INTERMEDIATE COURT OF APPEALS
OF WEST VIRGINIA

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HAROLD MURPHY, Decedent
LINDA MURPHY, Dependent

Petitioner,

Intermediate Court No.: 23-ICA-447
JCN: 2020023988
Claim No.: 15089088
BOR Order: 09/11/2023

v.

ST. GOBAIN CERAMICS & PLASTICS,

THE INSURANCE COMMISSIONER
OF WEST VIRGINIA IN ITS CAPACITY
AS ADMINISTRATOR OF THE OLD FUND,

Respondents.

BRIEF OF RESPONDENT
THE INSURANCE COMMISSIONER OF WEST VIRGINIA IN ITS CAPACITY AS ADMINISTRATOR OF THE OLD FUND

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November 13, 2023

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I. STATEMENT OF THE CASE

Linda Murphy ("Petitioner" or "Claimant") appeals the September 11, 2023 Order by the Workers' Compensation Board of Review ("Board of Review" or "BOR"), which affirmed the Claim Administrator's May 20, 2021 order denying Claimant's application for dependent's benefits. Respondent, The Insurance Commissioner of West Virginia in its Capacity as Administrator of the Old Fund ("Old Fund"), submits this brief in response to Claimant's appeal.

This claim arises from an application for dependent's benefits filed by the employee's widow, Linda Murphy ("Claimant"). (Petitioner's Appendix "Pet. Appx." pp. 23-24.) Along with the application, Claimant associated documents including a letter of administration identifying her as the administrator of Decedent's estate. (Pet. Appx. pp. 26-30.)

Claimant also filed a copy of the Death Certificate with the Claim Administrator. (Pet. Appx. p. 25.) The death certificate listed the immediate cause of death as Respiratory Failure. Secondary conditions were listed as chronic obstructive pulmonary disease ("COPD") and occupational lung disease.

An Autopsy Report dated March 3, 2020 (Pet. Appx. pp.32-38), identified "extensive evidence of severe, end-stage disease with marked honeycombing. Patchy areas of anthracosis and silicates are noted, *suggesting* possible exposure to silicates and dust as a contributing factor for the interstitial lung disease." The pathologist wrote that one "pleural plaque is noted, but no definitive ferruginous bodies or asbestos fibers were identified microscopically." The pathologist remarked that the circumstances of the death were not entirely known, but opined that the death was "a result of severe cor pulmonale and respiratory failure in the context of underlying interstitial lung disease." He opined that significant comorbidities contributing to the death included hypertensive and atherosclerotic cardiovascular disease. He stated the autopsy found pleural and pericardial fibrous plaques, which, although nonspecific, have been associated with asbestos exposure. The autopsy found no evidence of mesothelioma or any other malignancy.

The claim was referred to the Occupational Pneumoconiosis Board ("OP Board") whose review included consideration of extensive medical records concerning Mr. Murphy's evaluation and treatment by various providers at various locations from March 29, 2010, until his death on January 5, 2020, as well as the autopsy results.

Upon consideration of the information set forth above, the OP Board issued its findings on March 18, 2021. (Pet. Appx. p.392) The OP Board concluded that

occupational pneumoconiosis was not a material contributing factor in Mr. Murphy's death. The OP Board cited CT scans of the chest dated May 29, 2018 and July 22, 2018 from St. Joseph's Hospital, and a CT scan of the chest dated December 28, 2019 from United Hospital Center. The OP Board found that all studies demonstrated similar findings of honeycombing diffusely in the right lung, with less severe changes in the left. There were no parenchymal nodules, no pleural plaque formation, no pleural calcifications, and no pathological adenopathy. The OP Board noted a dilated esophagus with air fluid level, and a moderate size hiatal hernia. The OP Board explained that honeycombing is a nonspecific finding, which, in the absence of pleural plaques, is most likely idiopathic pulmonary fibrosis. The differential remains broad for the end-stage pulmonary findings, however. The OP Board emphasized that there were no radiographic findings to suggest coal workers pneumoconiosis, or pleural plaques such as would be expected if the pulmonary findings represented asbestosis.

By Order dated May 20, 2021, the Claim Administrator adopted the OP Board's conclusions and denied the claim for dependent's benefits. Claimant filed a timely protest to the Claim Administrator's Order.

