

IN THE COURT OF APPEALS OF NORTH CAROLINA

No. COA18-770

Filed: 3 September 2019

N.C. Industrial Commission, I.C. No. 240941

GAIL CANUP HINSON, Executrix of the ESTATE OF WALTER DUNBAR HINSON,  
Deceased-Employee, Plaintiff-Appellant,

v.

CONTINENTAL TIRE THE AMERICAS, SELF-INSURED, Employer-Defendant-  
Appellee.

PART OF THE CONTINENTAL TIRE THE AMERICAS CONSOLIDATED  
ASBESTOS MATTERS.

Appeal by Plaintiff from opinion and award entered 25 January 2018 by the  
North Carolina Industrial Commission. Heard in the Court of Appeals 12 March  
2019.

*Wallace and Graham, PA, by Edward L. Pauley, for Plaintiff-Appellant.*

*Fox Rothschild LLP, by Jeri L. Whitfield and Lisa K. Shortt, for Defendant-  
Appellee.*

McGEE, Chief Judge.

This appeal is companion to four additional appeals, COA18-766, COA18-767,  
COA18-768, and COA18-769 (all five together, the “bellwether cases”), consolidated  
for hearing by order of this Court entered 8 June 2018. The four companion appeals  
will be decided by opinions filed concurrently with this opinion.

I. Procedural History

Decedent Walter Dunbar Hinson (“Plaintiff Hinson”) worked for Continental Tire the Americas (“Defendant”) at Defendant’s tire factory (the “factory”) in Charlotte from 1967 until 1999.<sup>1</sup> This case and the other bellwether cases involve workers’ compensation claims based on allegations that Plaintiff Hinson, along with the additional four plaintiffs<sup>2</sup> in the bellwether cases (“Bellwether Plaintiffs”), were exposed to levels of harmful airborne asbestos sufficient to cause asbestos-related diseases, including asbestosis.<sup>3</sup> The bellwether cases constitute a small percentage of a much larger number of related claims that were consolidated by the Industrial Commission (the “consolidated cases”).<sup>4</sup> Determination of the bellwether cases will impact not only the Bellwether Plaintiffs, but also the remaining plaintiffs from the consolidated cases (together with the Bellwether Plaintiffs, “Plaintiffs” or “Consolidated Plaintiffs”). The Full Commission (the “Commission”) explained the unique procedure that was adopted to handle the large volume of consolidated cases in five opinions and awards, entered on 25 January 2018, that decided the bellwether cases:

This case is part of a large group of cases (currently numbering 144) alleging occupational exposure to asbestos at [the] factory. The large group of [P]laintiffs contends

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<sup>1</sup> The factory was initially operated under the General Tire name.

<sup>2</sup> Douglas M. Epps, Bobby James Newell, Frank Lee Welch, and Charles Edward Wilson.

<sup>3</sup> Plaintiff Hinson filed a Form 18B with the Industrial Commission, completed 23 May 2002, alleging he had developed asbestosis as a result of exposure to asbestos while an employee at the factory.

<sup>4</sup> The Commission’s 25 January 2018 opinion and award states that there were “currently” 144 consolidated cases. However, the number of consolidated cases has fluctuated. Both Plaintiffs and Defendant moved to consolidate these cases.

that they developed asbestos-related disease, primarily asbestosis, caused by exposure to asbestos at the . . . factory[.] Defendant denied that the diagnoses of asbestosis were valid, and also denied that any employee could develop an asbestos-related disease as a result of employment with [D]efendant because there was insufficient exposure to asbestos in [the] factory.

[The consolidated cases] were postured so that there would be an “initial six” cases to be tried as bellwether cases. Although the 144 cases had many issues and facts in common, it was an impossibly large number to try individually, and too difficult to manage in one joint hearing. Therefore, [P]laintiffs’ counsel selected a group of six representative bellwether cases to be tried together in a consolidated manner. The evidence presented in this consolidated hearing regarding the factory, [asbestos] exposures to employees, the criteria for the diagnosis of asbestosis, the scientific evidence regarding asbestos exposure, and the potential for disease causation would be common to, and thus universally applicable to, all 144 claims. The parties agreed that evidence on the general issues was to be part of the record for all [consolidated cases], to the extent the evidence was applicable to each [P]laintiff’s issues. The [B]ellwether [P]laintiffs’ individual medical and employment histories would be addressed, as would scientific evidence applicable to all 144 claims regarding asbestos-related-disease-causing capabilities, including the exposure and medical causation testimony. In addressing the bellwether cases first and presenting evidence applicable to all extant claims, the assumption was that after the six cases proceeded through trial, decision and appeal, the parties would be in a better position to evaluate the remaining claims. The remaining [consolidated cases] could then be potentially resolved, or they could proceed to abbreviated hearings for the introduction of evidence regarding their individual medical and employment information.

One of these “initial six” [Bellwether P]laintiffs, Kirkland . . . , filed a Notice of Voluntary Dismissal with Prejudice on

13 November 2012. This left five Bellwether Plaintiffs to proceed through trial, decision, and appeal.<sup>5</sup> While under the jurisdiction of former Deputy Commissioner George Glenn, these matters were set on a course unlike that of most workers' compensation cases, in that each side was given the opportunity to have a "full trial on the science"—with freedom to prosecute the cases according to the civil procedure used in superior court. The parties were permitted to take as many pre-hearing depositions as they wished and could call as many hearing witnesses as they determined to be necessary. The [B]ellwether [P]laintiffs' cases were heard together in a consolidated posture by former Deputy Commissioner Gheen on a special-set basis in various locations over the course of thirty-eight hearing days beginning 14 February 2011 and concluding 18 February 2013. Former Deputy Commissioner Gheen's hearing of these claims also involved substantial pre-trial proceedings.<sup>[6]</sup> Much of the evidence presented was "common" evidence applicable to all 144 extant claims.

. . . . The Full Commission has reviewed and considered all hearing and deposition transcripts, along with all evidentiary exhibits, arguments, and briefs in reaching a decision in this claim.

After hearings had already commenced, the deputy commissioner entered a 27 July 2012 order requiring that "Plaintiffs who die during the pendency of these claims shall have at least 30 blocks of lung tissue preserved for autopsy and examination by an expert of Defendant's choice." The deputy commissioner based this order on the following findings and reasoning:

[Defendant] denies that any of its employees, including claimants, would have had sufficient exposure to asbestos

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<sup>5</sup> These five Bellwether Plaintiffs are the five Plaintiffs currently before us in the associated appeals.

<sup>6</sup> Three different deputy commissioners had been involved in the consolidated cases through entry of the initial opinions and awards for the bellwether cases by the deputy commissioner.

from working at its facility to either cause or contribute to an asbestos related disease. It has presented the testimony of multiple credible expert witnesses in support of this defense.

[] Plaintiffs' claims against [Defendant] are based, in part, on a "B-read" of an x-ray provided by Plaintiffs' expert.<sup>[7]</sup> As testified by the medical experts, radiological studies are only effective at identifying abnormal features on the x-ray that may be consistent with the disease of asbestosis, but also may be consistent with multiple other lung diseases. In order to make a diagnosis of asbestosis, a physician is called upon to rule out other possible conditions.

[] The medical experts representing both parties have repeatedly testified that the only way to positively identify whether or not a lung condition or other cancer is caused by asbestos exposure is to take a sample of and examine the actual lung tissue. However, due to the risks involved, this procedure is not done while the patient is alive; it is commonly performed at autopsy.

Therefore, the deputy commissioner ordered that Plaintiffs save lung tissue of any Plaintiffs who died so that their lung tissue could be examined. Plaintiffs did not fully comply with this order.

The deputy commissioner reasoned in a 30 April 2013 order: "The diagnoses [of asbestosis], or lack thereof, by the experts is based on the reading of the same radiology. Both sides argue the veracity of their own experts." "Given the opposing medical findings, . . . the undersigned Deputy Commissioner suggested to the parties" that they "jointly agree to independent medical experts or to experts chosen

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<sup>7</sup> See findings of fact 25 to 28, below, for an explanation of the "B-read" process.

by the Industrial Commission to review the radiology and any other relevant medical evidence, which experts' opinion both parties would accept as final." "Alternatively the parties debated whether the Plaintiffs should be compelled to submit to a high resolution computed tomography (hereinafter 'HRCT') scan to be interpreted by a physician selected by the Commission in order to determine the presence or absence of asbestosis." Defendant agreed to the suggestion, and agreed to pay for the HRCT scans and associated costs, but Plaintiffs did not agree.

During the hearings, "[m]uch of the evidence presented was 'common' evidence applicable to all 144 extant claims." Due to the resignation of the deputy commissioner who had presided over the hearings, the consolidated cases were assigned to a different deputy commissioner on 15 April 2015. Plaintiffs and Defendant completed submission of evidence to the deputy commissioner, and made their closing oral arguments on 26 and 27 January 2016. The deputy commissioner filed his opinions and awards in the bellwether cases on 19 December 2016, denying the claims of all five Bellwether Plaintiffs. Plaintiffs appealed to the Full Commission on 21 December 2016. The Commission heard the matters on 29 June 2017, and also denied Plaintiffs' claims by five opinions and awards entered 25 January 2018. The five 25 January 2018 opinions and awards filed in the consolidated cases each contain findings of fact common to all claims, which also include the ultimate findings and conclusions of law common to all claims. Following the common findings and conclusions, each of the five opinions and awards before us contain findings of fact

and conclusions of law sections that are specific to each individual Bellwether Plaintiff, as well as the Commission's rulings denying each of the Bellwether Plaintiffs' claims.

Bellwether Plaintiffs appealed, and Plaintiffs and Defendant filed a motion with this Court on 30 May 2018 requesting consolidation of the bellwether cases for appeal.<sup>8</sup> This Court ordered that a single record be submitted for all five bellwether cases, and that: "The parties shall each submit one general brief addressing common issues and five specific briefs addressing individual [P]laintiff issues." Plaintiffs and Defendant each filed a single "general brief"—ostensibly the "general brief addressing common issues" ordered by this Court. Plaintiffs' general brief is in reality the statement of facts for Plaintiffs' individual briefs. In addition, each of the five Bellwether Plaintiffs filed "specific" individual appellant briefs that are nearly identical, and almost exclusively argue common issues. Defendant responded to the Bellwether Plaintiffs' individual briefs by filing five separate appellee briefs addressing the issues specific to each of the five Bellwether Plaintiffs. Although Plaintiffs' individual briefs do not address the "common issues" separately from the "individual issues," we address all Plaintiffs' arguments concerning the "common issues" that were decided in the Commission's 25 January 2018 opinions and awards in this opinion—COA18-770. Our holdings for the "common issues" will be

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<sup>8</sup> Because the "common issues" sections of the 25 January 2018 opinions and awards apply to all Consolidated Plaintiffs, we treat them as appellants as well.

incorporated by reference in our opinions for the remaining four bellwether cases—COA18-766, COA18-767, COA18-768, and COA18-769. The “individual issues” will be addressed separately in each opinion.

## II. General Factual History

Plaintiffs all allege they were exposed to asbestos while working at the factory, and further allege they developed compensable asbestos-related diseases as a result. As explained in a *Fact Sheet* published by the National Cancer Institute (“NCI”)—which was entered into evidence:<sup>9</sup>

Asbestos is the name given to a group of minerals that occur naturally in the environment as bundles of fibers that can be separated into thin, durable threads. These fibers are resistant to heat, fire, and chemicals and do not conduct electricity. For these reasons, asbestos has been used widely in many industries.

. . . .

Asbestos minerals are divided into two major groups: Serpentine asbestos and amphibole asbestos. Serpentine asbestos includes the mineral chrysotile, which has long, curly fibers that can be woven. Chrysotile asbestos is the form that has been used most widely in commercial applications. Amphibole asbestos has straight, needle-like fibers that are more brittle than those of serpentine asbestos and are more limited in their ability to be fabricated.

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<sup>9</sup> We include this NCI publication as a general introduction to asbestos, asbestos-exposure, and related disease. This is just one piece of evidence considered by the Commission—it was not specifically adopted by the Commission in its opinions and awards.



National Cancer Institute, U.S. Department of Health and Human Services, *Fact Sheet: Asbestos Exposure and Cancer Risk* 1 (1 May 2009) (“NCI *Fact Sheet*”)

(citations omitted). According to the NCI *Fact Sheet*:

People may be exposed to asbestos in their workplace, their communities, or their homes. If products containing asbestos are disturbed, tiny asbestos fibers are released into the air. When asbestos fibers are breathed in, they may get trapped in the lungs and remain there for a long time. Over time, these fibers can accumulate and cause scarring and inflammation, which can affect breathing and lead to serious health problems.

. . . . According to [the International Agency for Research on Cancer], there is sufficient evidence that asbestos causes mesothelioma (a relatively rare cancer of the thin membranes that line the chest and abdomen), and cancers of the lung, larynx, and ovary. Although rare, mesothelioma is the most common form of cancer associated with asbestos exposure. There is limited evidence that asbestos exposure is linked to increased risks of cancers of the stomach, pharynx, and colorectum.

Asbestos exposure may also increase the risk of asbestosis (an inflammatory condition affecting the lungs that can cause shortness of breath, coughing, and permanent lung damage) and other nonmalignant lung and pleural disorders, including pleural plaques (changes in the membranes surrounding the lung), pleural thickening, and benign pleural effusions (abnormal collections of fluid between the thin layers of tissue lining the lungs and the wall of the chest cavity).

. . . .

Everyone is exposed to asbestos at some time during their life. Low levels of asbestos are present in the air, water, and soil. However, most people do not become ill from their exposure. People who become ill from asbestos are usually

those who are exposed to it on a regular basis, most often in a job where they work directly with the material or through substantial environmental contact.

....

Although it is clear that the health risks from asbestos exposure increase with heavier exposure and longer exposure time, investigators have found asbestos-related diseases in individuals with only brief exposures. Generally, those who develop asbestos-related diseases show no signs of illness for a long time after exposure. It can take from 10 to 40 years or more for symptoms of an asbestos-related condition to appear.

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Several factors can help to determine how asbestos exposure affects an individual, including:

- Dose (how much asbestos an individual was exposed to).
- Duration (how long an individual was exposed).
- Size, shape, and chemical makeup of the asbestos fibers.
- Source of the exposure.
- Individual risk factors, such as smoking and pre-existing lung disease.

Although all forms of asbestos are considered hazardous, different types of asbestos fibers may be associated with different health risks. For example, the results of several studies suggest that amphibole forms of asbestos may be more harmful than chrysotile, particularly for mesothelioma risk, because they tend to stay in the lungs for a longer period of time.

*Id.* at 2-3 (citations omitted). The NCI *Fact Sheet* also states that initial examination for someone who suspects they may have an asbestos-related disease would generally

include “[a] thorough physical examination, including a chest x-ray and lung function tests[.] . . . . Although chest x-rays cannot detect asbestos fibers in the lungs, they can help identify any early signs of lung disease resulting from asbestos exposure.” *Id.* at 4 (citations omitted). However, the NCI further stated: “A lung biopsy, which detects microscopic asbestos fibers in pieces of lung tissue removed by surgery, is the most reliable test to confirm exposure to asbestos.” *Id.*

Plaintiffs worked in different sections of the factory, but all allege they were exposed to airborne asbestos in quantities and of a type sufficient to cause asbestos-related diseases—primarily asbestosis. The Commission made the following relevant findings of fact related to the “common issues” raised by Plaintiffs’ claims:

1. Asbestos is a generic term for a group of six naturally-occurring, fibrous silicate minerals that are ubiquitous in ambient air. The general public is exposed to asbestos from natural and artificial sources through food, water, and in other ways. The background level of asbestos to which the general public is exposed varies based on several factors including geography and proximity to urban centers. Low levels of asbestos can be found in the lungs of virtually 100% of the general population. [N.C.G.S.] § 97-62 defines asbestosis as “a characteristic fibrotic condition of the lungs caused by the inhalation of asbestos dust.”

