Vanni v. Honeywell Int'l

Court of Appeal of California, Second Appellate District, Division Eight

September 13, 2021, Opinion Filed

B299594

Reporter
2021 Cal. App. Unpub. LEXIS 5803 *

BARBARA VANNI et al., Plaintiffs and Respondents, v. HONEYWELL INTERNATIONAL, INC., Defendant and Appellant.

Core Terms

asbestos, mesothelioma, balls, pericardial, drilling, exposure, bowling, studies, disease, fiber, chrysotile, exposure to ***asbestos***, ***asbestos*** exposure, cases, air, exposed, ***asbestos*** fiber, causation, percent, registry, patient, tissue, testing, dust, epidemiological, plastic, causes, opined, inhalation, breathing

Opinion

[*1] APPEAL from a judgment of the Superior Court of Los Angeles County, Michele E. Flurer, Judge. Affirmed.

Horvitz & Levy, Lisa Perrochet, Curt Cutting; McDermott Will & Emery and Alice Wong for Defendant and Appellant.

Waters Kraus & Paul, Michael B. Gurien and Michael P. Connett for Plaintiffs and Respondents.

INTRODUCTION

Honeywell International, Inc. (Honeywell) asks us to reverse the jury verdict and judgment entered in favor of respondents Barbara Vanni and her two sons, Mark and Michael (the Vannis), in the amount of $ 1,970,716. Respondents are the wife and sons of Donald Vanni. Donald Vanni owned and operated a bowling alley for 30 years with his brother Fred. The jury was asked to decide whether Donald Vanni's death by pericardial mesothelioma was caused by his exposure to ***asbestos*** from drilling bowling balls that contained ***asbestos*** filler manufactured and supplied by Honeywell's predecessor, the Bendix Corporation (Bendix).

The two issues on appeal are whether the verdict rests on unfounded speculation that Bendix exposed Vanni to ***asbestos*** and whether the verdict rests on unfounded expert opinions about causation. In other words, did respondents prove that **[*2]** Bendix exposed Donald Vanni to ***asbestos*** and, if so, was his pericardial mesothelioma caused by that exposure to ***asbestos***?

We conclude the evidence is sufficient to support the jury's verdict on both issues and affirm.

PROCEDURAL BACKGROUND

A. *Evidence Code Section 402 Hearing*

In 2012, Donald was diagnosed with pericardial

mesothelioma. He died in June 2013 at age 78. In 2014, respondents filed a complaint alleging two causes of action for negligence and strict product liability against Honeywell and others. After motions for summary judgment were denied, Honeywell filed motions in limine challenging the foundation for and admissibility of the testimony of the Vannis' expert on

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causation.1Honeywell moved to exclude the Vannis' theory of general causation (that inhalation of chrysotile ***asbestos*** can cause pericardial mesothelioma) on the ground that no reliable science supported Dr. Barry Horn's expert opinion. Honeywell also moved to exclude the Vannis' theory of specific causation (that HD-100, Bendix's ***asbestos*** product, caused Donald's pericardial

mesothelioma) on the ground that the Vannis' expert, Dr. Carl Brodkin, relied on unfounded assumptions about how much ***asbestos*** would be released **[*3]** from drilling bowling balls containing HD-100. The court scheduled a hearing on Dr. Horn's proposed opinion testimony. The motion in limine as to Dr.

Brodkin's opinion was denied without a hearing.

On April 8, 2019, the trial court conducted a hearing pursuant to *Evidence Code2section 402*. The purpose of the hearing was to determine whether Dr. Horn should be permitted to testify at trial to his expert opinion that exposure to chrysotile ***asbestos*** causes pericardial mesothelioma.

At the hearing Dr. Horn testified he is a critical care specialist and pulmonologist. He testified mesothelioma is a cancer which develops in serosal tissues. Serosal tissues are the membranes which surround the lung, heart, intestines, and scrotum. These membranes are all exactly the same cells, despite the different locations. Dr. Horn testified he has seen "a lot" of instances where individuals exposed to ***asbestos***, usually by

1 For clarity, we refer to respondents Barbara, Michael, and Mark Vanni as the Vannis. We refer to Donald Vanni as Donald.