Claimant submitted a report dated April 1, 2022, by Ronald E. Gordon, Ph.D. (Pet. Appx. pp. 258-259.) In reviewing lung and lymph node blocks, Dr. Gordon detected in the lung tissue chrysotile and amphibole type asbestos fibers in concentrations of 2,509 fibers per gram wet weight. There were no fibers in the lymph node tissue. Dr. Gordon opined that Decedent "had a mixed occupational asbestos exposure which is documented by his fiber burden of chrysotile and amphibole asbestos fibers, crocidolite, amosite, and tremolite," and that such "was a causative factor for Mr. Murphy's severe asbestosis with

honey combing and pleural plaques." Dr. Gordon opined that Decedent's severe asbestosis caused his right heart to enlarge and develop right heart failure.

The Employer submitted a report dated December 27, 2022, by Robert H. Swedarsky, M.D. (Pet. Appx. pp. 401-440.) Dr. Swedarsky extensively reviewed pertinent medical records and administrative documents, noting that on December 17, 1999, the Office of Judges made a finding of fact that there was no respirable dust hazard at the plant where Claimant was employed since the first study was done there in August 1983. Consequently, Decedent had not been exposed to hazardous levels of dust since he began working there on June 1, 1985. Dr. Swedarsky advised that Dr. Gordon's report was of limited value because it does not meet the guidelines established by the Asbestos Committee of the College of American Pathologists and the Pulmonary Pathology Society. Moreover, the 2509 fibers/g. wet weight found by Dr. Gordon most likely represents nonindustrial ambient exposure, and not disease. Dr. Swedarsky reviewed the lung tissues and found that they reveal nonspecific end-stage honeycombing, and no asbestos bodies. He stated that "criteria for a histologic diagnosis of asbestosis are not met." There were deposits of black pigment and birefringent particles. However, these were scant. Although consistent with exposure, these deposits were insufficient to diagnose mixed dust pneumoconiosis. Dr. Swedarsky explained that honeycomb lung is an end-stage pattern of unclear etiology. Idiopathic pulmonary fibrosis is a condition that can result in honeycomb lung, and Decedent had comorbid conditions which can contribute to honeycomb lung. These included reflux disease with aspiration, pulmonary emboli, congestive heart failure, episodes of pneumonia, and gout. Dr. Swedarsky's

conclusion was that Decedent's "autopsy does not identify an acute process or an immediate cause of death."

The evidence encompasses a voluminous array of medical records, in which may be found references to comorbid conditions such as referenced by Dr. Swedarsky. A chest CT was taken on May 7, 2008, with reference to a clinical history of pneumonia and shortness of breath. CT findings suggested early pneumonic fibrosis, as well as fluid in the esophagus, possibly related to reflux disease and hiatal hernia. (Pet. Appx. p. 450.)

Kevin J. Tveter, M.D. saw Decedent on October 14, 2009, for surgical evaluation of a recent achalasia diagnosis. (Pet. Appx. pp. 394-396.) Reference was made to a history of pneumonia, sleep apnea and GERD. Decedent had recently undergone upper G.I. endoscopy showing dismotility of the esophagus and a hiatal hernia.

At an office visit for routine follow-up on pulmonary fibrosis on April 9, 2015, Ronald Mudry, M.D. noted congestive heart failure, coronary artery disease, GERD, and gout. (Pet. Appx. pp. 460-464.) At that time, Decedent had undergone total joint replacements in one shoulder, one hip, and both knees. Notably, the autopsy report documented chronic tophaceous gout, and reflects that Decedent ultimately had joint replacements of the bilateral shoulders, hips and knees. (Pet. Appx. p. 38.)

A CT scan of the chest was done on October 12, 2015. (Pet. Appx. p. 476.) Findings were suspicious for achalasia of the esophagus. There were extensive chronic lung changes as well as honeycomb lung changes which may suggest chronic interstitial pneumonitis but with some superimposed densities that could represent aspiration pneumonia.

St. Joseph's Hospital records from July 22, 2018 note primary pneumonia as the discharge diagnosis. (Pet. Appx. p. 354.)