2. Plaintiffs allege that they contracted asbestosis caused by exposure to airborne asbestos during employment with [D]efendant at the . . . factory[.] Additionally, some Plaintiffs allege that they also contracted diseases other than asbestosis caused by exposure to airborne asbestos during employment at [the] factory. Asbestos is not a tire component. The [P]laintiffs allege workplace exposure in the factory from one or more of four main sources: 1) airborne asbestos originating from damaged or

deteriorated asbestos-containing pipe insulation; 2) powdered talc allegedly contaminated with asbestos used as a non-stick agent in certain areas of the factory; 3) asbestos-containing dust released into the air by sawing and/or otherwise working with asbestos-containing gaskets; and 4) airborne asbestos-containing brake dust that allegedly emanated from forklifts and other factory vehicles during maintenance and use. Plaintiffs allege that they were exposed to asbestos through one or more of these methods in such form and quantity and with such frequency that it caused asbestosis.

3. The . . . factory was constructed in the late 1960s and began manufacturing tires by 1969. . . . The factory ceased tire production on 4 July 2006. . . .

4. The tire-making process began in the Banbury/mixing department, a three-story area open to the rest of the factory. On the top floor of the Banbury/mixing area, chemicals and rubber were received, weighed, and mixed. On the second floor of this area, these raw materials were put into heated mixing machines. From these mixers the material was dropped down chutes to the mills on the main floor. The mills pressed the chunks of rubber material into sheets. The sheets of rubber were then hung on a line and dried using fans. Once dry, the sheets were put on pallets and sent to the “calendaring and extruding” area.

5. In the calendaring and extruding area, the rubber material was compressed into different thicknesses, shapes, and sizes for eventual use as the different components of a tire. . . . The compressed rubber was then transferred to the “stock prep” area, where it was cut to the correct dimensions for tire building.

6. In the “tire building” area, all of the tire pieces were layered together and pressed in a tire-building machine. . . . The “green tires” were then transported to the curing department.

7. There were 147 clam-shell-shaped curing presses/ovens

in the curing department. . . . The curing process, during which the “green tires” were placed in a mold and vulcanized under heat and pressure, was very hot and was operated by steam. For this reason, the curing area had more condensate and steam piping than any other area in the factory. Much of this piping was located in trenches that ran to the curing presses/ovens.

8. After curing, the tires went to the “final finish” area where they were trimmed, cleaned, and inspected. The tires that passed inspection were put on pallets and transported to the warehouse[.]

9. Steam and condensate pipes ran throughout the factory. . . . There were at least 26,180 linear feet of insulated steam and condensate piping in the factory. The insulation was comprised of one to two inches of an asbestos-containing cement, Thermobestos, encapsulating the steam and condensate pipes. The pipes had protective canvas and glue surrounding the Thermobestos. Asbestos insulation was removed from the market in the early 1970s and, as such, expansions at the . . . factory after a certain date would not have included the installation of asbestos-encapsulated piping. Most of the insulated steam and condensate piping was at ceiling level, 20-30 feet above the factory floor, or below floor level in the trenches that ran between and into the curing presses. The floor-level and trench-level pipe insulation was susceptible to damage by foot traffic. Forklifts could damage floor-level pipe insulation and also could damage pipe insulation at higher levels. For example, while stacking tires high in the warehouse, it was possible for the forklift payloads to strike the insulated piping.

10. Plaintiffs allege exposure to airborne asbestos originating from deteriorated pipe insulation. Plaintiffs allege that it was damaged through external molestation by workers walking on pipes, climbing on pipes, and striking the pipes with forklifts and forklift payloads. Plaintiffs also argue that internal pipe damage from ruptures forced steam to leak out of the pipes with

sufficient force to cause insulation damage and cause asbestos to become airborne. Plaintiffs further allege that workers used compressed air near the damaged insulation, causing asbestos to become airborne. There is conflicting evidence regarding the amount of pipe insulation damage present at [the] factory.

11. The highest concentration of insulated piping in the factory existed in the curing department, with much of the piping at or below floor level. Plaintiffs allege that workers used a band saw to cut large asbestos-containing gaskets in the curing department. If [P]laintiffs' allegations are correct, it would be logical to expect high levels of airborne particulates in the curing department originating from damaged pipe insulation and gasket-sawing. However, the greater weight of the evidence does not support this conclusion.

12. In 1979, the . . . factory took part in an air contaminant assessment study in conjunction with The National Institute for Occupational Safety and Health (hereinafter "NIOSH"). At the time, NIOSH was studying the best methods and technologies to control air quality in the tire industry. The report reflects that [the] factory was selected for the study because it had "among the better controls for air contaminants in the industry." NIOSH performed area and personal air monitoring in each area of the plant that it expected to find measurable dust or emissions. Specifically, NIOSH measured for dust—both airborne and respirable, as well as petroleum distillates, rubber solvent, Benzene, and Toulene. The dust measurements would have measured any particulates in the air—whether the particulates were asbestos, talc, or something else. The 1979 NIOSH dust measurements found that the measured dust levels in the curing department were 1/100th of the permissible level. This was possibly due to the curing department's powerful exhaust system, which drew air up and out of the area. Except for an outlier measurement created by an employee jumping up and down in a dusty trash bin, the 1979 dust measurements at [the] factory were five to ten times less

than the permissible exposure level (hereinafter “PEL”) in place in 2013. NIOSH concluded that the particulate and vapor concentrations at [the factory] were well below the PEL established by the Occupational Safety and Health Administration (hereinafter OSHA), NIOSH, and the American Conference of Governmental Industrial Hygienists. NIOSH also concluded that the environmental controls (exhaust and ventilation systems) were effective.

13. There was also environmental air sampling for asbestos at [the] factory in 1985 when asbestos-containing insulation was removed from a furnace on the third floor of the mixing area. This sampling was done with background air monitoring as well as with personal air monitors on the personnel conducting the removal. In 1985, there were no regulations regarding wetting down insulation as it was removed. Therefore, the air measurements taken during this removal process record a scenario very favorable for the creation of airborne dust. However, the 1985 background air monitoring that took place showed results well below the then-current OSHA PEL. The highest recorded personal air monitoring result during the removal was also below the then-existing OSHA PEL.

14. As a result of the 1986 federal asbestos regulations, large-scale asbestos abatement procedures were undertaken at [the] factory. This process required pre-abatement area air quality monitoring to measure pre-removal levels. For this reason, there were background air samples collected for abatement projects in 1989 (curing), 1995 (calendar and extruding), and 2003 (powerhouse). In all of these areas, these measurements show that at no time was the potential exposure above the OSHA PEL. Background monitoring reflected levels to which the public at large is exposed.

15. In areas with ceiling-level piping, such as the warehouse, the evidence demonstrates that any small amount of asbestos potentially disturbed and released at ceiling level due to pipe insulation damage would have dissipated before reaching workers and would not have

created any meaningful exposure.

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17. Plaintiffs also allege exposure to asbestos through the inhalation of powdered talc which they allege contained asbestos. Talc is used ubiquitously by the general population in things such as makeup and baby powder. It is the most common non-asbestos mineral found in general population lung tissue. Talc was used in [the] factory as a non-stick agent. However, the amount of talc used in the . . . factory is a contested factual issue. Defendant avers that routinely, workers mistook other powdery materials used in great quantities at the factory for talc. Specifically, [D]efendant contends that clay [kaolin], calcium carbonate, and zinc oxide were commonly used in vastly larger quantities and were routinely incorrectly referred to by the workers as “talc.” ....

18. While talc from certain mines is known to be contaminated by asbestos, there was disagreement among the experts regarding the likelihood of asbestos being a contaminant in the talc used at [the] plant. Furthermore, in 1995, air monitoring was done in the calendaring area while calendaring work continued. Plaintiffs allege significant talc usage in this area. If [P]laintiffs’ allegations are correct, it would be logical to expect high levels of airborne particulates in calendaring. However, the 1995 measurements, performed as calendaring work continued, found airborne particulate levels well below the then-existing OSHA PEL, and EPA clearance levels.

....

20. .... There was contradicting testimony on the issue of cutting gaskets—with some witnesses testifying that gaskets came from the manufacturer already made to fit and did not require any sawing and other testimony that any such sawing, if it took place, would have been done in the maintenance shop, not in curing.



. . . .

24. In 1986, federal government regulations mandated new procedures to identify, encapsulate, and abate workplace asbestos. As part of these new regulations, in 1987, certain employees at [the] factory were trained for the possibility that small asbestos removals would have to be performed by [Defendant's] employees. . . . All removal and abatement procedures were performed by outside contractors. Subsequent to the 1986 regulations, the new asbestos management policies were made known to all employees, masks were provided, and safety protocols, such as the prohibition of using compressed air on damaged insulation, were enacted. Furthermore, known asbestos-containing materials were labeled, encapsulated, and removed.

25. An asbestosis B-read is a test in which NIOSH-certified physicians view a patient's chest x-ray and score it from 0/0 (for normal lungs) through 3/3 (for lungs with severe disease). B-readers become certified (and re-certified every 4 years) based on their tested proficiency in scoring a set of standard x-rays. The first number in a B-read score reflects that reader's first impression of the film, with the second number reflecting a different number if the reader has a "second thought" or if the reader thinks another B-reader could arrive at a different conclusion. For example, a 1/0 score reflects a B-reader's conclusion that the film is mildly abnormal, but that another B-reader could read the film as normal.

26. The 1986 American Thoracic Society criteria required a B-read to be 1/1 or greater before the result was considered consistent with asbestosis. The 1986 criteria also stated: "the benefit of the doubt should be given whenever the clinical features and occupational exposure data are compatible with the diagnosis." The 2004 American Thoracic Society criteria liberalized the standard to define a 1/0 read or greater as consistent with asbestosis, but removed the "benefit of the doubt" language. Many common non-asbestos-related conditions are consistent

with a 1/0 B-read. For example, cigarette smoking can cause opacities consistent with a 1/0 B-read.

27. The [P]laintiffs in these cases took part in a mass screening of chest x-rays of [Defendant's] former . . . factory workers. This mass screening was organized by [P]laintiffs' attorneys. These x-rays were reviewed by B-readers selected by [P]laintiffs' attorneys. Over 80% of [P]laintiffs in these cases were evaluated by [P]laintiffs B-readers to have "1/0" B-reads. Plaintiffs were subsequently referred by [P]laintiffs' attorneys for mass diagnostic examinations at a hotel in Charlotte performed by pulmonologists selected by [P]laintiffs' attorneys.

28. Defendant's B-readers evaluated the [P]laintiffs' x-rays as 0/0. This consistent disparity of B-reads, which, by definition, are meant to be read to a consistent standard, raises the issue of possible B-reading bias by one or both sides.

29. Asbestos-related diseases follow a dose-response relationship—the higher the cumulative [asbestos] exposure dose, the greater the risk of disease, with asbestosis generally requiring the highest dose. Pleural plaques, pleural thickening, and mesothelioma are asbestos-related conditions that generally form at a lower dose.

30. In the general population, approximately 80% of people diagnosed with asbestosis will also have bilateral pleural plaques. However, experts in these cases only identified about 10% of the [P]laintiffs diagnosed with asbestosis as also having bilateral pleural plaques. This outcome is statistically improbable. Because pleural plaques require less exposure, it is not logical that such a large group diagnosed with asbestosis would have so few with pleural plaques.

31. Pursuant to an order issued by former Deputy Commissioner Gheen, [D]efendant has been entitled to autopsies and lung tissue examinations of deceased

[P]laintiffs to allow pathological examinations. Although 18 [P]laintiffs have died to date, [D]efendant has only been able to obtain autopsy results and tissue examinations of five deceased [P]laintiffs—Walter Hinson, Johnnie Jones, Charles Gibson, Homer Hunt, and Lloyd Cox. Walter Hinson is the only [Bellwether P]laintiff who had post-mortem pathology[.]

32. Pathological examination of lung tissue is a definitive method of determining whether an individual has an asbestos-related disease. x-rays are inherently limited in that they can only identify markings that are consistent with a pneumoconiosis such as asbestosis. These markings, as seen on radiological scans, can also be consistent with a number of unrelated conditions and diseases.

33. The accepted scientific method to diagnose asbestosis pathologically requires diffuse interstitial fibrosis AND either two or more asbestos bodies per centimeter squared OR a count of uncoated asbestos fibers that falls within that lab's range for asbestosis (accounting for the background levels found in that lab's reference population/control group). Labs also may have different methodologies to digest and identify fibers, making cross-lab comparisons problematic. Asbestos bodies are fibers that have been coated by the body as a defense mechanism. Diffuse interstitial fibrosis or scarring can be caused by numerous things other than asbestosis. Many non-asbestos-related diseases and conditions can result in a 1/0 B-read.

34. Of the five deceased [P]laintiffs who had post-mortem pathological study of their lung tissue, (Walter Hinson, Johnnie Jones, Charles Gibson, Homer Hunt, and Lloyd Cox), none had pathological evidence of asbestosis. Pathology is the most reliable method to diagnose asbestosis.

35. Pursuant to the Helsinki, OSHA, and NIOSH standards, fibers shorter than 5 micrometers [or microns]

are not counted pathologically for purposes of asbestosis diagnosis or risk assessment. Fibers shorter than 5 micrometers, due to their length, are cleared quickly by the lungs and are not believed to contribute to the disease. Only fibers longer than 5 micrometers become lodged into the lung tissue, as they are too big to navigate through the lymphatic channels to be cleared by a human lung's defense mechanisms.

36. Samples of the pipe insulation at the . . . factory show the presence of two types of asbestos—amosite and chrysotile. Amosite is an amphibole. Chrysotile is a type of [serpentine] asbestos, often shorter than five micrometers, that is particularly susceptible to being broken down quickly in acidic environments, such as a human lung. Due to its length and fragility in the human lung, the clearance half-life of chrysotile asbestos in humans has been estimated to be a few weeks to a few months. Plaintiffs argue that the tissue fiber analyses in these cases under-assessed the number of fibers by not counting the chrysotile fibers because they are quickly cleared from the human lung. Many experts believe that chrysotile asbestos does not cause or contribute to asbestosis or asbestos-related disease due to its short clearance half-life and that fact that persistence of a fiber within the lung is a crucial determinant of its pathogenicity. By contrast, amphibole asbestos fibers are not susceptible to being dissolved by lung tissue and have a clearance half-life in the human body measured in decades. Because the pipe insulation at the . . . factory had both chrysotile and amphibole asbestos, the [P]laintiffs' lung pathology would show occupational exposure, if it existed, in the form of amphibole fibers.