2 Undesignated statutory references are to the Evidence

Code.

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occupational exposure, developed pericardial plaques (scarring) right on the heart. He concluded it was inconceivable that an individual **[*4]** would develop a localized plaque without ***asbestos*** actually getting to that site. This must mean that ***asbestos*** get to the pericardium, the tissue surrounding the heart, although how it does so is not entirely clear. He was aware of other diseases, like lung and liver cancer, for which science has determined the causes (tobacco and vinyl chloride, respectively), but not the precise mechanisms.

The literature Dr. Horn reviewed reports that individuals exposed to ***asbestos*** may develop non-malignant disease of the pericardium as well as malignant disease of the pericardium, which is pericardial mesothelioma, Donald's diagnosis. He reviewed case reports published in the literature by clinicians who presented their observations of and experiences with how patients present with symptoms, and how the disease naturally progresses. These case reports differed from epidemiological studies where large groups of individuals are compared and contrasted with control groups. A small portion of the case reports noted the patients had occupational exposure to ***asbestos***. Where there was a history of ***asbestos*** exposure, the clinicians attributed pericardial mesothelioma to ***asbestos*** exposure. Dr. Horn testified **[*5]** there are also case reports where exposure to ***asbestos*** is unknown. He testified that this variant can be explained. One explanation is that, in fact, the patient was not exposed to ***asbestos***. A second explanation is that the patient had already died and investigators had very limited information on whether there was exposure. The third explanation is the authors of the case reports were not sufficiently knowledgeable about how to inquire whether in fact there was exposure, or the

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patient himself had no knowledge that he had been previously exposed. About a third of the case reports he reviewed reported ***asbestos*** exposure.

In addition to case reports about individual patients, there are national registries where investigators try to identify all the cases of mesothelioma in the country. There are tumor registries in Italy, Japan, and Germany. There is nothing equivalent in the United States. A registry reviews the pathology to be sure the diagnosis is correct. It also keeps records. It interviews patients with the disease or patient relatives to determine whether the diagnosed individuals were exposed to ***asbestos***. There are four particular studies he reviewed dated 2010, 2013, 2012, and **[*6]** 1982. In these registry studies, approximately 60 percent of the patients had prior exposure to ***asbestos***, which is not the percentage of exposure in the general population. Dr. Horn relied on the case reports and registry studies in forming his opinion that Donald's pericardial mesothelioma was caused by exposure to ***asbestos***.

Dr. Horn was also familiar with the Helsinki Criteria for Attribution of Diseases to ***Asbestos*** Exposure. Several dozen investigators from around the world who were experts in ***asbestos***-related disease reviewed the literature and came up with consensus statements about ***asbestos*** and asbestosis, ***asbestos*** and mesothelioma, and ***asbestos*** and lung cancer. They concluded ***asbestos*** inhalation affects all serosal membrane surfaces in the body. They concluded that an occupational history of ***asbestos*** exposure combined with a diagnosis of mesothelioma is enough to attribute

the mesothelioma to ***asbestos*** exposure.

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At the *section 402* hearing, Dr. Horn also testified it is not surprising that epidemiological studies do not find pericardial mesothelioma per se even in the most heavily exposed cohorts to ***asbestos***. The largest epidemiological study involved 17,800 ***asbestos*** insulation workers in **[*7]** North America. Nine percent died of mesothelioma. This is an enormous number. None had pericardial mesothelioma. They had pleural (lung) mesothelioma and peritoneal (intestinal) mesothelioma. The World Health Organization (WHO) accumulated 92,000 cases of mesothelioma from various countries around the world and pericardial mesothelioma represented 0.3 percent of the group. That means it would occur in three of 1000 people. So if a researcher studied just 400 people with mesothelioma, it would be completely random whether even one person with pericardial mesothelioma would be encountered. If a researcher did not find one person, that would not mean ***asbestos*** does not cause pericardial mesothelioma. One would need a study of multiple times 400 cases of mesothelioma in order to make that determination. That kind of study has never been done. The subject groups are just not big enough to answer the question by using epidemiological studies.