On March 11, 2019, Decedent was seen by Jimmy Doumit, M.D. at UHC Pulmonary Clinic, Medbrook Medical Assoc. (Pet. Appx. pp. 6-11.) Conditions then present included congestive heart failure, chronic aspiration, achalasia of esophagus, chronic tophaceous gout, gout, and hiatal hernia with GERD. It was noted that Decedent had been admitted to the hospital in mid-February of that year, and was found to have multiple pulmonary emboli of the right lower, upper, and main pulmonary artery. Dr. Doumit wrote that Decedent's chest CT of July 2018 showed bilateral pulmonary fibrosis, but by far more significant on the right side. Dr. Doumit characterized the fibrosis as "***mainly secondary to chronic aspiration*** contributing to more unilateral fibrosis. This is atypical and not seen in pneumoconiosis."

St. Joseph's Hospital records dated June 24, 2019 (Pet. Appx. pp. 309-350) reflect a history of achalasia of the esophagus, arthritis, chronic systolic congestive heart failure, chronic tophaceous gout, gout, hiatal hernia with GERD, and various other conditions. His current symptoms were concerning for congestive heart failure versus pneumonia. A prior history of pneumonia was noted. (*Id.* at 324.)

Scarring from pneumonia was noted in St. Joseph's Hospital discharge records dated July 22, 2022. (Pet. Appx. p. 358.)

Claimant submitted Dr. Gordon's affidavit, dated February 2, 2023. (Pet. Appx. pp. 539-546.) Dr. Gordon averred that he reviewed Dr. Swedarsky's report. He opined the presence of crocidolite and amosite fibers indicate Decedent had occupational exposure to those commercial fibers, and the level of fibers other than chrysolite demonstrated

occupational exposure. He stated that studies cited by Dr. Swedarsky were problematic in terms of their applicability to this case. He cited the relatively short time between last exposure and analysis in many of the studies, exposure histories differing from that of Decedent, and changes in analysis technique over time. He stated his own methods of tissue analysis are consistent with and in compliance with "commonly accepted principles of fiber burden analysis."[1] He reaffirmed his conclusion that Decedent's lung tissue shows fibrosis, occupational fiber burden[2], and the presence of commercial asbestos fiber types, and therefore Decedent clearly had occupational exposure to asbestos.

The members of the OP Board testified on June 28, 2023. (Pet. Appx. pp. 547-570.) Dr. Leef testified that the OP Board had several CT scans available, emphasizing the most recent scan, dated December 28, 2019. He testified that the high-resolution CT is of good diagnostic quality, and shows a diffuse fibrotic process throughout the lungs with honeycombing. Dr. Leef discussed other findings seen on the scan, then explained that the type of fibrosis pattern present, i.e. the honeycombing, is indicative of idiopathic pulmonary fibrosis. There was no radiographic evidence of calcified pleural plaques to suggest any kind of asbestos exposure. The fibrosis does not have the classic pattern or pathognomic pattern of asbestosis or coalworker's occupational pneumoconiosis.

Dr. Kinder testified that Dr. Gordon's report contained thought-provoking discussion, in which Dr. Gordon attempted to reconcile the end of Decedent's exposure in 1985, his development of breathing problems in May 2008, and the fiber count of approximately 2,000 versus the medical standard of one million fibers to support a finding

[1] But he does not indicate why fibers which have cleared over time would still be important to a current diagnosis if the fibers are no longer present.

[2] He cannot state with any certainty that the source of asbestos exposure is occupational or sufficient to have caused asbestosis

of clinical asbestosis. Dr. Gordon opined that the body's ability to degrade and digest the fibers would yield a very low number of fibers years after exposure ceased[3]. However, Dr. Kinder noted there are no studies of cases so many years removed from exposure, such as to quantify expected fiber counts in individuals such as Decedent. Dr. Kinder stated that the members of the OP Board must opine based on the evidence. In that regard, the CT scan does not have the markers normally seen with occupational exposure. There is not a calcified pleural plaque. One would expect quite a few plaques, and not just one noncalcified plaque. Moreover, the plaque was visceral and not parietal. The plaques in asbestosis or asbestos related pleural disease are parietal. Dr. Kinder "cannot make the connection between this gentleman's pulmonary fibrosis and his occupational exposure." Dr. Gordon's "digestion tissue studies come short of what has been proven and standardized in the past.… There's nothing out there that says there is a standard." Consequently, Dr. Kinder concluded that Dr. Gordon's unproven theory cannot be used to nullify the opinion of Dr. Swedarsky, and Dr. Kinder does not believe Decedent's occupational exposure materially contributed to his death. He made a finding that, in this presumptive case, there is a non-occupational explanation for Decedent's condition, that being idiopathic pulmonary fibrosis.