37. [Plaintiff] Hinson . . . worked for 32 years, mainly in the curing department. The curing department had the highest concentration of insulated piping in the factory, with much of it at floor level or in exposed trenches. According to [Plaintiff] Hinson, he was also exposed to significant asbestos dust from using a band saw to cut large asbestos gaskets. If [P]laintiffs' arguments are correct,

[Plaintiff] Hinson would have been exposed to a significant amount of airborne asbestos. [Plaintiff] Hinson was given a 1/0 B-read by Dr. James Johnson[.] Dr. Craig Hart at York pathology performed [P]laintiff Hinson's lung autopsy. Dr. Hart found no evidence of asbestos bodies or fibrosis, but did see evidence of smoking. The tissue was sent to Dr. Oury, who examined the sample and confirmed Dr. Hart's conclusions. Although it was not required for diagnostic purposes due to the lack of fibrosis, a fiber count analysis was done by Dr. Oury upon [D]efendant's request. The fiber count analysis found 5 asbestos bodies per gram, which is a level well below that seen in individuals with asbestosis and in the range of control individuals with no history of [occupational] asbestos exposure.

38. Decedent Johnnie Jones . . . worked for 25 years in the calendar area. If [P]laintiffs' arguments are correct, he would have been subjected to significant airborne asbestos-contaminated talc exposure in his workplace environment. Decedent Johnnie Jones had a 1/0 B read from Dr. Crim. However, when Dr. Roggli performed a pathological examination of Jones' lung tissue, he found no histologic evidence of asbestosis or elevated asbestos content. Based on decedent Jones' employment history at [the factory] and his pathology results, Dr. Roggli testified that there was not sufficient exposure to asbestos at the factory . . . to contribute to or to cause an asbestos-related disease for Mr. Jones or anyone in his position.

39. Decedent Charles Gibson . . . worked at the . . . factory . . . for 31 years—holding jobs in the tire-building and warehouse departments. If [P]laintiffs' arguments are correct, Gibson would have been subjected to significant airborne asbestos exposure in his workplace environment. Decedent Gibson was found to have a 1/0 B-read according to Dr. Crim. Decedent Gibson's lung tissue was collected by York Pathology Associates after his death. Dr. Jenkins with York Pathology performed a gross tissue examination. Dr. Jenkins found no pleural plaques. Dr. Oury also examined the tissue and found no evidence of pulmonary fibrosis, no asbestos bodies, and no fibers. Talc and

vermiculite were found, but the source of these materials was impossible to discern.

40. Decedent Homer Hunt . . . was employed at the . . . factory for 17 years as a mechanic—working in all areas of the factory. Among many other tasks, decedent Hunt replaced forklift brakes. If [P]laintiffs' arguments are correct, decedent Hunt would have been subjected to significant amounts of airborne asbestos-containing brake dust in his workplace environment. Decedent Hunt was a 45-year smoker who died of lung cancer in 2012. His lung tissue was collected by York pathology pursuant to the Autopsy Order. Dr. Richard Johnson and Dr. Oury examined decedent Hunt's lung tissue. No fibrosis was found in areas of the lung not impacted by the unrelated carcinoma tumor. Furthermore, there were no asbestos bodies or fibers found.

41. Decedent Lloyd Cox . . . worked at [the] factory for 31 years in the stock and bead prep area. Decedent Cox died in 2014 of viral pneumonitis complicated by other factors. Although decedent Cox had "end stage asbestosis" written on his death certificate by the Lancaster County coroner, this diagnosis is of dubious reliability in that it apparently has little or no scientific basis. The coroner does not have a college degree and did not consult with the county pathologist before writing that conclusion on the death certificate. Decedent Cox's lung tissue was collected and examined by York Pathology Associates. Surgical pathologist Dr. Sporn performed a "transbronchial biopsy." Dr. Sporn did not find any asbestos bodies and no condition was found on the biopsy that would have been caused by or contributed to by asbestos exposure. Dr. Sporn articulated that viral pneumonia was the likely cause of death. Dr. Hart performed the pathology exam, microscopically and grossly, and found that there was no interstitial fibrosis, no asbestos bodies, no pleural plaques, no asbestos fibers, and no evidence of exposure to asbestos above the general population.

42. Despite [P]laintiffs' theories of exposure, pathology

results from the lung tissue of five long-term employees from a variety of departments and factory locations uniformly show a lack of fibrosis, a lack of asbestos bodies, and a lack of fibers.

. . . .

44. Drs. Ghio, Barrett, Goodman, and Alexander concluded that [P]laintiffs did not have findings consistent with diagnoses of asbestosis. Given the preponderance of the evidence in view of the entire record, their opinions are given greater weight than those of Drs. Crim, Ohar, Schwartz, and Frank [Plaintiffs' experts].

Plaintiffs argue that some of these findings are erroneous, incomplete, or misstate the facts. We will address Plaintiffs' arguments concerning the findings of fact below. Based upon these common findings of fact, the Commission determined that Plaintiffs had not meet their burden of proving they were exposed to levels of hazardous airborne asbestos capable of causing—or significantly contributing to—their alleged asbestos-related diseases. Additional facts will be discussed below.

### III. Relevant Workers' Compensation Law

#### *A. Standard of Review*

The issues before us are controlled by Article 1, Chapter 97 of the General Statutes—the “Workers’ Compensation Act” (the “Act”). “The employee seeking workers’ compensation benefits bears the burden of proving every element of compensability. The degree of proof required of a claimant under the Act is the ‘greater weight’ or ‘preponderance’ of the evidence.” *Hardin v. Motor Panels, Inc.*, 136

N.C. App. 351, 354, 524 S.E.2d 368, 371 (2000) (citations omitted). This Court's standard of review is well established:

“Appellate review of an award from the Industrial Commission is generally limited to two issues: (i) whether the findings of fact are supported by competent evidence, and (ii) whether the conclusions of law are justified by the findings of fact.” Unchallenged findings of fact are presumed to be supported by competent evidence[.] The Commission's conclusions of law are reviewed *de novo*.

*Penegar v. United Parcel Serv.*, \_\_ N.C. App. \_\_, \_\_, 815 S.E.2d 391, 394 (2018) (citations omitted). “Whether an injury arose out of and in the course of employment is a mixed question of law and fact, and the Industrial Commission's findings in this regard are conclusive on appeal if supported by competent evidence.” *Culpepper v. Fairfield Sapphire Valley*, 93 N.C. App. 242, 247, 377 S.E.2d 777, 780 (1989) (citation omitted).<sup>10</sup> The Commission's findings, including its ultimate findings, are binding “when they are supported by direct evidence or by reasonable inferences drawn from the record.” *Kennedy v. Duke Univ. Med. Center*, 101 N.C. App. 24, 30, 398 S.E.2d 677, 680 (1990) (citations omitted). “[T]he Commission is required to evaluate the credibility of the evidence and reject any evidence it finds as not convincing.” *Phillips v. U.S. Air, Inc.*, 120 N.C. App. 538, 542, 463 S.E.2d 259, 262 (1995) (citation omitted).

[T]he Commission has sole authority to make findings of fact. This Court does not weigh the evidence. We determine only whether there is *any* evidence of substance in the record to support the Commission's findings; if there

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<sup>10</sup> We refer to the Commission's resolution of these mixed questions of law and fact as “ultimate findings.”



is, we are bound by the findings, even though the record may contain evidence supporting findings contra. There must be a complete lack of competent supporting evidence to justify disregarding the Commission's findings of fact. Where medical testimony is conflicting, the Commission decides which testimony to give the greater weight.

*Carroll v. Burlington Industries*, 81 N.C. App. 384, 387-88, 344 S.E.2d 287, 289–90 (1986) (citations omitted).

*B. Workers' Compensation; Occupational Diseases*

Most, if not all, Consolidated Plaintiffs allege they developed asbestosis as a result of their work at the factory.<sup>11</sup> See N.C.G.S. § 97-53(24) (2017). Two Bellwether Plaintiffs, Wilson and Epps, alleged they have occupational diseases as defined by N.C.G.S. § 97-53(13); colon cancer and tonsil cancer, respectively—caused by asbestos exposure at the factory. Normally, the Commission would first determine whether a plaintiff had proven an occupational disease, and only after determining that the plaintiff had met that burden would the Commission consider evidence related to compensability, or whether the occupational disease had any causal connection to the plaintiff's employment. However, for these cases we are asked to review the Commission's determinations that conditions at the factory could not have exposed Consolidated Plaintiffs to airborne asbestos of a type and in sufficient amounts to cause asbestosis, or other asbestos-related diseases—*before* the Commission

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<sup>11</sup> Or, for deceased Plaintiffs, their estates allege that the deceased Plaintiffs had developed asbestosis.

determines whether any Consolidated Plaintiffs actually have asbestos-related diseases.<sup>12</sup> In light of the unusual procedure employed, a review of workers' compensation law and procedure applicable to cases of alleged compensable asbestos-related diseases is appropriate.

“The underlying purpose of [the 1929 adoption of the] Act . . . [wa]s to provide compensation for workmen who suffer disability by accident arising out of and in the course of their employment.” *Henry v. Leather Co.*, 234 N.C. 126, 127, 66 S.E.2d 693, 694 (1951). Initially, the Act only allowed compensation for “injury by accident.” *See id.* at 127, 66 S.E.2d at 694. However, the Act was amended in 1935 to include benefits for employees who developed compensable occupational diseases. *Id.* at 128, 66 S.E.2d at 694–95; N.C.G.S. § 97-52 (2017). The amendment enumerated specific diseases—like asbestosis—that were designated as “occupational diseases within the meaning of [Article 1].” *Henry*, 234 N.C. at 128, 66 S.E.2d at 694 (citation omitted); N.C.G.S. § 97-53. Later, the Act was amended to allow employees to prove that a disease *not* specifically enumerated in N.C.G.S. § 97-53 was a “compensable occupational disease” based upon the specific facts of the plaintiff’s claim. N.C.G.S. § 97-53(13). N.C.G.S. § 97-53(13) states in relevant part: “Any disease . . . which is proven to be due to causes and conditions which are characteristic of and peculiar to a particular trade, occupation or employment, but excluding all ordinary diseases of

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<sup>12</sup> Excluding the Bellwether Plaintiffs, for whom the Commission has concluded no asbestos-related diseases have been proven to exist.

life to which the general public is equally exposed outside of the employment” “shall be deemed to be [an] occupational disease[] within the meaning of” the Act. N.C.G.S. § 97-53(13).

“[T]he addition of G.S. 97-53 to the Act ‘in nowise relaxed the fundamental principle which requires proof of causal relation between injury and employment.’” *Booker v. Medical Center*, 297 N.C. 458, 475, 256 S.E.2d 189, 200 (1979) (citations omitted). “It is overwhelmingly apparent that . . . disablement resulting from an occupational disease . . . must arise out of and in the course of the employment, *i.e.*, there must be some causal relation between the injury and the employment[.]” *Morrison v. Burlington Industries*, 304 N.C. 1, 12, 282 S.E.2d 458, 466 (1981).

Now, all provisions of the Act that had formerly applied only to injuries by accident also apply to compensable occupational diseases—so long as they do not conflict with more specific provisions in the Act specifically pertaining to occupational diseases. N.C.G.S. § 97-52. “[A]n employee becoming disabled by asbestosis [or other occupational disease] . . . within the terms of the specific definition embodied in G.S. [§] 97-54 should be entitled to ordinary compensation measured by the general provisions of the . . . Act. G.S. [§] 97-64.” *Young v. Whitehall Co.*, 229 N.C. 360, 366, 49 S.E.2d 797, 801 (1948).

Therefore, the Act now provides that the terms “injury,” “personal injury,” or “injury by accident” also encompass “[d]isablement or death of an employee resulting from an occupational disease described in G.S. 97-53[.]” N.C.G.S. § 97-52; N.C.G.S. §

97-2(6) (2017) (emphasis added) (“Injury.—‘Injury and personal injury’ shall mean only injury by accident arising out of and in the course of the employment[.]”); *see also Henry*, 234 N.C. at 128, 66 S.E.2d at 694 (citation omitted) (The amendment also “broadened or extended the meaning of the word ‘accident’ as used in the original Act so as to include a disablement or death resulting from an occupational disease described in G.S. § 97-53[.]”). “Nothing is said in [N.C.G.S. § 97-52] or cases construing it which could be interpreted as allowing compensation for injury from occupational disease which falls short of ‘disablement.’” *Harrell v. Harriet and Henderson Yarns*, 56 N.C. App. 697, 699, 289 S.E.2d 846, 847 (1982); N.C.G.S. § 97-64 (2017).

Generally, “disablement” means a diminished ability to earn wages resulting from an injury sustained due to employment. N.C.G.S. § 97-2(9); N.C.G.S. § 97-54 (2017). “The term ‘disability’ as used in [the Act] means the state of being incapacitated as the term is used in defining ‘disablement’ in G.S. 97-54[.]” N.C.G.S. § 97-55 (2017), and is therefore, in all ways relevant to this opinion, synonymous with “disablement.” “The term ‘death’ as a basis for a right to compensation means only death resulting from an injury.” N.C.G.S. § 97-2(10).

In order to be compensable, a plaintiff-employee must prove, *inter alia*, that the plaintiff’s alleged occupational disease, including one—like asbestosis—that is specifically enumerated in N.C.G.S. § 97-53, “was incident to or the result of the *particular* employment in which the workman was engaged.” *Booker*, 297 N.C. at

475, 256 S.E.2d at 200 (citation omitted). Stated differently, “to demonstrate a causal link between the condition for which plaintiff seeks compensation and plaintiff’s employment[,]” the plaintiff must prove that the plaintiff’s “employment ‘significantly contributed to, or was a significant causal factor in, the disease’s development.’” *James v. Perdue Farms, Inc.*, 160 N.C. App. 560, 562, 586 S.E.2d 557, 560 (2003) (citations omitted). As noted in finding of fact 2, Consolidated Plaintiffs do not argue that employment at the factory “significantly contributed to” the development of their alleged asbestosis diagnoses. Instead, “Plaintiffs allege that they were [each] exposed to asbestos [while working at the factory] in such form and quantity and with such frequency that it *caused* asbestosis.” (Emphasis added). Therefore, with respect to asbestosis, our review will be limited to whether Plaintiffs proved work at the factory “was a significant causal factor in” development of Plaintiffs’ alleged asbestosis. *Id.*

Pursuant to N.C.G.S. § 97-53(13), colon cancer or tonsil cancer

may be an occupational disease provided the occupation in question exposed the worker to a greater risk of contracting this disease than members of the public generally, and provided the worker’s exposure to [asbestos] significantly contributed to, or was a significant causal factor in, the disease’s development. This is so even if other non-work-related factors also make significant contributions, or were significant causal factors.

*Rutledge v. Tultex Corp.*, 308 N.C. 85, 101, 301 S.E.2d 359, 369–70 (1983) (citation omitted). When determining whether an occupational disease is compensable, “[t]he factual inquiry . . . should be whether the occupational exposure was such a

significant factor in the disease's development that without it the disease would not have developed to such an extent that it caused the physical disability which resulted in claimant's incapacity for work." *Id.* at 102, 301 S.E.2d at 370. "[I]f a disease is produced by some extrinsic or independent agency, it may not be imputed to the occupation or the employment." *Id.* at 103, 301 S.E.2d at 370 (citations omitted).