Dr. Horn acknowledged science has not yet discovered why some people get mesothelioma and some do not. In addition people get non-malignant disease in the pericardium more frequently than malignant disease. But malignant disease does occur. He cannot **[*8]** explain the randomness of the disease.

On cross-examination, Dr. Horn stated exposure to ***asbestos*** is the only thing he has seen that explains the pericardial plaque or scarring, which looks exactly like pleural plaque. He generally relies on epidemiological studies to

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determine the etiology of cancer. Although he has done epidemiological studies himself and has been a coauthor, he does not consider himself an epidemiologist. He is unaware of any epidemiologic studies linking Honeywell's ***asbestos***, HD-100, with pericardial mesothelioma or any studies showing that drilling holes in bowling balls increases the risk of pericardial mesothelioma. And all studies but one on auto mechanics exposed to brake dust with ***asbestos*** do not conclude there is an increased risk of pericardial mesothelioma from exposure to brake dust with ***asbestos***.

There are no epidemiological studies or cohort studies focused on the specific disease of pericardial mesothelioma. There are only registry studies and case reports. The national registry studies do not compare an identical control group with a population. The national studies just look at the total number of cases in the country. In the case reports it has been **[*9]** stated multiple times that a potential association between pericardial mesothelioma and exposure to chrysotile ***asbestos*** specifically (as opposed to exposure to ***asbestos*** generally) has not been established.

There are insufficient data to believe another type of ***asbestos***, amphiboles fibers, are more potent than chrysotile fibers as a cause of pericardial mesothelioma. There are no data showing different types of fibers have the same potency for causing pericardial mesothelioma. But the serosal tissues throughout the body are the same, so Dr. Horn concludes the tissue, no matter its location, responds in the same manner to ***asbestos***. There is a dose-dependent relationship between development of ***asbestos***-related malignancies. There is a general dose-dependent relationship with all fiber types. Specific data as

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to pericardial mesothelioma are not published because there are not enough cases.

Of the 48 registry studies for pericardial mesotheliomas where an effort was made to gather a work history, 30 found ***asbestos*** exposure. The type of ***asbestos*** was not recorded. Because the tissue is the same, Dr. Horn did not agree that the type of ***asbestos*** is significant in determining the cause of pericardial **[*10]** mesothelioma. If ***asbestos*** affects the serosal tissue in the lungs, it would affect the same tissue elsewhere in the body. The 30 cases of exposure did not state the intensity, duration, or frequency of exposure.

To Dr. Horn, exposure to ***asbestos*** in 30 out of 48 patients established a cause and effect relationship, that is, exposure to ***asbestos*** causes pericardial mesothelioma.

Dr. Horn testified no one in the State of California has seen and diagnosed more people with ***asbestos*** exposure and ***asbestos***-related disease than he. The registry studies are reliable because they are done by "really good investigators who understand what we're

talking about here. That is, they're looking at rare disease and looking to determine whether there's a relationship with ***asbestos*** exposure. We're talking about people who have published multiple papers in the world's literature, particularly, the group from Italy which represents the bulk of these cases that have been published. These are careful. These are knowledgeable investigators." He has been reading literature for

50 years since 1956 and is perfectly competent to interpret it. According to multiple studies he has reviewed, chrysotile ***asbestos*** causes **[*11]** mesothelioma.

Pericardial mesothelioma is this rare: In the United States there are about 2,500 cases per year of mesothelioma. The data

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from WHO shows 0.3 percent of mesotheliomas are pericardial mesothelioma. This amounts to eight cases in the United States per year. Dr. Horn could not give the likelihood of someone in the general population just developing pericardial mesothelioma without exposure to ***asbestos***. He estimates it would be some fraction of the eight.

After listening to the testimony at the *section 402* hearing, the court summarized the evidence: "So, because - I'm summarizing. Because chrysotile causes meso, we don't know the exact mechanism of pericardial meso. All those workers were exposed to ***asbestos***. They had pericardial mesothelioma and on that basis you believe this causal connection exists." Dr. Horn answered, "I do, and so do much of the people who addressed this issue around the world do as well, as evidenced by the Helsinki criteria published about 20 years ago."