On cross-examination, Dr. Kinder acknowledged that the medical records reflect Decedent's history of gastroesophageal reflux disease, as well as achalasia, which is an esophageal issue that can contribute to gastroesophageal reflux and possibly aspiration

[3] There is no postulation as to whether any fibers, regardless of length of retention, are important to the analysis of the presence of asbestosis.

pneumonia. However, there is nothing in the medical records that specifically diagnoses pulmonary complications from gastroesophageal reflux disease or achalasia.[4]

Dr. Kayi testified that he concurred with Dr. Kinder's testimony, stating there is no evidence radiologically or "clinically to suggest he has any evidence for asbestosis or any asbestos-related disease, really".

In the BOR's Order dated September 11, 2023 (Pet. Appx. pp. 571-579.), Board Member Patricia A. Jennings weighed the evidence, citing the OP Board's testimony explaining why Dr. Gordon's findings and conclusions did not change the OP Board's opinion. The OP Board was unable to connect Decedent's pulmonary fibrosis to his occupational exposure. She noted although the case is presumptive, the OP Board made a specific finding that there is a nonoccupational explanation for Decedent's disease (i.e., idiopathic pulmonary fibrosis), and none of the imaging evidence was indicative of asbestos or asbestosis.

Board Member Jennings made a conclusion of law that the OP Board's findings are not clearly wrong, and that occupational pneumoconiosis did not materially contribute to Decedents death. Consequently, the BOR affirmed the Claim Administrator's order of May 20, 2021, which denied dependents benefits. Claimant appealed. The Old Fund submits this brief in response to Claimant's appeal.

II. SUMMARY OF ARGUMENT

The preponderance of the evidence proves that occupational pneumoconiosis did not materially contribute to decedent's death. The OP Board's opinion was consistent with that of Dr. Swedarsky and the OP Board provided sufficient explanation for their finding

[4] But see radiology that mentions pulmonary changes likely related to GERD.

that occupational pneumoconiosis did not materially contribute to the decedent's death. Claimant fails to demonstrate the findings of fact of the BOR are manifestly against the weight of evidence. *See* W. Va. Code § 23-5-12a(b).

III. STATEMENT REGARDING ORAL ARGUMENT AND DECISION

Respondent submits that the facts and legal arguments are adequately presented in the briefs and record on appeal, and the decisional process would not be significantly aided by oral argument.

IV. ARGUMENT

A. Standard of Review

The Intermediate Court of Appeals "shall reverse, vacate, or modify [an] order or decision of the Workers' Compensation Board of Review, if the substantial rights of the petitioner or petitioners have been prejudiced because the Board of Review's findings are: (1) [i]n violation of statutory provisions; (2) [i]n excess of the statutory authority or jurisdiction of the Board of Review; (3) [m]ade upon unlawful procedures; (4) [a]ffected by other error of law; (5) [c]learly wrong in view of the reliable, probative, and substantial evidence on the whole record; or (6) [a]rbitrary or capricious or characterized by abuse of discretion or clearly unwarranted exercise of discretion." W. Va. Code §23-5-12a(b).

B. The Board of Review was not clearly wrong in finding Claimant failed to demonstrate that occupational pneumoconiosis did not materially contribute to decendent's death

A claimant in a workers' compensation proceeding has the burden of proving his claim. *See e.g.*, Syl. pt. 2, *Clark v. State Workmen's Compensation Com'r*, 155 W. Va. 726, 187 S.E.2d 213, 214 (1972); Syl pt. 1, *Staubs v. S.W.C.C.,* 153 W. Va. 337, 168 S.E.2d 730 (1969). "Pursuant to W.Va. Code § 23-4-1g(a) (2003) (Repl. Vol. 2010), a