Therefore, generally, in order for a claim of occupational disease to be compensable under the Act, the plaintiff must prove (1) that the plaintiff has an injury—specifically an occupational disease; (2) that the occupational disease “arose out of” and “in the course of” some employment, *Gallimore v. Marilyn's Shoes*, 292 N.C. 399, 402–03, 233 S.E.2d 529, 531–32 (1977),—*i.e.* that the “employment ‘significantly contributed to, or was a significant causal factor in, the disease’s development[,]’” *James*, 160 N.C. App. at 562, 586 S.E.2d at 560 (citations omitted); and (3) that the occupational disease resulted in “disability,” N.C.G.S. § 97-54.<sup>13</sup> “In general, the term ‘in the course of’ refers to the time, place and circumstances under

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<sup>13</sup> Generally, Plaintiff Hinson’s estate would not need to prove that his employment with Defendant was the “origin or cause” of his disablement, *Penegar*, \_\_ N.C. App. at \_\_, 815 S.E.2d at 398—it could prove a causal connection between his alleged asbestosis and any employment prior to or including his work at the factory. Defendant would then be liable for any disability due to Plaintiff Hinson’s asbestosis if his estate could also prove that he “was last injuriously exposed to the hazards of” asbestosis, as defined in N.C.G.S. § 97-57, while working for Defendant. N.C.G.S. § 97-57 (2017). However, the Commission found as fact that Plaintiffs all “allege that they contracted asbestosis caused by exposure to airborne asbestos . . . during employment with [D]efendant[,]” and that Plaintiff “Hinson apparently had no exposure to asbestos through his prior employment.” Plaintiff Hinson’s Estate does not contest these findings, so it must prove that Plaintiff Hinson developed asbestosis due to exposure to asbestos while working at the factory—and that his asbestosis led to disablement as defined by the Act at some point prior to his death. N.C.G.S. § 97-52; N.C.G.S. § 97-2(6); N.C.G.S. § 97-54.

which an accident occurs, while the term ‘arising out of’ refers to the origin or causal connection of the accidental injury to the employment.” *Gallimore*, 292 N.C. at 402–03, 233 S.E.2d at 531–32 (citations omitted). “In determining whether a claimant’s [alleged occupational] exposure to [asbestos] has significantly contributed to, or been a significant causative factor in, [an asbestos-related] disease, the Commission may, of course, consider medical testimony, but its consideration is not limited to such testimony.” *Rutledge*, 308 N.C. at 105, 301 S.E.2d at 372 (citation omitted). Our Supreme Court has stated:

In the case of occupational diseases proof of a causal connection between the disease and the employee’s occupation *must of necessity be based on circumstantial evidence*. Among the [non-exclusive] circumstances which may be considered are the following: (1) the extent of exposure to the disease or disease-causing agents during employment, (2) the extent of exposure outside employment, and (3) absence of the disease prior to the work-related exposure as shown by the employee’s medical history.

*Booker*, 297 N.C. at 476, 256 S.E.2d at 200 (citations omitted) (emphasis added).

Only after a plaintiff has proven that the plaintiff’s occupational disease is compensable, must the plaintiff prove a defendant-employer’s liability—by proving the plaintiff was “last injuriously exposed” to the hazards of the disease while working for that defendant-employer. N.C.G.S. § 97-57. N.C.G.S. § 97-57 states in part:

In any case where compensation is payable for an occupational disease,<sup>[14]</sup> the employer in whose

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<sup>14</sup> *I.e.*, once the plaintiff has proven a compensable occupational disease.

employment the employee was last injuriously exposed to the hazards of such disease . . . shall be liable.

*For the purpose of this section* when an employee has been exposed to the hazards of asbestosis . . . for as much as 30 working days, or parts thereof, within seven consecutive calendar months, such exposure shall be deemed injurious but any less exposure shall not be deemed injurious[.]

N.C.G.S. § 97-57 (emphasis added). If a plaintiff fails to prove that the plaintiff has a compensable occupational disease, compensation will be denied and “last injurious exposure” analysis pursuant to N.C.G.S. § 97-57 will not be necessary. N.C.G.S. § 97-57 is not meant to establish the burden for proving a causal relationship between a particular employment and *development of* an occupational disease. Instead:

[T]he purpose of the “last injurious exposure” doctrine is “to eliminate the need for complex and expensive litigation of the issue of relative contribution by each of several employments to a plaintiff’s occupational disease.” The doctrine provides a plaintiff with a reduced burden by requiring only a showing that the occupational exposure augmented a disease, “however slight[.]” as opposed to demonstrating how much each exposure resulted in the disease.

*Penegar*, \_\_ N.C. App. at \_\_, 815 S.E.2d at 398 (citations omitted). In the present cases, Plaintiffs alleged their sole occupational exposure to asbestos occurred working in the factory.

### *C. The “Bellwether Cases” Approach*

As noted above, in the ordinary case—because it is the plaintiffs’ burden to prove they “suffer[ed] from [] compensable occupational disease[s,]” *Hardin*, 136 N.C.



App. at 354, 524 S.E.2d at 371 (citations omitted)—the Commission would *first* determine whether the plaintiffs had met their burden of proving they suffered from an occupational disease. If the plaintiffs failed to meet that burden, the Commission could deny their claims without making any further determinations such as compensability and liability. *See, e.g., Payne v. Charlotte Heating & Air Conditioning*, 172 N.C. App. 496, 616 S.E.2d 356 (2005); *Clark v. ITT Grinnell Ind. Piping, Inc.*, 141 N.C. App. 417, 539 S.E.2d 369 (2000).

However, because of the bellwether cases approach, the Commission addressed the issues common to all Consolidated Plaintiffs first—and only then made individual determinations specific to the individual Bellwether Plaintiffs. The Commission's determinations concerning whether any individual Consolidated Plaintiff had asbestosis will necessarily require review of the medical evidence specifically relevant to that particular Plaintiff—*i.e.*, thorough review of all relevant documentary and testimonial evidence for every one of the 144 Consolidated Plaintiffs. Pursuant to the bellwether cases approach, review of the medical evidence for the alleged asbestos-related diseases for all Consolidated Plaintiffs will only be necessary *if* Plaintiffs first prove that working in the factory exposed them to asbestos, in a form and in quantities, that could have caused the alleged asbestosis; or caused—or significantly contributed to—the development of *other* alleged asbestos-related diseases.

The Commission determined employment in the factory did not expose Plaintiffs to airborne asbestos of a kind and in amounts sufficient to cause or

contribute to asbestosis.<sup>15</sup> If this determination is affirmed, most, if not all, of the Consolidated Plaintiffs' asbestosis claims can be decided without the time and cost involved in conducting full hearings for all 144 cases. *Booker*, 297 N.C. at 472, 256 S.E.2d at 198. In light of the inverted approach applied in the bellwether cases, the Commission essentially assumed, *arguendo*, that the Consolidated Plaintiffs actually had asbestosis that resulted in disablement or death—and focused solely on whether Plaintiffs proved work at the factory was a significant causal factor in development of the alleged asbestosis. The Commission has not, of course, made this determination<sup>16</sup>—but will do so if required by this Court's resolution of the bellwether cases and factual circumstances particular to the remaining Consolidated Plaintiffs. The Commission also determined that, with respect to employment at the factory, neither colon cancer nor tonsil cancer were occupational diseases pursuant to N.C.G.S. § 97-53(13).

#### *D. The Bellwether Plaintiffs' Claims*

Plaintiffs and Defendant presented weeks of expert testimony concerning the common issue of whether Plaintiffs could have been subjected to sufficient airborne asbestos—chrysotile or amphibole—while working at the factory to cause compensable asbestosis. *James*, 160 N.C. App. at 562, 586 S.E.2d at 560. Evidence

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<sup>15</sup> *I.e.*, that any alleged asbestos-related disease could not have “arisen out of” employment with Defendant. *Gallimore*, 292 N.C. at 402–03, 233 S.E.2d at 531–32; N.C.G.S. § 97-54.

<sup>16</sup> Except for the five Bellwether Plaintiffs currently before us. Because these five cases were actually tried, the Commission considered the evidence relevant to the “common issues,” as well as the evidence uniquely relevant to each individual Bellwether Plaintiff.

was also presented for Bellwether Plaintiffs' individual claims, including evidence related to whether Bellwether Plaintiffs had the diseases alleged. Based upon this testimony, deposition testimony, and the exhibits tendered, the Commission determined that Plaintiffs failed to meet their burdens on all accounts.

#### IV. Plaintiffs' Common Issues Arguments

Plaintiffs did not brief the common issues separately from the individual issues, so we look to Bellwether Plaintiffs' individual briefs for the common issues arguments. Because our analysis pertaining to the common issues will apply to the claims of all Consolidated Plaintiffs—not just Plaintiff Hinson or the other Bellwether Plaintiffs—where Plaintiff Hinson's brief refers to "Plaintiff," we will substitute "Plaintiffs" or "Consolidated Plaintiffs," which will refer to *all* Consolidated Plaintiffs.

Generally, Plaintiffs argue: (A) The Commission did not apply the appropriate burden of proof in reaching its determinations concerning Plaintiffs' exposure to asbestos as employees in the factory; (B) the Commission relied on incompetent evidence in reaching its conclusions; (C) certain of the findings of fact were not supported by sufficient competent evidence; and (D) the ultimate findings/conclusions of law are incorrect.

##### *A. Burden of Proof*

Plaintiffs first argue that the Commission "placed an impermissible burden of establishing the amount of exposure to asbestos" on Plaintiffs. We disagree.

As a general matter: “The employee seeking workers’ compensation benefits bears the burden of proving every element of compensability. The degree of proof required of a claimant under the Act is the ‘greater weight’ or ‘preponderance’ of the evidence.” *Hardin*, 136 N.C. App. at 354, 524 S.E.2d at 371 (citations omitted).

Plaintiffs specifically argue:

One of the critical issues in the [consolidated cases] was whether Plaintiff[s] [were] exposed to asbestos and whether such exposure was medically capable of causing a disease. . . .

[The] Commission made findings and conclusions regarding the amount of exposure [] Plaintiff[s] had to asbestos and whether that level was sufficient to cause a disease.

The Commission specifically, and repeatedly, [determined] that [Plaintiffs] “[were] not exposed to asbestos in such form and quantity, and used with such frequency, as to cause asbestosis or any asbestos-related condition.” What the . . . Commission . . . did was place the burden on [] [Plaintiffs] to establish the level of exposure to [asbestos]. Under North Carolina law, that is impermissible.

We first note that Plaintiffs’ counsel acknowledged during the hearings that Plaintiffs’ burden was to prove “what [Plaintiffs’] actual exposures were” to asbestos “[a]t the plant.” In addition, Plaintiffs have not challenged finding of fact 2, which states in part: “Plaintiffs allege that they were exposed to asbestos [while working in the factory] in such form and quantity and with such frequency that it caused asbestosis.” As we must take this unchallenged finding as correct, Plaintiffs now challenge the application of a standard they approved while arguing before the

Commission. Nonetheless, Plaintiffs contend that the Commission erroneously applied the following burden of proof contained in N.C.G.S. § 97-53—and that by so doing, the Commission imposed upon Plaintiffs the impermissible burden of “establish[ing] the[ir] level[s] of exposure” to asbestos. N.C.G.S. § 97-53 contains different requirements depending on the type of injury alleged, including the following:

Occupational diseases caused by chemicals shall be deemed to be due to exposure of an employee to the chemicals herein mentioned only when as a part of the employment such employee is exposed to such chemicals in such form and quantity, and used with such frequency as to cause the occupational disease mentioned in connection with such chemicals.

N.C.G.S. § 97-53. Defendant’s counsel referred to this section of N.C.G.S. § 97-53 in the opening statement to the deputy commissioner, and Plaintiffs did not object. On appeal, Plaintiffs do not specifically challenge the applicability of this part of N.C.G.S. § 97-53 to cases involving exposure to asbestos—but do state that “this language speaks of ‘chemicals’ and not necessarily asbestos. There was no testimony or evidence that asbestos would be considered a ‘chemical’ under the statute.” Plaintiffs further contend: “Regardless, this statute, as applied by the Commission, would be in

direct conflict with . . . case law whereby [employees were] not required to establish the amount of exposure.”<sup>17</sup>

We note that this part of N.C.G.S. § 97-53, by its plain language, only applies to “[o]ccupational diseases caused by chemicals” “herein mentioned[,]” and only to “occupational disease[s] mentioned in connection with such chemicals.” N.C.G.S. § 97-53. Even assuming that asbestos would be considered a “chemical” for the purposes of this section, *asbestos* is not “mentioned” in N.C.G.S. § 97-53, and asbestosis is not “mentioned in connection with” asbestos, or any other “chemical.” *Id.*; *cf.*, *e.g.* N.C.G.S. § 97-53(24) and (12) (compare “[a]sbestosis” to “[p]oisoning by benzol, or by nitro and amido derivatives of benzol”).

However, the Commission did include language that tracks the language of this part of N.C.G.S. § 97-53 in five of its ultimate findings. For example, finding 45 states in part: “The greater weight of the evidence in view of the entire record does not show that [P]laintiffs, through their employment at [the] factory, were exposed to asbestos *in such form and quantity and [] with such frequency as to cause or significantly contribute to the development of* asbestosis[.]” (Emphasis added). The italicized portion of this ultimate finding tracks the language of N.C.G.S. § 97-53.

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<sup>17</sup> While this Court is generally bound by its prior decisions interpreting a statute, *In re Civil Penalty*, 324 N.C. 373, 384, 379 S.E.2d 30, 37 (1989), no such interpretation of this part of N.C.G.S. § 97-53 has occurred. Since there are no appellate opinions interpreting N.C.G.S. § 97-53 in the manner suggested by Plaintiffs, this Court is bound by the language of the statute itself, not the principles of law discussed in the two cases cited by Plaintiffs—one of which is unpublished—that do not address this provision.

However, though the underlined portion is not found in N.C.G.S. § 97-53, it does correspond with the correct burden for proving a causal connection between a particular employment and alleged asbestosis. “Asbestosis may be [a compensable] occupational disease provided that the worker’s exposure to . . . [asbestos] ‘*significantly contributed to, or was a significant causal factor in,*’ the development of the disease.” *Patton v. Sears Roebuck & Co.*, 239 N.C. App. 370, 375, 768 S.E.2d 351, 355 (2015) (citations omitted) (emphasis added).

As discussed below, we find that the burden applied by the Commission was a correct application of the law to the facts of the consolidated cases. The testimony of both Plaintiffs’ and Defendant’s experts included opinions that support the Commission’s focus on “exposure to airborne asbestos,” “in such form,” in sufficient “quantity,” and “with such frequency”—*i.e.* recurring exposures over time, or duration of exposure—in making its determination of whether Plaintiffs had met their burden of proving a causal connection between their alleged asbestosis and their work at the factory. These categories conform with factors enumerated by the NCI concerning elevated risk for asbestos-related diseases: “Dose (how much asbestos an individual was exposed to)[; d]uration (how long an individual was exposed)[; and s]ize, shape, and chemical makeup of the asbestos fibers.” NCI *Fact Sheet* at 2-3 (citations omitted). They also conform to one of the non-exclusive circumstantial factors appropriate for consideration in determining whether a plaintiff has met the burden of proving a causal relationship: “[T]he extent of exposure to the disease or disease-

causing agents during employment[.]” *Booker*, 297 N.C. at 476, 256 S.E.2d at 200 (citations omitted). Therefore, consideration of these factors was appropriate in determining whether asbestos exposure at the factory “was a significant causal factor in,’ the development of” Plaintiffs’ alleged asbestosis. *Id.* (citations omitted).

