, the proposition that Claimant's GBS occurred coincidental to his chemical exposure, but was aggravated by his chemical exposure, is not challenged by the reports or testimony of Drs. Bardana and Dawson. While it is difficult to say that this amounts to an abundance of proof, the Commission concludes that there is no basis to disregard Dr. Friedman's testimony, and that it, in conjunction with our other findings, is legally sufficient to establish, on a more probable than not basis, the following: Claimant suffered an injury from an accident arising out of and in the course of his employment. The accident caused respiratory and other injuries, but did not initiate Claimant's contemporaneous GBS. However, the industrial accident aggravated Claimant's non-work related GBS, causing the condition to manifest more severely than it would have absent the work accident, further complicating Claimant's condition. Therefore, all of the expenses associated with Claimant's hospitalization and recovery are a compensable part of the work accident. This decision does not address whether Claimant's industrial accident resulted in permanent worsening of his neurological injury.

51. **Medical care.** Idaho Code § 72-432 provides in pertinent part:

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related hearing loss. It is interesting that of the many questions to which he responded in his deposition, the only two he misheard were those in which he originally gave answers which were unhelpful to Claimant's theory of the case. In their brief, Defendants objected to Dr. Dodson's changes, and this prompted Claimant to admonish Defendants for their "disgraceful attack on a service-related disability." (Claimant Post Hearing Reply Brief at 13). Having carefully reviewed Dr. Dodson's deposition, the Commission shares Defendants' skepticism of the impetus for the changes. The Commission sustains Defendant's objection. To allow such substantive change to hearing testimony after the close of evidence, without the opportunity to cross-examine, seems overly prejudicial. However, even if Dr. Dodson's new answer is allowed to stand, the answer is not particularly probative of the question of whether Claimant's chemical exposure worsened his non-work related GBS. All Dr. Dodson says is that the workplace exposures contributed to Claimant's need for medical treatment, not that the workplace exposure aggravated an underlying injury. It is Dr. Friedman's testimony which seems to speak to this last point.

> the employer shall provide for an injured employee such reasonable medical, surgical or other attendance or treatment, nurse and hospital services, medicines, crutches and apparatus, as may be reasonably required by the employee's physician or needed immediately after an injury or manifestation of an occupational disease, and for a reasonable time thereafter. If the employer fails to provide the same, the injured employee may do so at the expense of the employer.

Of course an employer is only obligated to provide medical treatment necessitated by the industrial accident, and is not responsible for medical treatment not related to the industrial accident. Williamson v. Whitman Corp./Pet, Inc., 130 Idaho 602, 944 P.2d 1365 (1997).

52. Claimant, having proven he suffered an industrial accident on or about January 15, 2015, causing personal injuries, to include respiratory injuries and an aggravation of pre-existing GBS, has proven his entitlement to reasonable medical treatment therefore.

53. The reasonableness of medial treatment for an industrial injury is determined by the totality of the circumstances. Chavez v. Stokes, 158 Idaho 793, 353 P.3d 414 (2015). Claimant has proven that Defendants are liable for Claimant's medical care including, but not limited to, extensive hospitalization and follow-up care, for his respiratory, neurological, and other injuries, consistent with this opinion.

CONCLUSIONS OF LAW AND ORDER

1. Claimant has proven he suffered an industrial accident on or about January 15, 2015, of toxic chemical exposure at work causing personal injuries to his skin, mucus membrane, and respiratory system, including an aggravation of pre-existing Guillain-Barre syndrome.

2. Claimant has proven Defendants are liable for his reasonable and necessary medical care including but not limited to extensive hospitalization and follow-up care for his respiratory and other injuries caused or aggravated by the January 15, 2015, industrial accident.

3. The Commission reaches no decision on the extent, if any, to which the industrial injury resulted in a permanent worsening of Claimant's neurological condition.

4. Pursuant to Idaho Code § 72-718, this decision is final and conclusive as to all matters adjudicated.

DATED this ___20th___ day of February, 2020.

INDUSTRIAL COMMISSION

_____/s/_____
Thomas P. Baskin, Chairman

_____/s/_____
Aaron White, Commissioner

_____/s/_____
Thomas E. Limbaugh, Commissioner

ATTEST:

_____/s/_____
Assistant Commission Secretary

CERTIFICATE OF SERVICE

I hereby certify that on the ___20th___ day of __February__, 2020, a true and correct copy of the foregoing **FINDINGS OF FACT, CONCLUSIONS OF LAW, AND ORDER** was served by regular United States Mail upon each of the following:

JUSTIN P AYLSWORTH
PO BOX 6190
BOISE ID 83707-6190

NEIL MCFEELEY
PO BOX 1368
BOISE ID 83701-1368

_____/s/_____