BEFORE THE KANSAS WORKERS COMPENSATION APPEALS BOARD

APRIL S. ABBOTT	)	
Claimant	)	
V.	)	
	)	
PROMISE HOSPITAL OF OVERLAND PARK INC.	)	AP-00-0458-067
Respondent	)	CS-00-0442-897
AND	)	
	)	
ACCIDENT FUND INSURANCE CO. OF AMERICA	)	
Insurance Carrier	)	

ORDER

The respondent and its insurance carrier (respondent), through Benjamin Gary, requested review of Administrative Law Judge (ALJ) Kenneth Hursh's preliminary Order dated May 13, 2021. William Manson appeared for the claimant.

RECORD AND STIPULATIONS

The record consists of:

1. The preliminary hearing transcript dated April 22, 2020, with all attached exhibits, including the claimant's October 1, 2019 deposition transcript.

2. The deposition transcript of Robert Beck dated May 4, 2020, with all attached exhibits.

3. The preliminary hearing transcript dated October 1, 2020, with attached exhibits.

4. The preliminary hearing transcript dated May 12, 2021, with attached exhibits.

5. The pleadings and the case file.

Any stipulations are adopted.

ISSUE

Did the claimant sustain injury by repetitive trauma or occupational disease arising out of and in the course of her employment?

FINDINGS OF FACT

The claimant alleges she sustained a work-related personal injury by repetitive trauma or occupational disease, namely anti-synthetase syndrome interstitial lung disease (ASILD) due to exposure to black mold, with an asserted injury date of October 10, 2018.

The claimant worked for the respondent as a respiratory therapy manager. In August 2018, the respondent discovered water damage and mold in their central supply room. For two days, the claimant moved respiratory therapy supplies from the supply room. She was not wearing personal protective equipment. Shortly thereafter, perhaps within less than a week, the claimant began coughing and having shortness of breath. The claimant testified her cough was chronic within three weeks after her exposure. The mold in the central supply room was remediated after the claimant's exposure.

The claimant testified she was also exposed to mold in a patient's room (Room 412) and the skilled nursing facility. She testified she went into Room 412 perhaps more than five times for patient care during the summer of 2018, and saw mold on the walls. In September 2018, she went to the skilled nursing facility once or twice a week to gain access to medication. The claimant believed she was exposed to mold in the skilled nursing facility. These rooms were closed off and drywall was removed due to mold.

The claimant reported her symptoms and was initially seen by the workers compensation clinic at her workplace. An inhaler was prescribed without benefit. On September 12, 2018, she was seen at Concentra for persistent and worsening cough and shortness of breath. The claimant reported developing a cough one or two days after moving respiratory equipment from storage. A chest CT scan was read as abnormal, and the claimant was referred to Kathryn Gillen, M.D., a board certified pulmonologist. On October 4, 2018, Dr. Gillen performed a bronchoscopy, which showed the claimant had an autoimmune disorder. The claimant was referred to Stephen Ruhlman, M.D., who is board certified in internal medicine and rheumatology.

On October 10, 2018, the claimant had significant coughing and shortness of breath while working. That evening, the claimant was admitted to Advent Health and not discharged until October 25, 2018. The claimant was unsuccessfully treated with IV steroids and antibiotics. She then underwent a lung biopsy. The Mayo Clinic reviewed the biopsy as showing evidence of cellular interstitial pneumonia, neuroendocrine cell

hyperplasia and multiple carcinoid tumorlets. The claimant was discharged on supplemental oxygen. She never returned to work for the respondent.

On November 9, 2018, the claimant began treatment with Dr. Ruhlman. The claimant had extensive testing and was diagnosed with anti-synthetase syndrome.

The claimant testified she saw mold in the basement of the respondent's facility in August 2018 when obtaining respiratory supplies. The claimant testified she went to the basement about once per month. She testified she attended meetings with respondent's management about known mold in the building. Flooding in the basement, on account of a damaged sewer line, was discovered in November or December 2018. Testing was done in the basement for mold, but not in the remainder of the respondent's facility.