### 1. “Form” of Asbestos

As recognized by the NCI, the “size, shape, and chemical makeup of the asbestos fibers” are relevant in determining the likelihood exposure will result in disease. NCI *Fact Sheet* at 2-3 (citations omitted). Plaintiffs’ “expert in electron microscopy and lung tissue analysis,” Mark Wilson Rigler, PhD (“Rigler”),<sup>18</sup> agreed with the NCI *Fact Sheet* that “asbestiform minerals come in a couple of different broad classes”—“serpentine,” which “is mainly comprised of chrysotile” asbestos and is “like a tube[.]” and “amphibole,” which is “mainly composed of blocky structural forms.” Rigler clarified that only when these minerals are in fiber form are they capable of causing disease. Rigler testified that “probably ninety-five percent of the products [] manufactured, at least in America, had chrysotile asbestos[.]” “and the other five percent would’ve probably had amosite [an amphibole form], and that might’ve been pipe coverings, that kind of thing.”

Rigler further testified that the human body handles chrysotile asbestos differently than amphibole asbestos:

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<sup>18</sup> Because Plaintiffs argue on appeal that only medical experts are qualified to give certain causation opinion testimony, in order to avoid confusion concerning which experts were medical doctors and which were PhDs, we will only use the honorific “Dr.” when referring to medical doctors.



Chrysotile asbestos, which is the tubular type that we talked [about] earlier, . . . is not retained as long as the amphibole fibers are, so if you get chrysotile in the lung, it tends to move that out a bit quicker. Some of the [chrysotile] fibers are smaller. They are taken up a little bit easier into macrophages. . . . So . . . then they try to move out, if they can, through your lymphatic circulation. Some are removed out through the blood stream, but they do . . . migrate in the body. Now, the amphibole types of asbestos, they tend not to migrate like that. They tend to stay in the body for 45 [years], so you can, you know, after ten, twenty, thirty years, you can see amphibole asbestos in the body.

Defendant's expert, Dr. Victor Roggli ("Dr. Roggli"), testified that chrysotile asbestos fibers cleared from the body much more rapidly than amphibole fibers. Plaintiffs' expert industrial hygienist, William M. Ewing ("Ewing"), agreed that amosite asbestos is "recognized as being more potent when it comes to cancer and exposure" than chrysotile asbestos, but testified that he did not know if "there is a general understanding among [other] industrial hygienists . . . that chrysotile is less potent with respect to asbestosis as well[.]" Dr. Roggli testified that exposure to commercial amphibole fibers can cause disease at a lower dosage than other asbestos fibers, such as chrysotile. He stated the difference was very significant for lung cancer, and that "it's believed" by many experts that someone would require a greater exposure to "chrysotile to get to asbestosis than for commercial amphiboles." Dr. Roggli testified that chrysotile asbestos fibers do not "form asbestos bodies as well" as amphibole asbestos fibers, and that "it really takes huge amounts of exposure to chrysotile to get asbestosis." Dr. Thomas Sporn ("Dr. Sporn") testified: "In general my opinions have

been that exposure [to] chrysotile containing end products [commercial products such as insulation, gaskets, or brakes] do not particularly cause . . . asbestosis[.]”<sup>19</sup>

According to Dr. Roggli, asbestos fibers less than five microns in length “would not be disease-producing[.]” and that approximately ninety percent of the scientific community was of the same opinion. In prior testimony, Rigler also defined “larger structures” indicative of occupational exposure as “greater than five microns.” Dr. Roggli testified that chrysotile fibers of over five microns are rarely found in lungs—estimating that only about ten percent of chrysotile lung exposures include fibers longer than five microns. Rigler testified that “asbestos bodies” are created when tissue forms around an asbestos fiber, and they can be indicators of asbestosis. Defendant’s expert, Dr. Timothy David Oury (“Dr. Oury”), testified: “without asbestos bodies, [you cannot] make the pathological diagnosis for asbestosis[.]” Because of its generally smaller size, and the rapidity with which the human body evacuates it, Dr. Roggli testified that “of the asbestos types,” chrysotile is “the least effective at forming asbestos bodies.”

Dr. Roggli’s expert medical opinion was that “short fibers of chrysotile<sup>[20]</sup> are] more consistent with a background environmental exposure than a long fiber would be[.]” Dr. Roggli testified that short chrysotile fibers are commonly found in water supplies and products such as beer, wine, soft-drinks, and ketchup. Rigler testified

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<sup>19</sup> Assuming the product contained only chrysotile asbestos.

<sup>20</sup> Less than five microns.

that multiple governmental and science agencies required that a suspected asbestos fiber had to be at least five microns in length “in order to be counted as a fiber” for purposes of determining asbestos exposure—but stated that he disagreed with this requirement.<sup>21</sup> Rigler testified that a relatively small amount of short chrysotile fibers found in lung tissue would suggest a very significant prior exposure to chrysotile. Rigler saw no contradiction between his testimony in the present cases and testimony he had given in prior cases that “[t]ypically [a]n occupational exposure will be indicated by longer fibril structures.” “As far as the length of structures, you will not normally see [fibers longer than five microns] in non-occupational exposure. You may see some much, much smaller structures. That’s typically what you see in environmental type exposure.”

Dr. Roggli testified in response to the idea that finding short chrysotile asbestos fibers in lung tissue was indicative of a substantial prior occupational exposure to chrysotile asbestos with the following: “Well, if that’s the case, it means everybody in the general population has had a huge exposure to chrysotile in the past because that’s exactly what you find in lung tissue from the people from the general population—is a number of short chrysotile fibers.”

The Commission found as fact: “Chrysotile is a type of asbestos, often shorter than five micro[ns], that is particularly susceptible to being broken down quickly in

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<sup>21</sup> There are also minimum width requirements, and the structure must also “have an aspect ratio of three . . . to one.”

acidic environments, such as a human lung.” “Fibers shorter than 5 micro[ns], due to their length, are cleared quickly by the lungs and are not believed to contribute to [asbestosis].” “Many experts believe that chrysotile asbestos does not cause or contribute to asbestosis or asbestos-related disease due to its short clearance half-life and the fact that persistence of a fiber within the lung is a crucial determinant of its pathogenicity.” There was plenary evidence from which the Commission could determine that the “form” of the asbestos that Plaintiffs alleged they were exposed to at the factory was a relevant factor in determining whether Plaintiffs’ alleged asbestos exposure at the factory could have caused asbestosis.

## 2. Quantity

The quantity, or amount of asbestos exposure, was central to the Commission’s determination. The NCI refers to this as the “dose,” “how much asbestos an individual was exposed to[,]” and considers it an important factor to consider. NCI *Fact Sheet* at 3 (citations omitted). There are two general ways in which the amount of exposure impacts the Commission’s causation analysis: (1) Was the exposure sufficient to be a “significant causal factor” in the development of Plaintiffs’ alleged asbestosis; and (2) was the exposure “significantly greater” than the background environmental exposure.<sup>22</sup> “[T]he ‘causative danger must be *peculiar to the work and*

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<sup>22</sup> “*Significant* means ‘having or likely to have influence or effect: deserving to be considered: important, weighty, notable.’ *Significant* is to be contrasted with *negligible*, *unimportant*, *present but not worthy of note*, *miniscule*, or *of little moment*.” *Rutledge*, 308 N.C. at 101–02, 301 S.E.2d at 370 (citation omitted). Proving what constituted exposure “significantly greater” than environmental

*not common to the neighborhood.” Culpepper*, 93 N.C. App. at 248, 377 S.E.2d at 781 (citation omitted). If the answer to either of these questions was “no,” then any alleged asbestos-related diseases could not be causally linked to work at the factory.

Rigler testified that at his laboratory, in order to estimate the amount of exposure, they conduct a fiber analysis using “grid counting”; “we’ll count the number of asbestos [fibers].” According to Rigler, grid counting is “standard protocol.” Rigler testified that he would first use an electron microscope to count “what we call large fiber structures, ones that are larger than five microns or so.” Then Rigler increases magnification to the extent that he can count asbestos fibers “a half micron in size and up.” An estimated number of fibers per gram of lung tissue is extrapolated from the number of fibers actually detected in a smaller amount of tissue.

Although Rigler testified that he believed “background” exposure levels should be zero, he testified in 2000 that, based on his own research and the relevant literature, the environmental background range he had seen had “been upwards of two hundred and fifty thousand [asbestos fibers] [per gram of lung tissue]. Sometimes, again, it depends on the literature that you look at—half a million structures.” Rigler admitted that he used to compare the number of structures per gram against a cohort, or control group, developed from examining lung tissue of people with no reported occupational asbestos exposure. The range of structures per

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exposure for these cases was Plaintiffs’ burden, and a determination that could only be made by the Commission—absent consensus between the parties.

gram determined from the cohort constituted the range of non-occupational background environmental asbestos exposure of the general population. Rigler stopped comparing tissue samples examined in his lab against a cohort before his examination of Plaintiff Jones'<sup>23</sup> lung tissue sample, which Rigler opined showed asbestosis. Dr. Roggli testified that the number of structures counted is meaningless without a proper cohort to compare that number to.

The deputy commissioner questioned the basis of Rigler's opinion that any amount of asbestos fibers found in lung tissue would be indicative of occupational exposure, and Rigler's opinion that the amount of asbestos found in Plaintiff Jones' tissue indicated asbestosis:

THE COURT: The [Plaintiffs] I'm looking at are from Charlotte, . . . which is a major metropolitan area[.] So I would assume that you would expect that some people within the Charlotte area, who've never had an occupational history, would have some asbestos in their lungs.

[RIGLER]: I don't know.

THE COURT: Don't know. Wow.

Rigler then testified: "I think that you're going to see a lot of variation [in background level] depending upon where these people lived. It's always going to be dependent upon what they did and where they lived." Plaintiffs' expert, Dr. David A. Schwartz,

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<sup>23</sup> One of the deceased Consolidated Plaintiffs whose lung tissue was examined.

testified “I don’t think people in the general public are at risk of developing asbestosis based on their exposures to environmental concentrations of asbestos.”

Dr. Roggli testified: “The analysis that we did did not demonstrate that [Plaintiff Jones] was exposed to asbestos greater than that of [the] general population.” Dr. Roggli explained that the types of employment that could expose a worker to the levels of chrysotile asbestos required to cause asbestosis were those jobs where employees were working directly with the asbestos, such as “insulators,” “shipyard workers,” and specialized work within other industries, but he could not remember ever diagnosing a tire factory employee with asbestosis.

Therefore, there was evidence presented that most of the asbestos used in the factory was chrysotile, and that Plaintiffs would have had to have been exposed to “huge amounts” of it to develop asbestosis. Based on the evidence, the Commission needed to determine whether working at the factory exposed Plaintiffs to “quantities” of asbestos fibers sufficient to cause asbestosis, and whether working at the factory exposed Plaintiffs to quantities of asbestos “significantly” greater than the background levels to which the general public were exposed.

### 3. Frequency

Plaintiffs’ experts and Defendant’s experts disagreed concerning the likelihood that episodic exposures to elevated levels of asbestos were likely to cause asbestosis. The Commission found: “Asbestos-related diseases follow a dose-response relationship—the higher the cumulative exposure dose, the greater the risk of

disease, with asbestosis generally requiring the highest dose.” The NCI *Fact Sheet* included “duration”—“how long an individual was exposed” to airborne asbestos fibers—as one of the factors to consider when evaluating the risks of developing asbestos-related diseases. NCI *Fact Sheet* at 3 (citations omitted). It is the position of the NCI that though “it is clear that the health risks from asbestos exposure increase with heavier exposure and longer exposure times, investigators have found asbestos-related diseases in individuals with only brief exposures.” *Id.* It was the province of the Commission to determine from the record evidence if Plaintiffs had met their burden of proving sufficient frequency of exposure—whether by proving large, intermittent exposures, or lesser but more continuous exposures.

Plaintiffs’ expert, Ewing, testified concerning the relationship between “quantity,” “frequency,” and duration of asbestos exposure: “You would like to have exposure information [quantity], duration information, how long is that exposure going on, and then frequency information, so you’d like to have those three pieces of data. If you have that, then you can do some calculations that can give you a person’s dose.” Ewing agreed that for the Bellwether Plaintiffs, “at most, their exposures were episodic[.]” Defendant’s expert, Dr. Andrew J. Ghio (“Dr. Ghio”), testified that, in his expert medical opinion, he did not believe “an individual working at [the factory] [wa]s at increased risk for asbestosis.” The Commission noted: “Dr. Roggli testified that there was not sufficient exposure to asbestos at the factory in question to contribute to or to cause an asbestos-related disease for Mr. Jones or anyone in his



position.” Drs. Ghio and Roggli were both of the opinion that Plaintiffs would have only endured minor, infrequent episodic exposures to airborne asbestos fibers, and these minor episodic exposures would not have been significant enough to increase Plaintiffs’ risks of developing asbestos-related diseases. The Commission did not err in considering the “frequency” of Plaintiffs’ exposure to airborne asbestos fibers.

#### 4. Last Injurious Exposure

Plaintiffs make no arguments on appeal concerning the liability determinations made by the Commission pursuant to N.C.G.S. § 97-57. Therefore, any such arguments are deemed abandoned. Although, as discussed above, the Commission was not required to make any determination pursuant to N.C.G.S. § 97-57—once it determined that Plaintiffs had failed to prove their alleged asbestosis arose out of their employment, and was therefore not compensable—the Commission did make this determination in its ultimate findings 16, 19, 21, 23, 43 and 45.

For example, in ultimate finding 43 the Commission determined that Plaintiffs had not been “exposed to the hazards of asbestosis through [their] employment with [D]efendant for 30 days or parts thereof within a seven-month consecutive period which proximately augmented the disease process of asbestosis to the slightest degree.” That unchallenged determination relieved Defendant of any liability for Plaintiffs’ alleged asbestosis—even assuming, *arguendo*, that all Plaintiffs had asbestosis, and that their asbestosis was compensable. This is because the “last injurious exposure” analysis is only concerned with which employer—or insurance

company—will be held liable for a proven compensable occupational disease. *See Penegar*, \_\_ N.C. App. at \_\_, 815 S.E.2d at 398.

### 5. Conclusion

It was the province of the Commission to decide, based on competent evidence, what factors Plaintiffs needed to prove in order to meet the burden of proving asbestos exposure at the factory was a significant causal factor in the development of their alleged asbestosis. Based on the evidence presented, the “form” of the asbestos, and the “quantity” and “frequency” of exposure, were legitimate considerations in making this determination. Therefore, the Commission’s ultimate findings that state “[t]he greater weight of the evidence in view of the entire record shows that [P]laintiffs were [not] exposed to airborne asbestos . . . in such form and quantity and with such frequency as to cause . . . asbestosis” do not show that the Commission “placed an impermissible burden” on Plaintiffs.

In addition, the Commission also made the following ultimate finding in finding 43:

Given the evidence of air contaminant measurements taken at [the] factory, the pathology evidence collected from workers’ lungs, and the scientific and epidemiological literature presented on the subject, the greater weight of the evidence in view of the entire record does not demonstrate a causal connection between asbestosis and employment at the . . . factory.

This ultimate finding—which determined Plaintiffs failed to prove the required causal connection, *Patton*, 239 N.C. App. at 375, 768 S.E.2d at 355, and other

authority cited—does not contain the language to which Plaintiffs object. Finally, the Commission's unchallenged determinations pursuant to N.C.G.S. § 97-57 serve to relieve Defendant from any liability for Consolidated Plaintiffs' alleged compensable asbestosis. This argument is without merit.