claimant in a workers' compensation case must prove his or her claim for benefits by a preponderance of the evidence." Syllabus point 1, *Arch Coal, Inc. v. Jimmie Lemon*, 240 W.Va. 650, ___, 814 S.E.2d 667, 668 (2018)(quoting Syl. pt. 2, *Gill v. City of Charleston*, 236 W.Va. 737, 783 S.E.2d 857 (2016)). Great deference is given to the OP Board due to the fact that the physicians who sit as members on the OP Board have special expertise with regard to pulmonary disease, including occupational pneumoconiosis. *Newman v. Richardson*, 186 W. Va. 66, 410 S.E.2d 705, 708-709 (1991). Indeed, the Office of Judges[5] must affirm the Decision of the Occupational Pneumoconiosis Board made following hearing unless the Decision is clearly wrong in view of the reliable, probative and substantial evidence on the whole record. W. Va. Code § 23-4-6a. A decision of the OP Board is clearly wrong if it is not supported by the evidence of record, is clearly against a preponderance of evidence, or is based upon evidence which is speculative and inadequate to sustain the Decision of the Board. See Gibson v. State Compensation Commissioner, 127 W. Va. 97, 31 S.E.2d 555 (1944); Estep v. State Compensation Commissioner, 130 W. Va. 504, 44 S.E.2d 305 (1947); Barnette v. State Workers' Compensation Commissioner, 153 W. Va. 796, 172 S.E.2d 698 (1970); Smith v. State Workers' Compensation Commissioner, 155 W. Va. 883, 189 S.E.2d 838 (1972).

In this claim, the decision of the OP Board is not clearly wrong and is well supported by the evidence of record and the OP Board's testimony at hearing. Dr. Kinder reviewed the radiological evidence of record and indicated that he reviewed several CT scans which showed a diffuse fibrotic process throughout the lungs with honeycombing of a pattern indicative of idiopathic pulmonary fibrosis. He saw no calcified pleural plaques

[5] References to the Office of Judges in WV Code Chapter 23 also refer to the WV Workers' Compensation Board of Review due to the recent structural changes to the Workers' Compensation litigation system.

to suggest any kind of asbestos exposure radiographically. He stated that the of pleural and pericardial plaques on autopsy did not affect his opinion because the evidence was not present radiographically. Dr. Kinder added in his testimony that with regard to plaques, there were no ***calcified*** pleural plaques – not just one non-calcified plaque, noting additionally that the plaque was visceral and not parietal as typically seen in asbestosis or asbestos related pleural disease.

Dr. Kinder clearly testified that none of the experts made a diagnosis of asbestosis based on a finding of asbestos bodies or even fiber count. He stated that the digestion tissue studies came short of what has been proven and standardized in the past, and Dr. Gordon has nothing other than his proposal of degradation related to half-life to suggest that his theory about the fiber count is standard. Dr. Kinder found the theory interesting but without additional scientific support, he could not use Dr. Gordon's opinion to nullify that of Dr. Swedarsky which was consistent with the scientific standard and consistent with the OP Board's findings. Dr. Swedarsky reviewed the lung tissues and found that they revealed nonspecific end-stage honeycombing, and no asbestos bodies. He stated that "criteria for a histologic diagnosis of asbestosis are not met." Essentially, Dr. Kinder identified Dr. Gordon's hypothesis as speculative and unproven by additional studies to suggest that the decedent had an occupational lung disease that contributed to his death. By contrast, Dr. Swedarsky advised that Dr. Gordon's report was of limited value because it does not meet the guidelines established by the Asbestos Committee of the College of American Pathologists and the Pulmonary Pathology Society. Moreover, the 2509 fibers/g. wet weight found by Dr. Gordon most likely represent nonindustrial ambient exposure, and not disease. Dr. Swedarsky reviewed the lung tissues and found that they

reveal nonspecific end-stage honeycombing, and no asbestos bodies. He stated that "criteria for a histologic diagnosis of asbestosis are not met." There were deposits of black pigment and birefringent particles. However, these were scant. Although consistent with exposure, these deposits were insufficient to diagnose mixed dust pneumoconiosis. Dr. Swedarsky explained that honeycomb lung is an end-stage pattern of unclear etiology. Idiopathic pulmonary fibrosis is a condition that can result in honeycomb lung, and decedent had comorbid conditions which can contribute to honeycomb lung. These included reflux disease with aspiration, pulmonary emboli, congestive heart failure, episodes of pneumonia, and gout. His finding of idiopathic pulmonary fibrosis is consistent with the expert opinions of the OP Board.