Subsequently, the respondent's human resources director, Randy Russell, provided the claimant with photographs showing visible mold in the respondent's facility and posted warnings concerning the need for respirators and protective clothing in the medication supply room due to a microbial hazard.

Dr. Ruhlman authored a letter dated March 7, 2019, stating:

> The [claimant] was first seen 11/9/18 for pulmonary fibrosis with a subsequent diagnosis of anti-synthetase syndrome. This is after she was exposed to black mold at work August, 2018, and then developed a cough in August and dyspnea in September, 2018. She has been on steroids and azathioprine for months and thankfully is better.
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> There is literature regarding environmental triggers of autoimmunity in anti-synthetase syndrome which raise the possibility of the black mold being a trigger in her situation. By timing of onset and symptoms, this does seem likely. She is improving with either therapy, or length of time from the exposure. Given the way she is improving most recently with rapid clinical improvement, it does appear removal from exposure is a factor.[1]

On October 23, 2019, the claimant saw Mark Hamblin, M.D., who is board certified in internal medicine, pulmonary medicine and critical care medicine. Dr. Hamblin is an associate professor and Director of the University of Kansas ILD and Rare Lung Disease Clinic. The claimant reported persistent shortness of breath on exertion and an increased cough. She indicated she worked for the respondent from January through October 2018 and saw obvious and significant mold throughout the building. The claimant denied any prior respiratory problems and indicated she led a very active lifestyle before the mold exposure. Dr. Hamblin noted the claimant's lung function was restricted and impaired. Dr.

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[1] P.H. Trans. (Apr. 22, 2020), Cl. Ex. 8 at 33.

Hamblin diagnosed the claimant with autoimmune disease, chronic lung disease and occupational lung disease. Dr. Hamblin believed the claimant had likely reached maximal improvement and stated:

> We did discuss that many autoimmune diseases are starting to be associated with respiratory exposures. In particular, we do note that with mold exposures, the mold spores will induce [haptens] which can result [in] autoimmune reactions, particularly in the lung. Given the exposures that she has demonstrated in her prior place of employment, I do think that there is an association. Thankfully, she is now out of that environment, but clearly remains impaired as a result of her pulmonary disease process.[2]

At her attorney's request, the claimant saw William Barkman, Jr., M.D., on February 18, 2020. Dr. Barkman is board certified in internal medicine, with a subspecialty in pulmonary diseases. The claimant reported significant shortness of breath with exercise and an improved cough. Dr. Barkman noted the claimant had no chronic preexisting respiratory conditions. The claimant reported exposure to black mold in the supply room and other areas of the respondent's facility. Dr. Barkman made two diagnoses, diffuse interstitial pulmonary neuroendocrine cell hyperplasia (DIPNECH), a rare pulmonary disease he did not believe was related to the claimant's employment, and ASILD, which the doctor indicated was caused by the claimant's work. Dr. Barkman stated, "After considering all factors, I believe the primary (prevailing) factor leading to the development of the anti-synthetase lung disease was the August 2018 work related mold exposure at Promise."[3] Dr. Barkman stated there was a strong temporal relationship between the claimant's exposure to mold and the onset of her symptoms. After noting the absence of other potential causes, Dr. Barkman opined the claimant's ASILD was most probably due to occupational mold exposure at the respondent's facility.

A preliminary hearing was held on April 22, 2020. ALJ Hursh ruled the claimant's lung conditions arose out of and in the course of her employment with the respondent, on account of work-related mold exposure.

During a deposition of Robert Beck, the CEO of the respondent, on May 4, 2020, the parties agreed October 10, 2018, was the proper date of incapacitation or injury, under the Kansas occupational disease statute. Mr. Beck testified there was mold in the respondent's basement between August and October 2018, as based on an analysis from Pro-Lab. Mr. Beck believed the respondent's basement flooded in August or September 2018. Mold was remediated in the basement. Mr. Beck testified a chiller line broke and

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[2] *Id.,* Cl. Ex. 8 at 41.