*B. Competent Evidence*

We have held that Defendant cannot be held liable for Plaintiffs' alleged asbestosis, even were it compensable, due to the Commission's unchallenged N.C.G.S. § 97-57 determinations. However, in light of the number of Consolidated Plaintiffs impacted by this opinion, we address Plaintiffs' remaining arguments. Plaintiffs primarily argue that certain evidence relied upon by the Commission was not competent. However, as this Court has stated:

Although [Plaintiffs] point[] to . . . evidence which [they] feel[] was incompetent to support [some of] the . . . Commission's findings of fact, we find it unnecessary to decide those points of contention in light of the rule that findings of fact which are supported by competent evidence are conclusive on appeal, *even though other incompetent evidence may have been improperly admitted*.

*Kennedy*, 101 N.C. App. at 33, 398 S.E.2d at 682 (citation omitted) (emphasis added).

Therefore, even assuming, *arguendo*, the Commission relied upon some incompetent evidence, our review is limited to whether the competent evidence was sufficient to

support the Commission's findings of fact—including its ultimate findings—and whether the findings support its conclusions and rulings.<sup>24</sup> *Id.*

Plaintiffs' arguments fall into the following general categories: (1) The "air sampling" evidence and the "fiber year theory"; (2) reliance on "non-medical" expert testimony; (3) reliance on the lung pathology from the five deceased Plaintiffs; and (4) the Commission's reliance on the above allegedly incompetent evidence in support of its ultimate findings and conclusions.

### 1. Air Sampling and Fiber Year Theory

Plaintiffs argue that "the Commission erred in relying on the 'fiber year theory'" and air sampling to determine that Plaintiffs were not exposed to sufficient amounts of airborne asbestos at the factory to cause asbestosis. We disagree.

Specifically, Plaintiffs argue that the "Commission never stated what level of exposure was necessary to cause a disease except to subscribe to [D]efendant's usage of the 'fiber year theory.'" Plaintiffs further argue that Defendant "could not prove the amount of [Plaintiffs'] exposure to asbestos and could only provide a very broad guess at the level necessary to cause a disease." Plaintiffs also argue that the Commission ignored the fact that incidents of airborne asbestos being released in the factory were "occasional," not constant, and, therefore, "no one knows how much

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<sup>24</sup> Plaintiffs did not seek to suppress the evidence they now challenge on appeal—either prior to or during the hearings. Although hearings before the Commission are *quasi*-judicial, this Court has applied N.C.G.S. § 8C-1, Rule 702 in its review of workers' compensation claims. *Wise v. Alcoa, Inc.*, 231 N.C. App. 159, 752 S.E.2d 172 (2013).

asbestos was being damaged on any particular day. No one knows how much asbestos was inhaled by [Plaintiffs]. The only evidence of the levels of the asbestos in the air was from air sampling done in the facility.”

Plaintiffs may be correct that “[n]o one knows how much asbestos was inhaled by” Plaintiffs, but it was *Plaintiffs’* burden to prove that their alleged exposure to asbestos fibers at the factory caused or significantly contributed to their alleged asbestos-related diseases. The fact the Commission did not include findings of fact related to all the evidence that Plaintiffs believe supported their claims does not mean the Commission ignored this evidence. “[T]he Commission does not have to explain its findings of fact by attempting to distinguish which evidence or witnesses it finds credible.” *Deese v. Champion Int’l Corp.*, 352 N.C. 109, 116, 530 S.E.2d 549, 553 (2000) (citation omitted). “Requiring the Commission to explain its credibility determinations . . . would be inconsistent with our legal system’s tradition of not requiring the fact finder to explain why he or she believes one witness over another or believes one piece of evidence is more credible than another.” *Id.* at 116-17, 530 S.E.2d at 553. Further, the Commission stated that it had “reviewed and considered all hearing and deposition transcripts, along with all evidentiary exhibits, arguments, and briefs in reaching a decision[.]” The opinion and award contains over 25 pages devoted to listing the transcripts, depositions, and exhibits considered by the Commission. The Commission also stated multiple times throughout the opinion

and award that its determinations were based upon the “greater weight of the evidence in view of the entire record[.]”

Plaintiffs continue: “Yet the Commission found that [Plaintiffs were] not exposed to . . . levels” of asbestos sufficient to cause asbestosis. Plaintiffs again suggest that it was Defendant’s burden to disprove Plaintiffs’ claims when it was Plaintiffs’ burden to prove all the elements necessary to show compensable asbestos-related diseases. Further, Plaintiffs did not direct this Court to any part of the Commission’s opinion and award in which the Commission “found that [Plaintiffs were] not exposed to” sufficient “fiber years” of asbestos to cause asbestosis. This is because “fiber years” are not discussed in the opinion and award. There is no evidence that the Commission subscribed to the fiber year theory, or relied on it when making its relevant findings and conclusions.

Plaintiffs argue: “There was no evidence introduced by [D]efendant to establish whether [P]laintiffs’ exposure was consistent with the background level of the [factory]. There was no evidence introduced showing the levels of exposure when [P]laintiff[s] w[ere] damaging insulation, using compressed air on damaged insulation or cutting asbestos gaskets.” It was Plaintiffs’ burden to introduce this evidence, and Defendant had no burden to convince the Commission that Plaintiffs’ alleged injuries did not arise in the course of employment at the factory.

Although Plaintiffs presented evidence to the contrary, there was plenary evidence—including evidence unrelated to air sampling or the fiber year theory—to

support the Commission's ultimate finding that Plaintiffs were not—due to their work at the factory—exposed to asbestos sufficient to cause asbestosis. For example, Dr. Ghio testified as follows:

[DR. GHIO]: Again, the only [air sampling] levels that I'm aware of are those taken during the health hazard evaluation done by NIOSH in their review of the plants across the Midwest[.] . . . . And the ones that [Defendant's attorneys] forwarded to me regarding [the factory]. This is outside of my expertise though. I'm not an industrial hygienist. I'm a pulmonologist.

. . . .

Q. Sir, do you think it's proper for you to make an attribution of cause and effect when you acknowledge that you're unaware of any of the exposure levels for disease in the tire industries?

A. I'm aware of the levels at [the factory]. They were forwarded to me. Regarding a more global approach to that question, you know, can a physician be called upon to make a diagnosis of asbestosis without being aware of the actual dust levels in the environment, and it's very rare that, as pulmonologists . . . we're made [aware of] those values. We make diagnoses all the time of asbestosis. 99 percent of all diagnoses of asbestos[is] are made without any awareness of such levels. Dr. Ohar and Dr. Schwartz [Plaintiffs' medical experts] were unaware of levels when they diagnosed these patients to have asbestosis.

Dr. Ghio's testimony shows he was not, to any significant degree, "relying on the 'fiber year theory'" or the air sampling in order to reach his conclusions. Further, as noted in finding of fact 38: "Dr. Roggli testified that there was not sufficient exposure to asbestos at the factory in question to contribute to or to cause an asbestos-related

disease for Mr. Jones or anyone in his position.” This opinion was not based air sampling from the factory.

Dr. Ghio also testified: “[Asbestos] was simply there [in the factory], and by being there, [Plaintiffs] misinterpret that to [mean] that they’re at increased risk. They’re aware that piping in [the factory] had asbestos, and they have—they have the misconception that that increases their risk for asbestosis, and it does not.” Plaintiff’s expert, Ewing, agreed with Dr. Ghio in this regard, stating: “I’m not of the opinion that because pipe insulation is present there must be exposure. There has to be work going on on the pipe insulation or some disturbance of that material for the exposure to arise.” In forming his opinions, Dr. Ghio relied heavily on Plaintiffs’ patient histories, and review of their x-rays:

[PLAINTIFFS’ COUNSEL:] Dr., with respect to exposure history, you’ve testified in the past, have you not, that you put more emphasis on what the patient says than you do actually specific [airborne] fiber levels, sir. Do you remember that testimony?

A. I do. I follow the ATS [(“American Thoracic Society”)] criteria which is—I base my diagnosis whenever possible on an accurate occupation history.

Q. All right. So in the case of [Plaintiffs], you really never looked at what the industrial hygiene reported as to what the individual exposures were or the sampling was. Is that not true, sir?

A. I have been provided actual values of fiber measurements, and actual fiber values of fiber measurements were in agreement with the histories, and that is that the exposure was minimal.



Q. Well, let me ask you, sir. [W]hen you gave your report and had your opinions about [Plaintiffs], you did not at any time mention the fiber levels. Did you, sir, or the industrial hygiene results?

A. I don't believe I did.

Q. All right. And you've testified repeatedly . . . that patient histories are the best indicator of exposures, even over specific fiber levels. Has that been your testimony, sir?

A. Well, it's very rare for me to get . . . specific fiber levels. So, yes, that has been my testimony in the past. And as a physician, we take occupational histories. That's what we do.

. . . .

Q. Sir, . . . you do require an occupational environmental history, but you go along to also require that the industry and occupation place the patient at an increased risk. And also, you require a marker of exposure, usually pleural plaque, sir. Do you see that?

A. I don't require all three. I require an occupational, and environmental history that increases the patient's risk for asbestosis or I need a marker of exposure. If I see pleural plaques that are bilateral, I assume that that individual is at an increased risk.

THE COURT: And that's the marker.

[DR. GHIO]: That's the marker. You know, if that person has had enough fiber, you know, even though [their occupation is] lower in the pyramid, it's way down at the bottom, I assume, you know, they had bilateral plaques, I'm going to give them the benefit of the doubt they had the exposure.

THE COURT: And you, in looking at all the x-rays from [Plaintiffs], you didn't see anything that was a marker in any of them.

[DR. GHIO]: Not a single one.

Dr. Ghio stated that his opinions were based on the following:

[DR. GHIO]: [T]he tire industry has never been reported in the medical literature to be associated with asbestosis. I've looked at the industrial hygiene behind the exposures of [the factory], and forty years would not make the criteria of twenty-five fiber years. I've been looking at a lot of these chest x-rays. I've not seen any evidence of asbestosis. I'm not seeing any evidence for even those diseases that require very, very minute exposures to fibers, and those would be pleural plaques. *I don't see any evidence of a significant exposure. So I think because of . . . all the above, I don't think an individual working at [the factory] is at increased risk for asbestosis.* [Emphasis added].

To the extent that the Commission relied on the air sampling results as consistent with the testimony of Defendant's experts that asbestos exposure at Defendant's plant would not be sufficient to cause Plaintiffs' alleged asbestosis, because the air sampling reports never indicated significantly elevated levels of dust or asbestos fibers, the Commission did not err in considering that evidence. Had the air sampling results shown elevated levels of asbestos during the testing periods, that would have been relevant evidence favorable to Plaintiffs that the Commission would have properly considered. The fact that none of the air sampling indicated elevated asbestos levels does not alter the relevance of the evidence, nor render it incompetent—it simply tends to support Defendant's position more than Plaintiffs'.

It was for the Commission to determine the weight to give to that evidence, and Plaintiffs fail to demonstrate that the Commission abused its discretion in that regard. *See Wise*, 231 N.C. App. at 164, 752 S.E.2d at 175–76. This argument is without merit.

## 2. Medical Expert Evidence

Plaintiffs argue that “the Commission erred by relying only on non-medical expert testimony” because “the amount of exposure necessary to cause disease is a medical question [that] only a physician can answer.” We disagree.

Plaintiffs contend that there was “insufficient medical expert testimony for the Commission to determine that [Plaintiffs were] not exposed to sufficient levels of asbestosis to cause a disease.” Again, it was Plaintiffs’ burden to present evidence, medical or otherwise, to prove sufficient exposure to asbestos—not Defendant’s burden to prove insufficient exposure. Plaintiffs bore the burden of producing “competent evidence to support the inference that the [exposure] in question resulted in the injury complained of[.]” *Click v. Freight Carriers*, 300 N.C. 164, 167, 265 S.E.2d 389, 391 (1980). Plaintiffs rely in part on *Click* in support of their argument. *Click* states:

The quantum and quality of the evidence required to establish *prima facie* the causal relationship will of course vary with the complexity of the injury itself. There will be “many instances in which the facts in evidence are such that any layman of average intelligence and experience would know what caused the injuries complained of.” On the other hand, where the exact nature and probable

genesis of a particular type of injury involves complicated medical questions far removed from the ordinary experience and knowledge of laymen, only an expert can give competent opinion evidence as to the cause of the injury.

*Id.* (citations omitted). *Click* was not an asbestosis case, and the injury involved in *Click* required very different causation evidence. Each case is fact specific, and Plaintiffs cite to no authority that would *per se* exclude reliance on non-medical expert testimony when deciding whether a particular employment could have caused or contributed to development of an asbestos-related disease.<sup>25</sup>

In asbestosis cases, diagnosis of the disease itself requires expert medical testimony. However, once asbestosis is established, expert medical testimony is not necessarily required to establish a causal connection between the disease and the worker's employment. For example, if a plaintiff has been diagnosed with asbestosis, non-medical evidence that the only place the plaintiff was exposed to asbestos was while working for the defendant-employer should be sufficient to prove a causal connection. This Court has reasoned: "If a plaintiff has not been exposed in prior employment, and has asbestosis, then that could give rise to an inference that he was exposed (and last injuriously exposed) while working for defendant-employer." *Vaughn v. Insulating Servs.*, 165 N.C. App. 469, 474, 598 S.E.2d 629, 632 (2004). Conversely, if an industrial hygienist testified that the plaintiff's workplace

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<sup>25</sup> Further, as noted above, Plaintiffs did not object to the testimony of Defendant's industrial hygienists.

contained no asbestos, the Commission could properly determine that the plaintiff had failed to prove a causal connection. In *Vaughn*, the “plaintiff argued the Commission improperly required him to produce scientific or medical evidence of exposure to asbestos for the relevant time period while in defendant’s employ.” *Id.* at 473, 598 S.E.2d at 631 (citation omitted). This Court held that “[p]laintiff [wa]s correct that there [wa]s no need for such expert testimony.” *Id.* “This does not mean, however, that the Commission cannot consider expert testimony, or the lack thereof, along with lay testimony, in weighing the evidence and determining whether claimant has met his burden of proof.” *Id.* at 473, 598 S.E.2d at 632. The Commission was free to consider all the evidence, lay and expert, to inform its conclusion that Plaintiffs failed to meet their burden on the issues of exposure and causation.

In addition, Plaintiffs acknowledge that their contention is not accurate, admitting: “The Commission did rely on [Defendant’s expert] Dr. Ghio who is a medical expert.” Plaintiffs then incorrectly argue: “The only other medical expert offered by [] Defendant was Dr. [Selwyn] Spangenthal” (“Dr. Spangenthal”).<sup>26</sup> For example, Defendant also presented live testimony from Dr. Kenneth Samuel Karb,

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<sup>26</sup> Further, Plaintiffs are also incorrect in claiming Dr. Spangenthal “testified that [Plaintiffs] had sufficient exposure to asbestos to cause a disease.” Dr. Spangenthal was asked by Plaintiffs’ counsel, for each Bellwether Plaintiff: “And if [the individual Plaintiff’s] testimony is true [concerning his exposure to asbestos in the factory], assuming that for the sake of my question, . . . would that exposure have been significant . . . enough to cause disease?” Dr. Spangenthal responded “yes,” but added “I just assume that, you know, [Plaintiff] knows where he works, and that’s his impression of what . . . his exposure was, and that’s what I’ve noted down. Whether it’s true or not, I have no idea.” The Commission was not required to give weight to Bellwether Plaintiffs’ own accounts of alleged asbestos exposure—whether given as testimony during the hearings, or given to physicians taking their histories.