The trial court noted that "[i]f the opinion is based on materials in which the expert may reasonably rely in forming the opinion and flows in a reasoned chain of logic from those materials rather than speculation **[*12]** or conjecture, the opinion may pass even though the experts disagree with its conclusions or the methods and materials used to reach that opinion." The court acknowledged that defense experts had contrary opinions. "I think it will definitely go [to the] issue of weight versus admissibility. [¶] The court concludes that the matters upon which Dr. Horn relies to support his opinion on whether or not exposure was a substantial factor in causing the plaintiff is reasonably of the type that may be relied on in forming such opinions. In other words, it provides a reasonable basis for the particular opinion offered, and it is not based on mere speculation

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or conjecture. Therefore, the court denies the motion of Honeywell to exclude the testimony of Dr. Horn."

B. *Trial and Verdict*

The trial was essentially a battle of the experts. Drs. Steven Compton, Carl Brodkin, and Barry Horn testified on behalf of the Vannis. Dr. James Crapo, Dr. Suresh Moolgavkar, Sheldon Rabinovitz, and Renee Kalme testified on behalf of Honeywell. The jury found in favor of the Vannis on their negligence and product liability causes of action and found

Bendix was a substantial factor in contributing to Donald's risk **[*13]** of mesothelioma. It awarded $397,716 in economic damages and $4 million in noneconomic damages. There were other defendants. The jury assigned 40 percent fault to Honeywell, 37 percent fault to Ebonite, and 23 percent fault to Ebonite's other ***asbestos*** suppliers. It found that Honeywell did not act with malice, oppression, or fraud.

The trial court entered judgment against Honeywell in the amount of $1,970,716, reflecting the jury's allocation of fault and offsets for the Vannis' settlements with other defendants. The court awarded an additional $101,500 in costs.

EVIDENCE AT TRIAL

A. The Drilling

In 1957, 22-year-old Donald and his brother Fred Vanni

opened the Arcata Bowl bowling alley, which they operated until October 1986. They shared bartending, bookkeeping, cooking, oiling the lanes and repairing the pin-setting machines. They started the bowling alley with 60 rubber bowling balls with pre-drilled finger holes. Plastic bowling balls became popular in the

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mid-1960's and were the predominant bowling balls by the late 1960's. In 1962, Donald and Fred purchased a ball-drilling machine. Only Donald began drilling finger holes in plastic balls in a four by eight foot unventilated "ball-drilling **[*14]** room." At a minimum it took Donald about 30 minutes to drill the holes and he routinely

drilled five to 10 balls a week, for a total weekly drilling time of two and one-half to five hours. He also used a hand file and sandpaper to file, sand, and smooth out the rough edges of the drilled holes. Donald did not wear a mask or breathing protection when he drilled and he breathed dust from the balls because he was "right over the top of" them when he was drilling. Donald would remove the dust from the bowling ball by turning the ball over and blowing it off the top. After he was done drilling, Donald would sweep and clean up the dust and inner-ball material, causing the air to become dusty.

There were three "equally popular" brands of bowling balls that Donald drilled: Ebonite, AMF and Brunswick. Ebonite became the "premier brand" by the late 1960's.

*B. The **Asbestos** in the Bowling Balls*

The use of ***asbestos*** in Ebonite's bowling balls was not established with precision. Ebonite had destroyed its records. According to one former Ebonite employee, at least from 1967 on, Ebonite used a combination of various materials in the core of plastic bowling balls, including resin, peroxide, barytes, styrene **[*15]** and ***asbestos***. Not all the materials went into every ball. The evidence at trial did not definitively establish that ***asbestos*** was included in all balls.