[3] *Id*., Cl. Ex. 4 at 4 and Cl. Ex. 5 at 3.

caused wet carpet in the director of nursing's office and medication room. He stated the water damage was repaired in those rooms, but mold testing was never done.

Dr. Barkman issued a letter dated May 12, 2020, stating the claimant would not need medical treatment for her ASILD but for her workplace mold exposure.

The claimant returned to Dr. Gillen on May 21, 2020, with worsening symptoms. The doctor admitted the claimant to Advent Hospital for exacerbation of her anti-synthetase syndrome. She was diagnosed with acute hypoxic respiratory failure, acute exacerbation of interstitial lung disease and cough. The claimant's oxygen requirements were increased. She was discharged on May 23, 2020.

On June 3, 2020, the claimant returned to Dr. Gillen. She reported worsening symptoms. The doctor reinstated oxygen at a higher level and recommended a lung transplant referral.

A second preliminary hearing was held on October 1, 2020. Among various rulings, ALJ Hursh authorized continued medical treatment with Drs. Barkman, Gillen and Ruhlman.

At the respondent's request, Stuart Levy, M.D., an internal medicine and pulmonary disease specialist who is board certified in internal medicine and pulmonary medicine, reviewed the claimant's medical records, but he did not examine the claimant. Dr. Levy believed the claimant was exposed to indoor mold for less than 16 hours, but also had alleged brief exposure to mold in Room 412 and the skilled nursing facility medication room a few times per week. Dr. Levy noted the claimant's symptoms developed less than one week after working in the central supply room. The doctor stated the opinions of Drs. Barkman and Hamblin lacked scientific basis. According to Dr. Levy, correlation between mold exposure, onset of symptoms and diagnosis of ASILD does not indicate causation. Dr. Levy opined the claimant had preexisting ASILD and her symptoms were likely caused by volatile organic compounds (VOCs), because ASILD does not have an acute onset and the claimant's symptoms were nearly immediate after exposure to indoor mold. The doctor also noted hypersensitivity pneumonitis (HP) can be caused by mold exposure, but the claimant did not have HP.

Dr. Levy's January 5, 2021 report stated:

> It is more likely than not that April Abbott had antisynthetase syndrome manifested by diffuse interstitial pulmonary disease, at a time when she was exposed to indoor mold. The cough she reported *immediately* after the mold exposure on September 21, 2018 was, within a reasonable degree of probability, caused by the byproduct of mold metabolism (VOCs) and later interstitial pulmonary fibrosis and DIPNECH. Although the VOC's were likely a factor in the cough becoming manifest at work, the

> cough would not have persisted were it not for the antisynthetase syndrome. At most the VOC's could have caused a temporary aggravation of Ms. Abbott's underlying lung disease but would not have resulted in advancement of the disease beyond ordinary progression.
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> It is not only plausible but likely that the DIPNECH occurred in this case in the setting of preexisting antisynthetase syndrome related diffuse interstitial pulmonary disease, as in other chronic lung conditions associated with inflammation. Antisynthetase antibody seen in patients with pulmonary manifestations of the syndrome has been shown to demonstrate proinflammatory properties suggesting a role for the propagation of disease in genetically susceptible individuals.
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> With the exception of HP, there is no definitive unambiguous firmly grounded *objective* evidence establishing the causation of any other interstitial lung disease from exposure to indoor mold. The only interstitial disease associated with indoor mold exposure is HP, which is rare in subjects exposed to indoor mold. The interval between exposure to mold and a CT scan which revealed extensive interstitial lung disease in this case would imply an acute process (less than 3 weeks). *The diagnostic criteria for acute HP have not been fulfilled in this case.*[4]

At the respondent's request, Philip Harber, M.D., a professor at the University of Arizona, also reviewed the claimant's medical records, but he did not examine the claimant. Dr. Harber is board certified in internal medicine, pulmonary medicine and occupational medicine. In his March 12, 2021 report, Dr. Harber noted diagnoses of DIPNECH and ASILD, both rare conditions. Dr. Harber stated it was unlikely the claimant had ASILD, even though she had interstitial findings and a positive anti-synthetase test, because the claimant did not have myositis or a particular antibody frequently associated with ASILD. He opined the claimant's workplace mold exposure was not the prevailing factor, stating:

> Ms. Abbott's exposure levels and time course are not consistent with the mechanism by which the environmental exposure might hypothetically induce the disease. If indeed the disease was in some way related to the 1-2 days of work moving the equipment, there would have been insufficient time from the exposure to the onset of symptoms to develop the allergic response and subsequently have the body become allergic to altered pulmonary components. Thus, while one of the pulmonary specialists [felt] the two days of exposure was the critical triggering factor, he does not explain the rationale for how this might possibly have occurred. Alternatively, one should consider whether longer-term exposures could have in some fashion triggered the disease. Notably, her exposures were not intense, nor were they uniform across the skilled nursing and hospital facilities. Carefully looking at the available exposure information, which was not apparently available to her

[4] P.H. Trans. (May 12, 2021), Levy Report at 6.

> clinicians who opined on a relationship, shows that there were two distinct problems related to water and mold growth. The more common one was in the basement of the facility, an area of where she very rarely entered. The material present from the water chiller line in the nursing director office and adjacent rooms was likely to be much lower in magnitude. She did not spend much time in these areas, only occasionally visiting the nursing director office or getting supplies from an adjacent storeroom or medication room. Thus, the exposures are likely to be low, brief, and intermittent.
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> . . .
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> Her primary diagnosis is interstitial lung disease of uncertain specific category. It is possible that she has diffuse interstitial pulmonary neuroendocrine cell hyperplasia. Some of her findings are consistent with the anti-synthetase syndrome, whereas others are not. Thus, viewing all the available information, the only certain diagnosis is interstitial lung disease of unknown category; this is not an unusual situation with diseases in this group.
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> I do not believe her employment was a prevailing factor in causing her disease for the reasons discussed in detail above. (While it was very appropriate for Dr. Barkman and others to consider the *possibility*, this is very distinct from converting a possibility, however remote, to a probability. None of the other reports provide a clear rationale for how this might have occurred).[5]

Dr. Hamblin prepared a report dated April 1, 2021. The doctor stressed the claimant did not have preexisting interstitial lung disease prior to her employment for the respondent. Dr. Hamblin wrote, "Consequently, the idea that the presence of an autoimmune disease process negates the contribution of her occupational exposure to her lung disease couldn't be farther from the truth. To make such a claim indicates a lack of knowledge and expertise in interstitial lung disease and the various forces that contribute to the development of autoimmunity."[6]

A third preliminary hearing was held on May 12, 2021. ALJ Hursh ruled:

> The respondent sought to have the claim found not compensable based on new evidence. Following a May 8, 2020 preliminary hearing, the court found, based on the opinion of Dr. Barkman, the pulmonologist treating the claimant, the claimant's interstitial lung disease and anitsynthetase syndrome arose from work related mold exposure. Following a second preliminary hearing, October 1, 2020, the court found the respondent liable for medical treatment for a recent worsening of the condition, including evaluation for a lung transplant.

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[5] *Id.*, Harber Report at 8-9.

[6] *Id.*, Cl. Ex. 11 at 3.

> The respondent's new evidence consisted of reports from two medical/legal experts who did not personally see the claimant, but reviewed her medical records. Dr. Harber is an occupation and environmental medicine specialist. His March 12, 2021 report noted diagnoses of diffuse interstitial pulmonary neuroendocrine cell hyperplasia (DIPNECH) and anti-synthetase syndrome. He described both conditions as rare, and concluded they were probably not related to the claimant's workplace mold exposure. Dr. Harber's report went into great detail about why he reached this conclusion. Essentially, he thought there should have been better reasoning and testing to back up the opinion the conditions were related to the mold exposure.
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> Dr. Levy is an internal medicine and pulmonary disease specialist. His January 5, 2021 report said anti-synthetase syndrome has no known cause. He said DIPNECH, while rare, can result from exposure to fungi, but the only type of indoor fungi that produces it produces a condition called hypersensitivity pneumonitis (HP), which generally requires more lengthy exposure than in the claimant's case. He further said the diagnostic criteria for acute HP were not fulfilled in this case.
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> The two new reports did not so much provide new evidence as point out ways one might poke holes in the testimony of other physicians. The court considers the opinion of Dr. Barkman, who has seen and is treating the claimant, more persuasive. It is still held the claimant's respiratory conditions arose out of and in the course of employment. The previous preliminary orders remain in effect.