Dr. David Allen Hayes (“Dr. Hayes”), and Dr. Roggli. Deposition testimony was presented from, *inter alia*, Dr. Oury, Dr. Gregory S. Parsons, Dr. Robert Reuter, and Dr. Sporn. Medical records and reports were entered into evidence from multiple additional physicians.

Dr. Roggli offered, *inter alia*, his opinions that he would not “typically expect to see asbestos-related disease” associated with work within the tire industry, and the medical literature supports his opinion that tire plant workers are not exposed to a greater risk of asbestosis than the general public; that in his medical practice he has “not seen any asbestos-related lung cancers or asbestosis, to my recollection, from anybody . . . working with the tire industry”; that, contrary to Plaintiffs’ evidence, the appearance of “a few short chrysotile fibers in the lung” of an individual is “exactly what you find in lung tissue from the people from the general population”; that it would be very unusual to examine lung tissue from the general population and fail to find any measurable asbestos fibers; that, contrary to Plaintiffs’ evidence, asbestos fibers less than five microns in length “would not be disease-producing”; that approximately ninety percent of the scientific community was of the same opinion; and that, based upon his pathological analysis of Plaintiff Jones’ lung sample, he was “completely ruling out asbestosis[.]” Further, based on his fiber count analysis of Plaintiff Jones’ lung tissue, Dr. Roggli testified to his opinion “within a reasonable degree of scientific certainty,” that neither Plaintiff Jones, nor “a person in [Plaintiff]

Jones' position" would have received "sufficient exposure to asbestos at [the factory] . . . to contribute to an asbestos-related disease[.]"

Dr. Ghio testified concerning why he did not trust the Plaintiffs' experts methodology when only ten percent of Plaintiffs diagnosed as having asbestosis also showed signs of pleural plaques:

[DR. GHIO:] . . . . *Eighty percent of the time [patients] have pleural plaques if they have significant exposure to asbestos.*  
. . . .

THE COURT: Well, let me ask it more specifically. On a . . . one zero,<sup>[27]</sup> are they going to have pleural plaques?

[DR. GHIO]: Yes, eighty percent of them will.

THE COURT: Okay.

[DR. GHIO]: *If it's truly the result of significant exposure to fibers, eighty percent of the chest x-rays[—S]omewhere between fifty and eighty percent—and I like the eighty actually. More of the studies have come out with eighty percent will show pleural plaques if it is attributable—if it's truly the result of fiber exposure. [Emphasis added].*

Dr. William Franklin Alleyene, II ("Dr. Alleyene"), Plaintiffs' expert, agreed that pleural plaques will be present in approximately eighty percent of people who have asbestosis. Dr. Ghio further testified:

[DR. GHIO:] [T]here have been many diagnoses of asbestosis that I've made on individuals who have come in, and they're part of that twenty percent or the fifty percent that don't have pleural plaques. But that's an individual.

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<sup>27</sup> The deputy commissioner is referring to a B-readers assessment of 1/0 as explained in finding of fact 25.

*When you have a group of 157, you have the benefit of numbers here. And if in 157 individuals you're looking at zero plaques, then you have to come to some conclusion about the exposure.*

THE COURT: Well, you're saying—basically from your testimony, if I'm—and I want to make sure I understand what you've testified correctly. I'm just trying to clarify. Out of the 158, you would expect something like 120 to have pleural plaques on their x-rays.

THE WITNESS: That is correct.

THE COURT: Therefore, and because there are no pleural plaques, if other people are diagnosing one zeros on 157 x-rays, [and] somewhere about . . . 75 or so to 120 don't have pleural plaques, you're saying that those x-rays generally are being misread.

[DR. GHIO]: That's correct.

. . . .

[DR. GHIO]: And there's nothing in the medical literature to support this possibility.

Dr. Ghio then expresses his medical expert opinion that Plaintiffs were not exposed to sufficient asbestos working at the factory to cause disease:

I don't think that any contribution . . . from the work environment [in the factory] would significantly increase one's risk [of contracting an asbestos-related disease]. But[, hypothetically,] these individuals may have [been exposed to significant amounts of asbestos fibers in prior employment]. . . . I believe that they could be diagnosed to have asbestosis if they're a Continental Tire worker, but it had nothing to do with the environment at [the factory].

THE COURT: To any degree?



[DR. GHIO]: To any degree.

In addition, Plaintiffs' expert Dr. Alleyene, testified concerning the superiority of CT scans over x-rays in diagnosing asbestosis:

X-rays are based on technology that's over a hundred years old, and it's like looking at something with a naked eye when you've got a microscope right next to you. And I can see things on a CT scan that is—are much more detailed [and] three-dimensional, and, also, it takes a lot of the guess work out of it. The—somebody looks at an x-ray, a B-reader, and they say, “Well, gee, I see these shadows, and they look like 1/0” versus on a CT scan I can see fibrosis, I can see pleural plaquing, I can see all types of things and so when physicians talk about making that diagnosis whether it's in a board review course or anything, no one even mentions a B-read chest x-ray. That is something that exists only in the courtroom. Physicians, when we talk about asbestosis, when we talk[] about diagnosing asbestosis and how to manage patients with asbestosis, we speak specifically about high resolution CT scans. We don't even talk about an x-ray other [than] to say, “Gee, if your x-ray is unclear or suspicious, you get a CT scan to confirm.”

The deputy commissioner, based on Dr. Alleyene's testimony, proposed that Plaintiffs and Defendant agree to allow independent doctors who were experts in asbestos-related diseases to administer and analyze high resolution CT scans of every living Plaintiff. Defendant wanted the deputy commissioner to order Plaintiffs to obtain high resolution CT scans, and Defendant agreed to pay for the procedures and the analysis. Plaintiffs' counsel informed the trial court that they would not agree to having Plaintiffs undergo high resolution CT scans. The deputy commissioner responded that Plaintiffs' refusal “cuts both ways. And based on—based on the

evidence I have heard thus far, I again say that decision . . . cuts both ways—okay—cuts both ways because [P]laintiffs have the burden of proof here[.]”

Both Plaintiffs and Defendant presented medical expert testimony that the only certain method of diagnosing asbestosis is to examine a sufficient sample of the patient’s lung tissue. Plaintiffs’ attorney, in response to the deposition testimony of Dr. Spangenthal that CT scans were “the gold standard” for diagnosing asbestosis, stated: “Actually, the gold standard would probably be a biopsy, isn’t it?” Dr. Spangenthal agreed, noting that he was referring to procedures that he would generally perform on living patients. When Plaintiffs’ expert, Dr. Alleyene, was asked if there were any symptoms that are “pathopneumonic” for asbestosis—*i.e.* “a sign or a symptom or a finding that would be so closely correlated with a specific disease entity as to be virtually diagnostic”—he responded:

There are what we call asbestos bodies[.] And if one found a certain concentration of asbestos bodies per gram of lung tissue, that would be pathopneumonic of asbestosis. Having said that, the way that you would get that tissue would require a procedure that is fairly invasive called an open-lung biopsy where they literally spread your ribs, take a piece of lung tissue that would be significantly larger than one could obtain from other methods[.] This—you would get a nice wedge of lung tissue, literally. And if they then prepped that tissue . . . and looked at it under an electron microscope, you could see these linear—they’re not fibers but they’re actually coated asbestos fibers, and they call them asbestos bodies[.] And if one has an open-lung biopsy and if one has the finding of these asbestos bodies or sufficient numbers of these asbestos bodies either in the tissue itself or in the lung fluid . . ., then that would be pathopneumonic.

Q. Short of taking a wedge of somebody's lung, there's nothing pathopneumonic about asbestosis?

A. That is correct.

Dr. Oury agreed with Dr. Alleyene that “pathology [is] still the only way to definitively diagnose asbestosis[.]” Dr. Hayes also testified for Defendant, and agreed that pathology is the most accurate way to diagnose asbestosis, followed by high resolution CT scan. When Dr. Roggli was asked: “Would it be safe to say that, if you don't have it pathologically, you don't have it?” He responded: “Correct. That is assuming that you have a reasonable, decent sample of tissue, that would be correct.” Dr. Roggli also testified that the best tool for diagnosing asbestosis is “pathologic examination,” followed by “high-resolution CT—regular CT would be less sensitive, and least sensitive would be the routine chest x-ray.” Plaintiffs provided Defendant lung tissue from five of the eighteen deceased Plaintiffs; Defendant submitted lung tissue from all five for pathological examination, and none of the lung tissue from these deceased Plaintiffs, including Plaintiff Hinson, showed asbestos bodies or other signs of disease related to asbestos exposure.

In Plaintiffs' brief, they argue that the “Commission ignored the testing done by Plaintiffs' pathologists. This testing irrefutably showed excess levels of asbestos in the lungs of the workers.” However, Plaintiffs did not offer testimony from any

pathologists.<sup>28</sup> The Commission can “consider expert testimony, *or the lack thereof*, along with lay testimony, in weighing the evidence and determining whether claimant has met his burden of proof.” *Vaughn*, 165 N.C. App. at 473, 598 S.E.2d at 632.

The common issues findings of fact demonstrate that evidence from medical experts factored heavily in the determinations of the Commission—see findings 1, 25, 26, 27, 28, 29, 30, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, and 44—the Commission simply gave the testimony of certain of Defendant’s medical experts greater weight. It was the province of the Commission to determine the credibility of, and the weight to be given to, the various expert witnesses, including the medical experts. *Wise*, 231 N.C. App. at 164, 752 S.E.2d at 175–76.

Because Plaintiffs had “the burden of proving [their claims] by . . . a ‘preponderance of the evidence[.]’” they were required to “present credible evidence of [sufficient asbestos] exposure[.]” *Id.* We hold that the Commission properly relied on both medical and non-medical evidence—expert and lay—when considering the issue of causation in this matter. Plaintiffs’ refusal to agree to certain more accurate medical procedures was also proper to consider. *Id.* Plaintiffs’ argument is without merit.

### 3. Extrapolating the Evidence

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<sup>28</sup> The only record evidence of Plaintiffs having obtained analysis of a deceased Plaintiff’s lung tissue was the fiber count done on Plaintiff Jones’ lung tissue by Rigler—who is not a medical doctor.

As part of the section challenging certain findings of fact, Plaintiffs include the following two sentence argument: “[T]he Commission is attempting to use lung tissue samples from some workers to determine the amount of asbestos in the lungs of all [Plaintiffs]. That is speculation and there was no evidence of the relevance of the lung pathology to other workers.” We hold the results of the lung tissue analyses of the deceased Plaintiffs were a proper factor for the Commission to consider, and were relevant to the Commission’s decision. Plaintiffs’ brief includes testimony from multiple witnesses contending that exposure to asbestos in the curing department was worse than anywhere else in the plant. Plaintiffs argue on appeal that “[t]he condition of the insulation was especially bad in the curing department.” In finding 32, the Commission determined: “Pathological examination of lung tissue is a definitive method of determining whether an individual has an asbestos-related disease.” In finding 37, the Commission found that Plaintiff Hinson worked in the factory

for 32 years, mainly in the curing department. The curing department had the highest concentration of insulated piping in the factory, with much of it at floor level or in exposed trenches. According to decedent Hinson, he was also exposed to significant asbestos dust from using a band saw to cut large asbestos gaskets. If [P]laintiffs’ arguments are correct, decedent Hinson would have been exposed to a significant amount of airborne asbestos. . . . Dr. Craig Hart at York pathology performed [P]laintiff Hinson’s lung autopsy. Dr. Hart found no evidence of asbestos bodies or fibrosis, but did see evidence of smoking. The tissue was sent to Dr. Oury, who examined the sample and confirmed Dr. Hart’s conclusions. Although it was not required for

diagnostic purposes due to the lack of fibrosis, a fiber count analysis was done by Dr. Oury upon [D]efendant's request. The fiber count analysis found 5 asbestos bodies per gram, which is a level well below that seen in individuals with asbestosis and in the range of control individuals with no history of asbestos exposure.

Despite Plaintiffs' arguments and evidence suggesting Plaintiff Hinson should have been exposed to more airborne asbestos than Plaintiffs who worked in other areas of the factory, Plaintiff Hinson's lung pathology demonstrated that not only did he not have asbestosis, his exposure to asbestos was at "a level well below that seen in individuals with asbestosis and in the range of control individuals with no history of asbestos exposure." This evidence was relevant to the Commission's decision regarding the overall levels of airborne asbestos in the factory, and properly considered for that purpose.

Plaintiffs also argue that they were exposed to significant levels of asbestos in other areas of the factory. Plaintiffs claim that the asbestos insulation in the plant that "was in a damaged, deteriorated and [] dangerous condition" was "located in the work areas of the employees including the [P]laintiffs herein." One of Plaintiffs' witnesses testified that "the insulation was torn off, beaten off, and looked ragged[,] and "that this condition was consistent throughout the plant." Plaintiffs agree in their brief that "[t]he poor condition of the asbestos insulation was not restricted to a single area but was consistent throughout the plant." In addition, Plaintiffs argue they were exposed to airborne asbestos fibers throughout the plant due to the use of

talc contaminated with chrysotile asbestos; and the removal, replacement, and repair of gaskets and brakes that contained chrysotile asbestos. Plaintiffs argued to the Commission that the factory was “a very dusty plant” and that any plaintiff who worked throughout the factory “would have been in each of these departments and subject to the same type of exposures [as] everyone else.”

In finding 34, the Commission stated: “Of the five deceased [P]laintiffs who had post-mortem pathological study of their lung tissue, (Walter Hinson, Johnnie Jones, Charles Gibson, Homer Hunt, and Lloyd Cox), none had pathological evidence of asbestosis. Pathology is the most reliable method to diagnose asbestosis.” In findings 38, 39, 40, and 41, the Commission determined that deceased Plaintiff Jones worked for twenty-five years in the calendar area—where he would have been exposed to talc, along with pipe insulation; deceased Plaintiff Gibson worked for thirty-one years in the tire-building and warehouse departments; deceased Plaintiff Hunt worked for seventeen years throughout the factory as a mechanic—which would have exposed him to the alleged brake-related asbestos dangers as well as all other alleged causes of airborne asbestos in the factory; and deceased Plaintiff Cox worked for thirty-one years in the stock and bead preparation areas. The Commission determined in finding 42: “Despite Plaintiffs’ theories of exposure, pathology results from the lung tissue of five long-term employees from a variety of departments and factory locations uniformly show a lack of fibrosis, a lack of asbestos bodies, and a lack of fibers.”

The above findings of fact illustrate the relevance of the pathological examinations to the general issue of whether employment at the plant served to expose Plaintiffs to asbestos of the type and quantity that could cause or significantly contribute to the development of an asbestos-related disease. The lung tissue pathology was direct evidence that none of the deceased Plaintiffs had asbestosis or other asbestos-related diseases, and was also circumstantial evidence supporting the Commission's determination that Plaintiffs had failed to prove "a causal connection between asbestosis and employment at the . . . factory."