However, Bendix, Honeywell's predecessor, did have records. Bendix made automotive friction products, including brakes. Its friction products had an average ***asbestos*** content of

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50 percent by weight. Bendix used chrysotile ***asbestos***, which is the least toxic of the several types of ***asbestos***. The dust was called HD-100. Between 1967 and 1972 Bendix supplied at least 232,000 pounds of its HD-100 to Ebonite at its bowling ball manufacturing facility in Hopkinsville, Kentucky. Ebonite used the HD-100 as filler in its plastic bowling balls.

*C. Donald's Level of Exposure to **Asbestos***

Dr. Steven Compton is a physicist and materials scientist with ***asbestos***-testing expertise. He was asked to find out how much ***asbestos*** was in a product so that another scientist who specializes in the effects of ***asbestos*** in the body could assess the safety of the product.

Dr. Compton explained that chrysotile is a mineral that forms in the earth. Fibers protruding from the surface are ***asbestos*** fibers that can be mined, processed, and incorporated into a product. Chrysotile **[*16]** is the predominate form of ***asbestos*** used in commercial products. Historically, over 90 percent of commercial products use chrysotile ***asbestos***.

Asbestos is a mineral that is heat resistant and durable. On the other hand, it forms long thin fibers which can be woven into cloth in ways not possible with most other minerals. Each ***asbestos*** fiber is a micrometer, that is, a millimeter broken up into a thousand equal parts. When ***asbestos*** fibers are released into the air, they can remain suspended in the air for a number of hours, depending on conditions. Eventually they will settle onto various surfaces, like a lunch box or an individual's clothing, hair, or skin. They can be resuspended into the air if something causes them to become airborne again, like sweeping the floor or shaking clothing. They can stay in the air for hours. ***Asbestos***

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does not degrade so it can be suspended and resuspended without limit.

Honeywell obtained 26 Ebonite bowling balls manufactured in the 1970's before 1979. Dr. Compton tested nine of those bowling balls to determine ***asbestos*** content and the level of fiber release. Six of the balls were from before 1979 and three were manufactured after that date. The balls **[*17]** manufactured after 1979 contained no ***asbestos***. A bowling ball had two layers - an inside core and an outside veneer or cover. For plastic balls, the veneer is made from plastic.

Dr. Compton collected a sample of the inside materials by drilling and then analyzing the material using microscopes. A certified industrial hygienist drilled the balls. After he collected the inner material, he prepared it to be placed onto a glass microscope. They got rid of the non-***asbestos*** materials that might be present by exposing the material to heat and acid. That isolated the material that might be ***asbestos***. Then he analyzed the isolated material. Dr. Compton found that all six Ebonite bowling balls made before 1979 contained chrysotile ***asbestos*** ranging from 6 percent to 9 percent by weight.

Dr. Compton also filed the edge of the cover to make sure it did not have a sharp edge. He sanded the interior of the finger hole to smooth out the surface. Dust was created by the sanding activity. The last phase was sweeping the dust and debris off the floor.

Dr. Compton also tested the ***asbestos*** fiber release by drilling the balls. He opined that drilling finger holes released breathable ***asbestos*** fibers from 0.06 **[*18]** to 0.17 fibers per cubic centimeter of air, 10,000 times greater than the background level of ***asbestos*** fibers present in the ambient air everywhere. He

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testified that additional filing, sanding, and sweeping also resulted in ***asbestos*** in the air well above background levels. Dr. Compton concluded and opined that the balls manufactured before 1979 all contained chrysotile ***asbestos*** in quantifiable and visual amounts. He also concluded that the

activities of drilling, filing, sanding, and sweeping released fibers that exceed ambient background air levels. He concluded that someone drilling Ebonite balls from the 1970's would generate ***asbestos*** dust into the breathing space, no matter how much time the actual drilling took.

Dr. Compton was critical of Honeywell's testing. He noted Honeywell's experts never drilled an ***asbestos***-containing ball in a way that was represented by the facts of the case. When they vacuumed, they filtered the dust. They did not use a sophisticated microscope to precisely capture all the ***asbestos*** in the samples. And they did not get rid of the non-***asbestos*** material before they tried to quantify the amount of ***asbestos*** in the samples.