In fact, Dr. Kinder testified that although he couldn't identify a specific condition or cause within those that could result in idiopathic pulmonary fibrosis, he did note that if there were studies that showed gastroesophageal reflux, aspiration, pneumonia from aspiration, or gout, they would provide alternative diagnoses. But, he definitively confirmed that none of the evidence on x-ray or CT were indicative of asbestosis or asbestosis. Dr. Kinder did note, however, that there was evidence of gastroesophageal reflux disease as a risk factor. Dr. Kinder also acknowledged that the medical records reflect Decedent's history of achalasia, which is an esophageal issue that can contribute to gastroesophageal reflux and possibly aspiration pneumonia. In addition to multiple records noted above citing a history significant for gout and GERD (gastroesophageal reflux disease), the record also contains the March 11, 2019, note when Decedent was seen by Jimmy Doumit, M.D. at UHC Pulmonary Clinic, Medbrook Medical Assoc. (Pet. Appx. pp. 6-11.) Conditions then present included congestive heart failure, chronic

aspiration, achalasia of esophagus, chronic tophaceous gout, gout, and hiatal hernia with GERD. It was noted that Decedent had been admitted to the hospital in mid-February of that year, when he was noted to have multiple pulmonary emboli of the right lower, upper, and main pulmonary artery. Dr. Doumit wrote that Decedent's chest CT of July 2018 showed bilateral pulmonary fibrosis, but by far more significant on the right side. Dr. Doumit characterized the fibrosis as "***mainly secondary to chronic aspiration*** contributing to more unilateral fibrosis. This is atypical and not seen in pneumoconiosis." These observations are consistent with those of the OP Board.

The overwhelming evidence of record reflects that the Claim Administrator properly denied Claimant's application for dependent's benefits. The Occupational Pneumoconiosis Board Findings, as indicated above, found that occupational pneumoconiosis was not a material contributing factor in Mr. Murphy's death. The findings provide a detailed list of the records available for the OP Board's review, and it is clear that the medical records were extensive and covered a critical period through the time of Mr. Murphy's death. The OP Board testified further that occupational dust exposure was not a material contributing factor to Mr. Murphy's death and his fibrosis was a result of idiopathic pulmonary fibrosis and the clinical picture in the records and after a review of the expert reports did not comport with a finding of occupational pneumoconiosis as a material contributing factor to the decedent's death..

Claimant has not met her burden of proof. She has not established by a preponderance of the evidence that she is entitled to dependent's benefits because occupational pneumoconiosis or occupational dust exposure was a material contributing factor in her husband's death. The Claim Administrator properly denied dependent's

benefits. The December 22, 2015 Order denying Claimant's application for dependent's benefits should be affirmed.

For the above reasons, the Employer respectfully requests that the Intermediate Court of Appeals affirm the September 11, 2023 Decision of Workers' Compensation Board of Review.

V. CONCLUSION

Claimant failed to show that the BOR's Order was in violation of statutory provisions, in excess of the statutory authority or jurisdiction of the BOR, made upon unlawful procedures, affected by other error of law, clearly wrong in view of the reliable, probative, and substantial evidence on the whole record, or arbitrary or capricious or characterized by abuse of discretion or clearly unwarranted exercise of discretion. For the foregoing reasons, the Respondent requests this Court affirm the BOR's September 11, 2023 Order that affirmed the Claim Administrator's May 20, 2021 order, which denied the claim for dependent's.

THE INSURANCE COMMISSIONER OF WEST VIRGINIA IN ITS CAPACITY AS THE ADMINISTRATOR OF THE OLD FUND

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CERTIFICATE OF SERVICE

I, Karin L. Weingart, do hereby certify that the foregoing "**BRIEF OF THE INSURANCE COMMISSIONER OF WEST VIRGINIA IN ITS CAPACITY AS ADMINISTRATOR OF THE OLD FUND"** has been served upon all parties via electronic filing on this 13th day of November 2023 as follows:

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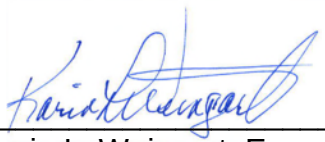

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