#### 4. The Entire Record

Plaintiffs challenge the Commission's findings 16, 19, 21, 23, 43, 44, and 45, because the Commission stated that it was basing its determinations on "the greater weight of the evidence in view of the entire record." Plaintiffs argue that the "entire record" language demonstrates that the Commission based these findings, *in part*, on incompetent evidence and, therefore, these findings and conclusions are invalid. Plaintiffs argue: "The 'entire record' consisted of the air sampling and testimony of experts regarding the amount of exposure for each [P]laintiff and the amount necessary to cause disease. As stated herein, [Plaintiffs] find[] that such evidence is not competent. Regardless, the Commission based its opinions on that evidence." Plaintiffs also contend that "all of the testimony relied upon by the Commission to establish the levels of exposure were based upon air sampling." As the Commission's findings of fact, and the small sampling of the expert testimony included herein



demonstrates, the Commission relied primarily on expert medical testimony, not air sampling, and Plaintiffs' argument fails for this reason.

In addition, "[b]efore the Commission makes findings of fact, it 'must consider and evaluate all of the evidence. Although the Commission may choose not to believe the evidence after considering it, it may not wholly disregard or ignore competent evidence.'" *File v. Norandal USA, Inc.*, 232 N.C. App. 397, 400, 754 S.E.2d 202, 205 (2014) (citation omitted). It would have been improper for the Commission not to have considered "the entire record" before making its determinations. Further, the Commission is not required to make specific findings indicating the evidence it is *not* relying on. *Bryant v. Weyerhaeuser Co.*, 130 N.C. App. 135, 139, 502 S.E.2d 58, 62 (1998). Finally, "findings of fact which are supported by competent evidence are conclusive on appeal, even though other incompetent evidence may have been improperly admitted." *Kennedy*, 101 N.C. App. at 33, 398 S.E.2d at 682 (citation omitted). This argument is without merit.

### *C. Findings of Fact*

Plaintiffs contest certain findings of fact, in whole or in part.<sup>29</sup> Our review is limited to "whether the findings of fact are supported by competent evidence[.]" *Penegar*, \_\_ N.C. App. at \_\_, 815 S.E.2d at 394 (citation omitted). Uncontested findings are binding on appeal. *Id.* We include as "unchallenged" many findings of

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<sup>29</sup> Plaintiffs challenge some of the "common issue" findings in certain individual briefs, but not others. For the sake of clarity, we address the challenges from all of the bellwether briefs related to common issues in this opinion.

fact that Plaintiffs purport to challenge on appeal—because Plaintiffs’ “challenges” to these findings are not based on any alleged insufficiency of supporting evidence. *Id.*

Plaintiffs challenge certain findings based on the following sentence: “In Findings of Fact 43, 44, 45, 47 the Commission relies on evidence that is not competent for the reasons set forth herein.” That is the totality of the challenge, and it is not sufficient for appellate review. Plaintiffs challenge findings 12, 13, 14, 15, 16, 18, 19, 21, and 23 by arguing that the Commission should not have relied on the air sampling reported in these findings. However, Plaintiffs do not contend that the findings themselves are not supported by competent evidence. These findings simply state uncontested facts concerning the air sampling done at the factory, and do not include any indication of how, or if, the Commission relied on these findings to make its decisions.<sup>30</sup>

Plaintiffs challenge findings 15, 35, 36, 37, 38, 39, 40, 41, 42, 44, and 47, on the basis that the Commission “gave more credibility to [the opinions and testimony of] Defendant’s experts” and “failed to consider, and plainly ignored, the evidence that contradicted” the opinions of Defendant’s experts. Again, this is not an argument concerning whether there was sufficient evidence to support these findings—Plaintiffs simply argue that they disagree with the weight and credibility

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<sup>30</sup> Findings 16, 19, 21, and 23 are ultimate findings, but they are not properly challenged in this section.

determinations of the Commission.<sup>31</sup> That is not a valid challenge to findings of fact, and this Court is without authority to make the determinations Plaintiffs ask of it:

In passing upon issues of fact, the Industrial Commission is the sole judge of the credibility of the witnesses and the weight to be given to their testimony. The Commission may accept or reject the testimony of a witness solely on the basis of whether it believes the witness or not. The findings of the Industrial Commission are conclusive on appeal when supported by competent evidence even though there be evidence to support a contrary finding.

*Hilliard v. Apex Cabinet Co.*, 305 N.C. 593, 595, 290 S.E.2d 682, 683–84 (1982) (citations omitted). Therefore, these “unchallenged” findings of fact are binding on appeal.<sup>32</sup> *Penegar*, \_\_ N.C. App. at \_\_, 815 S.E.2d at 394.

Plaintiffs challenge finding 11, arguing that the Commission’s determination that the greater weight of the evidence did not support a finding that there were high levels of airborne particulates in the curing department “ignores evidence and misstates the evidence. Plaintiff[s] never suggested that workers were damaging the asbestos on the pipes every minute of every day. It was occasional exposures.” We agree that “high levels of airborne particulates in the curing department originating from damaged pipe insulation and gasket-sawing” would have been intermittent. However, there was sufficient evidence to support this finding as written. As the Commission stated in its ultimate finding 43:

In occupational disease cases, a causal connection between

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<sup>31</sup> Finding 47 is an ultimate finding or conclusion, but Plaintiffs’ argument is insufficient to challenge this conclusion as well.

<sup>32</sup> We further hold that all these findings of fact are supported by competent record evidence.

the employment and the alleged disease must be proven. This analysis includes the extent of exposure. Of necessity, evidence on the subject of causation in these cases is often circumstantial. Given the evidence of air contaminant measurements taken at [D]efendant's factory, the pathology evidence collected from workers' lungs, and the scientific and epidemiological literature presented on the subject, the greater weight of the evidence in view of the entire record does not demonstrate a causal connection between asbestosis and employment at the . . . factory.

The Commission is correct in its statement of law: "In the case of occupational diseases proof of a causal connection between the disease and the employee's occupation must of necessity be based on circumstantial evidence." *Booker*, 297 N.C. at 476, 256 S.E.2d at 200. The pathology evidence, the testimony of Defendant's medical experts and industrial hygienists, the scientific and epidemiological literature, and other evidence presented, constituted sufficient evidence to support the finding that "the greater weight of the evidence [did] not support" that there were "high levels of airborne particulates in the curing department originating from damaged pipe insulation and gasket-sawing"—even though Plaintiffs presented evidence in support of a contrary finding.

Plaintiffs also argue that in finding 12 the Commission "completely ignored the fact that the 1979 study was not measuring for asbestos." However, finding 12 correctly states that the study "measured for dust—both airborne and respirable, as well as petroleum distillates, rubber solvent, Benzene, and Toulene. The dust measurements would have measured any particulates in the air—whether the

particulates were asbestos, talc, or something else.” We hold that all of the “common issues” findings of fact included in this opinion are binding on appeal.

*D. Ultimate Findings and Conclusions of Law*

Plaintiffs do not challenge the following ultimate findings/conclusions and they are therefore binding on appeal:

46. Plaintiff Charles Wilson[], one of the “initial five” plaintiffs, alleges that he also contracted colon cancer as a result of exposure to asbestos at the . . . factory. However, the greater weight of the evidence in view of the entire record shows that colon cancer is an ordinary disease of life to which the public is equally exposed. The greater weight of the evidence in view of the entire record does not show that colon cancer is characteristic of persons engaged in the tire manufacturing industry or that working at the . . . factory placed those who worked there at an increased risk of developing colon cancer.

47. Plaintiff Epps[], one of the “initial five” plaintiffs, alleges that he also contracted tonsil cancer as a result of exposure to asbestos at the . . . factory. However, the greater weight of the evidence in view of the entire record shows that tonsil cancer is an ordinary disease of life to which the public is equally exposed. The greater weight of the evidence in view of the entire record does not show that tonsil cancer is characteristic of persons engaged in the tire manufacturing industry or that working at the . . . factory placed those who worked there at an increased risk of developing tonsil cancer.

Plaintiffs challenge ultimate finding 16, which states:

16. The greater weight of the evidence in view of the entire record shows that plaintiffs were neither exposed to airborne asbestos as a result of damaged pipe insulation in such form and quantity and with such frequency as to cause or significantly contribute to the development of

asbestosis, nor were plaintiffs exposed to the hazards of asbestosis through this method in this employment for 30 days or parts thereof within a seven month consecutive period which proximately augmented the disease process of asbestosis to the slightest degree.

Plaintiffs argue: “In short, the Commission found that [P]laintiffs were not exposed to asbestos in a sufficient amount from damaged pipe insulation to cause asbestosis.

This finding was based upon ‘the entire record.’” Plaintiffs then state:

The Commission also found the exposures from asbestos-containing talc, gaskets and brakes were insufficient to cause disease based upon the entire record. (Findings of Fact 19, 21, 23). The “entire record” consisted of the air sampling and testimony of experts regarding the amount of exposure for each [P]laintiff and the amount necessary to cause disease. As stated herein, Plaintiff[s] find[] that such evidence is not competent. Regardless, the Commission based its opinions on that evidence.

Plaintiffs contest ultimate findings 43 and 45 on the same grounds.<sup>33</sup> We have already addressed Plaintiffs’ argument above—in section IV. B. 4. of this opinion—as well as noting that Plaintiffs failed to challenge the Commission’s “last injurious exposure” determinations made pursuant to N.C.G.S. § 97-57. In addition, we hold that there is sufficient competent record evidence to support the Commission’s ultimate findings.<sup>34</sup> *Culpepper*, 93 N.C. App. at 247, 377 S.E.2d at 780.

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<sup>33</sup> All of these ultimate findings are determinations that Plaintiffs’ alleged injuries did not “arise out of” their employment at the factory. See *Culpepper*, 93 N.C. App. at 247, 377 S.E.2d at 780.

<sup>34</sup> To the extent, if any, that the ultimate findings are, or contain, conclusions of law, Plaintiffs abandoned any challenge to them because they have failed to argue that they are not supported by the findings of fact. In addition, we hold that any conclusions in the common issues section of the bellwether opinions and awards are supported by the findings of fact.

Findings 43 and 45 state in relevant part:

43. . . . . Given the evidence of air contaminant measurements taken at [the] factory, the pathology evidence collected from [deceased Plaintiffs'] lungs, and the scientific and epidemiological literature presented on the subject, the greater weight of the evidence in view of the entire record does not demonstrate a causal connection between asbestosis and employment at the . . . factory.

. . . . .

45. The greater weight of the evidence in view of the entire record does not show that [P]laintiffs, through their employment at [the] factory, were exposed to asbestos in such form and quantity and used with such frequency as to cause or significantly contribute to the development of asbestosis[.]

Because both ultimate findings 43 and 45 include determinations that Plaintiffs failed to meet their burden of proving a sufficient causal relationship between employment at the factory and their alleged asbestosis, these ultimate findings defeat Consolidated Plaintiffs' asbestosis claims. Because the Commission determined in "findings" 46 and 47 that Plaintiffs have failed to prove that either colon cancer or tonsil cancer are "occupational diseases" as defined by N.C.G.S. § 97-53(13), these determinations also apply to the outstanding consolidated cases.

*E. Conclusion—Common Issues Arguments*

We affirm the Commission's common issues determinations. It did not err in: (1) Determining Plaintiffs failed to prove a causal connection between employment at the factory and asbestosis; (2) its determination, based upon the facts presented, that

Plaintiffs failed to prove that either colon cancer or tonsil cancer were occupational diseases pursuant to N.C.G.S. § 97-53(13); or (3) its unchallenged determination that Plaintiffs were not last injuriously exposed to the hazards of asbestosis at the factory. Further, we hold that the Commission's findings and ultimate findings are supported by competent evidence, and its conclusions and rulings are supported by the findings.

#### V. Plaintiff Hinson's Appeal

Although we affirm the Commission's opinion and award based on our holdings set forth above, we will also address the findings and conclusions specific to Plaintiff Hinson. Initially, Plaintiff Hinson does not make any argument that the findings of fact fail to support the conclusions of law; therefore, the conclusions of law stand. *Penegar*, \_\_ N.C. App. at \_\_, 815 S.E.2d at 394. For example, Plaintiff argues that, in support of conclusion of law 3, "the Commission . . . relied, in large part, on a determination of the amount of asbestos that [P]laintiff inhaled and how much was necessary to cause disease." Conclusion 3 states in part: "[I]n this case, it was not established, by the preponderance of the evidence in view of the entire record, that [Plaintiff] contracted asbestosis or any asbestos-related condition." Plaintiff argues this conclusion is not supported by competent evidence, but does not make an argument that the *findings of fact* fail to support this conclusion—therefore, this conclusion of law stands. *Id.*

Assuming, *arguendo*, Plaintiff Hinson has preserved challenge to the findings and conclusions specific to him, we hold that competent evidence supports the



relevant findings of fact and ultimate findings, which support the Commission's relevant conclusions of law. *Id.* Plaintiff Hinson does not challenge finding 34 which states in part that the “post-mortem pathological study of” Plaintiff Hinson’s “lung tissue” revealed no “pathological evidence of asbestosis. Pathology is the most reliable method to diagnose asbestosis.” Findings 55, 57, 58, 60, 61, 62, and 63 are also unchallenged. These findings include determinations by multiple doctors that Plaintiff Hinson’s x-rays did not show evidence of pleural abnormalities or asbestosis; that after Plaintiff Hinson told his treating physician of his asbestosis diagnosis, his physician told Plaintiff Hinson “to inform his attorney that the abnormal x-ray was due to pneumonia”; and that though Plaintiff Hinson “testified vehemently that he never smoked,” medical records and his pathology results indicated he had “a remote smoking history.”

The Commission found in findings 37 and 64 that during pathological examination of Plaintiff Hinson’s lung tissue, “Dr. Hart found no evidence of asbestos bodies or fibrosis, but did see evidence of smoking[,]” a conclusion “confirmed” by Dr. Oury, who also conducted a fiber count analysis and found results “well below that seen in individuals with asbestosis and in the range of control individuals with no history of asbestos exposure.” Plaintiff Hinson’s only argument concerning these findings is that the Commission “ignore[d] the pathology findings of [P]laintiff’s experts.” As noted above, Plaintiffs did not present any evidence from pathologists. To the extent Plaintiffs—including Plaintiff Hinson—mean to include non-

pathologist medical doctors, or non-physician scientists who work with lung tissue, under the definition of “pathologists,” it was the province of the Commission to weigh the evidence and the credibility of the witnesses. Because there is some competent evidence to support these findings, they are conclusive on appeal. Plaintiff challenges finding 65, in which the Commission states that Defendant’s medical experts were “given greater weight than” Plaintiffs’. Plaintiff Hinson’s challenge to this finding is based on Plaintiffs’ rejected “entire record,” “air sampling,” and “fiber year theory” arguments.

Our review of the record demonstrates that the evidence supports the ultimate finding—including in finding of fact 66 and conclusions of law 2 and 4—that Plaintiff Hinson failed to prove a causal connection between his employment at the factory and his alleged asbestosis. We further hold that the Commission’s findings, which are based on substantial competent evidence, support conclusion 3, in which the Commission determined that Plaintiff Hinson failed to prove he had asbestosis. Finally, Plaintiff Hinson does not challenge the determination made pursuant to N.C.G.S. § 97-57 that he was not “last injuriously exposed” to the hazards of asbestosis at the factory. For all the above reasons, we affirm the denial of Plaintiff Hinson’s claim.

AFFIRMED.

Judges DIETZ and COLLINS concur.