D. Causation of Mesothelioma

Dr. Carl **[*19]** Brodkin is an occupational and environmental

medical doctor with more than 30 years of experience in diagnosis, causation, and treatment of ***asbestos***-related diseases. Occupational medicine deals with the diagnosis or identification of disease and the management and treatment of disease, but it also deals with exposure-related illness, i.e., did an exposure cause an illness. To diagnose an ***asbestos***-related disease that may develop many years after exposure requires a systematic review of a patient's work history or else the researcher may not appreciate that it is an ***asbestos***-related disease.

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In 2015, Dr. Brodkin was asked to do a medical evaluation of Donald, who had died in 2013. He had to construct an occupational history without Donald's participation. He reviewed pathology reports and Donald's medical history and estimated Donald's exposure to ***asbestos***. Once he received Dr. Compton's actual testing and assessment, he reviewed it to reevaluate his initial opinion about what exposures Donald likely experienced.

Dr. Compton's testing was more specific and gave "greater resolution" and a more accurate basis for his own exposure calculations. Dr. Compton's numbers did not change his **[*20]** overall opinion. Dr. Brodkin also reviewed literature that dealt with similar activities, like drilling and sanding plastic resin materials similar to bowling balls.

Dr. Brodkin testified that ***asbestos*** is the preeminent cause of mesothelioma and that the more a person is exposed, the greater the risk of developing the disease. ***Asbestos*** has a tendency to break from larger bundles into smaller and smaller fibers. A fiber is typically five times longer than it is wide. If you put a thumb and forefinger as close as you can and see some air, that space is a millimeter. A fiber is one-thousandth of a millimeter. It is not visible to the naked eye. The fibers gain access to the lung. Chrysotile fiber is the most common type used in North America and Europe. It is about 95 percent of the ***asbestos*** that are used. He testified all types of ***asbestos*** fibers cause mesothelioma, including chrysotile, the type in the Ebonite bowling balls. Exposure to low levels of ***asbestos*** increases the risk of mesothelioma and no threshold has been found below which mesothelioma cannot occur from ***asbestos*** exposure.

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Dr. Brodkin explained that mesothelial tissue (or serosal membrane) is found in the lung and heart **[*21]** and it is the same tissue wherever it is located in the body. Once ***asbestos*** enters the body, it migrates via the lymphatic system to all mesothelial tissues in the body. The movement of ***asbestos***, called kinetics, allows the fibers to move throughout the body. The mesothelial tissue is like cellophane and an ***asbestos*** fiber is like a splinter which, if not removed, will scar over. ***Asbestos*** fibers cause scarring or plaques and cause inflammation and fluid to accumulate. Plaques develop randomly and unpredictably. In about 70 percent of cases of mesothelioma overall, there is no evidence of plaques.

Dr. Brodkin noted ***asbestos*** is considered a known human carcinogen because it damages DNA or genetic

material which can affect cell growth and it causes inflammation which over times causes cells to behave in an abnormal way. The cancer cells replicate and divide quickly. The agencies that study ***asbestos*** and mesothelioma do not make a distinction between different fiber types because all fiber types cause all the ***asbestos*** diseases. When he takes an occupational history, he is not interested in whether someone was exposed to chrysotile or amosite or crocidolite ***asbestos***. He is just looking for **[*22]** exposure to ***asbestos***. All types of ***asbestos*** can travel to all the different sites where mesothelioma can occur. Only research labs, not clinical labs, are specialized enough to discover mesothelioma in locations other than the lung. Dr. Brodkin testified he is unaware of any agency in the world that has concluded that not all ***asbestos*** fibers cause mesothelioma.

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With Donald's case, Dr. Brodkin had to find a well-characterized source of ***asbestos*** and then a well-characterized activity that disrupted the source. ***Asbestos*** has to be disturbed so that the fibers become airborne at a concentration that is sufficient to be breathed into the body. He also looked for direct as opposed to indirect exposure. This is the difference between the worker actually disturbing the ***asbestos*** through a task being done and someone infected by contaminated clothing brought home or by breathing in air someone else's activity has contaminated. He reiterated that no government or health agencies have identified a "safe level" of ***asbestos*** because a threshold has not been identified to date.