The respondent argues the claimant's injury by occupational disease did not arise out of and in the course of her employment. The respondent asserts the claimant's experts' opinions are based on speculation and not actual evidence. The respondent argues the opinions of Drs. Hamblin and Barkman lack scientific basis, and Dr. Barkman only relied on the temporal relationship between the onset of the claimant's cough and her asserted exposure to mold. The respondent argues there is no credible evidence the claimant even inhaled mold spores. Further, the respondent points out Dr. Barkman's use of the word "triggering" shows this claim is not compensable under K.S.A. 44-508(f)(2), which precludes compensability when work is a triggering factor for the injury. The claimant urges the Board to affirm the ALJ's preliminary Order.

PRINCIPLES OF LAW AND ANALYSIS

An employer is liable to pay compensation to an employee incurring personal injury by occupational disease arising out of and in the course of employment.[7] A claimant must

[7] See 44-501b(b).

prove his or her right to an award based on the whole record under a "more probably true than not true" standard.[8]

K.S.A. 44-508 states, in part:

> (e) "Repetitive trauma" refers to cases where an injury occurs as a result of repetitive use, cumulative traumas or microtraumas. The repetitive nature of the injury must be demonstrated by diagnostic or clinical tests. The repetitive trauma must be the prevailing factor in causing the injury. "Repetitive trauma" shall in no case be construed to include occupational disease, as defined in K.S.A. 44-5a01, and amendments thereto.
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> In the case of injury by repetitive trauma, the date of injury shall be the earliest of:
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> (1) The date the employee, while employed for the employer against whom benefits are sought, is taken off work by a physician due to the diagnosed repetitive trauma;
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> (2) the date the employee, while employed for the employer against whom benefits are sought, is placed on modified or restricted duty by a physician due to the diagnosed repetitive trauma;
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> (3) the date the employee, while employed for the employer against whom benefits are sought, is advised by a physician that the condition is work-related; or
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> (4) the last day worked, if the employee no longer works for the employer against whom benefits are sought.
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> In no case shall the date of accident be later than the last date worked.
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> (f)(1) "Personal injury" and "injury" mean any lesion or change in the physical structure of the body, causing damage or harm thereto. Personal injury or injury may occur only by accident, repetitive trauma or occupational disease as those terms are defined.
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> (2) An injury is compensable only if it arises out of and in the course of employment. An injury is not compensable because work was a triggering or precipitating factor. An injury is not compensable solely because it aggravates, accelerates or exacerbates a preexisting condition or renders a preexisting condition symptomatic.

[8] See K.S.A. 44-501b(c) and K.S.A. 44-508(h).

(A) An injury by repetitive trauma shall be deemed to arise out of employment only if:

(i) The employment exposed the worker to an increased risk or hazard to which the worker would not have been exposed in normal non-employment life;

(ii) the increased risk or hazard to which the employment exposed the worker is the prevailing factor in causing the repetitive trauma; and

(iii) the repetitive trauma is the prevailing factor in causing both the medical condition and resulting disability or impairment.

K.S.A. 44-5a01 provides in relevant part:

(a) Where the employer and employee or workman are subject by law or election to the provisions of the workmen's compensation act, the disablement or death of an employee or workman resulting from an occupational disease as defined in this section shall be treated as the happening of an injury by accident, and the employee or workman or, in case of death, his dependents shall be entitled to compensation for such disablement or death resulting from an occupational disease, in accordance with the provisions of the workmen's compensation act as in cases of injuries by accident which are compensable thereunder, except as specifically provided otherwise for occupational diseases. In no circumstances shall an occupational disease be construed to include injuries caused by repetitive trauma as defined in K.S.A. 44-508, and amendments thereto.