Dr. Brodkin noted there were four large national epidemiological studies in Lombardy, Italy, Japan, and Germany that investigated **[*23]** occupational history and pericardial mesothelioma and they all consistently showed a strong association between ***asbestos*** exposure and pericardial mesothelioma. He testified that pericardial mesothelioma represents less than 1 percent of mesotheliomas. Because of its extreme rarity, "you need a large national study with thousands of cases of mesothelioma" to find pericardial mesothelioma. As a result, he did not think it was significant that some studies reported no pericardial mesothelioma among the subject groups because the studies were not large enough to capture the small percentage of cases.

Dr. Brodkin also noted that case reports documenting a history of occupational exposure to ***asbestos*** attribute pericardial mesothelioma to that exposure. Case reports lacking information about a known history of ***asbestos*** exposure cannot make that connection. Getting a known history of ***asbestos*** exposure is

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difficult because often the diagnosed patient has already died, or the patient and his family is unaware whether the patient was exposed. However, the large registry studies which were "well designed to take an occupational history" showed a strong association between pericardial mesothelioma **[*24]** and ***asbestos*** exposure. He also noted that the Helsinki Criteria concluded that ***asbestos*** caused mesothelioma disease in any mesothelial or serosal tissue, whether it be in the lungs, heart, intestine, or testicles. The location was irrelevant.

Relying on Dr. Compton's testing and Bendix's own supply paperwork, Dr. Brodkin concluded Donald had sufficient exposure to ***asbestos*** from drilling the Ebonite bowling balls. His occupational history suggested no other possible source of ***asbestos***. Donald had a well-characterized source of ***asbestos*** (the bowling balls) and participated in an activity (drilling, sweeping, filing and sanding) that sufficiently disturbed the fibers so that they became airborne in a concentration sufficient to be breathed into the body. He opined that the Bendix ***asbestos*** filler in the

Ebonite balls was a substantial contributing factor to Donald's development of mesothelioma to a reasonable degree of medical certainty.

Donald was exposed to ***asbestos*** over an 11-year period between 1968 and 1978 when he was drilling bowling balls. The Ebonite brand of balls contained ***asbestos*** between 1968 and 1972 when it used Bendix brake dust as filler for the balls. Using the estimate **[*25]** of five drilled balls per week, Dr. Brodkin estimated that Donald drilled 100 to 200 bowling balls between 1968 and 1972. He opined that the drilling exposed Donald to ***asbestos***.

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Donald drilled two inches deep which is one and one-half inches into the ***asbestos***-containing core. That generated a route of exposure because his face was about one foot from the drilling site. The second activity was sanding and filing. The third activity was cleanup, which was a dusty operation. Dust settled on the ground and Donald swept it up with a vacuum and dumped the contents of the vacuum. Dr. Brodkin found a source of

exposure and activity that generated significant airborne exposures during the 1968 through 1972 timeframe and beyond to 1978. Sweeping resuspended the particles in the air, which increased the duration of the exposure. Donald did not use any mitigation measures like a mask, a respirator, an isolated separate space, or wetting of the material, all of which have been recommended in the United States since 1935.

Dr. Brodkin explained that based on Dr. Compton's testing, Donald generated .06 to .17 fiber per cubic centimeter (cc). A cc is the size of a sugar cube of air. He opined that **[*26]** the concentration of ***asbestos*** in ambient air is so low that one would have to breathe in 500,000 sugar cubes of air to inhale a first ***asbestos*** fibers. We take in 500 sugar cubes of air with each breath. It would then take 1000 breaths to get the first fiber of ***asbestos***, which is very low and has not been associated with any disease. With the air generated by the bowling ball drilling, one would inhale the first ***asbestos*** fiber after only 16 sugar cubes, instead of 500,000. At the upper end, one would only need to inhale five sugar cubes of air to get to the first fiber. Dr. Brodkin calculated that the magnitude of difference was 30,000 to 85,000 times the ambient level. He concluded this high concentration increased the risk for ***asbestos***-related disease like mesothelioma. Dr. Brodkin found that Donald was generating and breathing

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these fibers for four years for Bendix ***asbestos*** and another six years beyond that for other ***asbestos*** suppliers. His opinion was this high intensity and long duration of exposure increased

Donald's risk of mesothelioma.