(b) "Occupational disease" shall mean only a disease arising out of and in the course of the employment resulting from the nature of the employment in which the employee was engaged under such employer, and which was actually contracted while so engaged. "Nature of the employment" shall mean, for purposes of this section, that to the occupation, trade or employment in which the employee was engaged, there is attached a particular and peculiar hazard of such disease which distinguishes the employment from other occupations and employments, and which creates a hazard of such disease which is in excess of the hazard of such disease in general. The disease must appear to have had its origin in a special risk of such disease connected with the particular type of employment and to have resulted from that source as a reasonable consequence of the risk. Ordinary diseases of life and conditions to which the general public is or may be exposed to outside of the particular employment, and hazards of diseases and conditions attending employment in general, shall not be compensable as occupational diseases . . . .

Abbott sustained a compensable personal injury by repetitive trauma arising out of and in the course of her employment.

Personal injury may occur by accident, repetitive trauma or occupational disease. K.S.A. 44-508(d) and (e) and (f)(2)(A) and (B) specifies the "prevailing factor" requirement applies to accident and repetitive trauma. "Prevailing factor" does not apply to occupational disease claims, at least based on the strict language of our occupational disease statutes.

The greater weight of the evidence supports the claimant's claim. There was mold in the respondent's building, although only the respondent's basement was tested for mold. The claimant was exposed to black mold when she worked each day and developed a gradual worsening of symptoms. The medical evidence generally favors the claimant in terms of proving an injury by repetitive trauma. The treating doctors and Dr. Barkman understood the claimant's lack of a prior condition and her history of work-related exposure to mold. Drs. Barkman and Hamblin concluded the claimant had occupationally-related pulmonary problems. Dr. Barkman indicated the prevailing cause of the claimant's ASILD was mold at her work environment. As such, Dr. Barkman is saying the work-related exposure was the *cause* of the claimant's ASILD, and not a mere triggering event. The claimant's condition improved when away from the respondent's facility.

Dr. Levy's opinion is faulty in assuming the claimant had preexisting anti-synthetase syndrome. There are no medical records to support any such preexisting condition.

Dr. Harber opined the claimant's exposures to mold were insufficient. However, the claimant testified she was exposed to mold multiple times in different areas of the respondent's facility over a ten-month period.

Lastly, the claimant's own testimony supports her assertion that she was repetitively exposed to mold at work. A claimant's own testimony is sufficient to prove her condition.[9]

The claimant proved a compensable injury by repetitive trauma. Here, the employment exposed the worker to an increased risk or hazard to which the worker would not have been exposed in normal non-employment life. The increased risk or hazard to which the employment exposed the worker was the prevailing factor in causing the repetitive trauma. Lastly, the repetitive trauma was the prevailing factor in causing both the claimant's medical condition and resulting disability.

The Board need not explore, at least at this juncture of the claim, the possibility of an injury by occupational disease, because the finding of an injury by repetitive trauma

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[9] See *Graff v. Trans World Airlines*, 267 Kan. 854, 863-64, 983 P.2d 258 (1999); see also *Buchanan v. JM Staffing*, LLC, 52 Kan. App. 2d 943, 956, 379 P.3d 428 (2016), and *Hanson v. Logan U.S.D. 326*, 28 Kan. App. 2d 92, 95, 11 P.3d 1184 (2000), *rev. denied* 270 Kan. 898 (2001).

renders such additional analysis moot. Occupational diseases and injuries by repetitive trauma are mutually exclusive.[10]

WHEREFORE, the undersigned Board Member affirms ALJ Hursh's preliminary Order dated May 13, 2021.

IT IS SO ORDERED.

Dated this _____ day of July, 2021.

_____
JOHN F. CARPINELLI
BOARD MEMBER

Electronic copies via OSCAR to:
William Manson
Benjamin Gary
ALJ Kenneth Hursh

[10] See K.S.A. 44-508(e) and K.S.A. 44-5a01(a).