Finally, Dr. Brodkin testified that he relies on the Helsinki Criteria in his practice to diagnose an ***asbestos***-related mesothelioma. The Criteria **[*27]** applies to any mesothelial or serosal membrane in any location and does not distinguish between types of ***asbestos***. All the fiber types are potent so no distinction is made by clinicians, the Environmental Protection Agency, Occupational Safety and Health Administration, Centers for Disease Control, National Institute for Occupational Safety & Health, or the American Thoracic Society. The Helsinki Criteria also sets out a median latency period of 30 to 57 years and a minimum mesothelioma-free period of 10 years after exposure. In Dr. Brodkin's opinion, Donald fit the Criteria in that he had exposure, a diagnosis, an appropriate latency period of 44 years from 1968 to 2012, and no known differential risk factor other than exposure to ***asbestos***. He agreed with Dr. Horn that large epidemiological studies are still not large enough to capture this rare disease, so he also relies on case reports and national registry studies. He is unaware of any case of pericardial mesothelioma with a known history of ***asbestos*** exposure where the clinician did not attribute the cause to ***asbestos***. As for chrysotile ***asbestos***, Dr. Brodkin testified that in those parts of the world where chrysotile is made into **[*28]** products (brakes and textiles), there is a 400 to 3,000 percent increased rate of mesothelioma, which demonstrates a strong association between chrysotile and increased risk of mesothelioma. Animal studies

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show a 25 to 75 percent rate of mesothelioma in animals injected with chrysotile.

The third Vanni expert was Dr. Barry Horn who also gave an opinion on the cause of Donald's pericardial mesothelioma.

He acknowledged that the disease is always fatal and extremely rare. The only known cause of mesothelioma in men in the United States is prior exposure to ***asbestos***, including chrysotile ***asbestos*** which causes all forms of mesothelioma. He concurred with Dr. Brodkin that there is no established minimum exposure threshold below which mesothelioma cannot occur and that it takes very little exposure to ***asbestos*** to cause the disease. He also concurred with Dr. Brodkin that the disease is the same in all mesothelial or serosal tissue, regardless of the location of the tissue in the body.

Dr. Horn has seen patients with plaques or localized scarring on the mesothelial tissue in the pericardium from prior exposure to ***asbestos***. He opined that ***asbestos*** can reach the pericardium and flatly disagreed **[*29]** with anyone who did not hold the same opinion. He had reviewed the four large national registry studies which showed that approximately 60 percent of those with pericardial mesothelioma had known prior ***asbestos*** exposure. He basically repeated the testimony he gave at the Evidence Code *section 402* hearing, including his testimony that the case reports which noted an occupational exposure to ***asbestos*** all attribute pericardial mesothelioma to the ***asbestos*** exposure. He went on to note that if a case report did not note exposure, it was probably written by a doctor who was not an expert in taking occupational exposure histories for the purpose of identifying ***asbestos*** exposure. That was why the registry studies were very

significant, in his opinion, because they were

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compiled by people who were experienced and knowledgeable in taking occupational exposure histories.

Based on Dr. Compton's testing, Donald's history of drilling the Ebonite bowling balls, and Donald's medical records, Dr.

Horn opined that Donald was exposed to ***asbestos*** from drilling bowling balls that had ***asbestos*** at the core and that he developed pericardial mesothelioma due to that occupational exposure.

The defense presented four expert **[*30]** witnesses to counter the Vanni expert testimony. First up was Renee Kalmes, a certified industrial hygienist. Her employer, Exponent, had searched Craigslist and eBay for Ebonite bowling balls made between 1967 and 1990. They acquired over 20, all of which were manufactured in 1973 or later. None of the rubber Ebonite balls contained ***asbestos***. The six plastic Ebonite balls made between 1973 and 1978 contained some amounts of ***asbestos***. In eight other plastic Ebonite balls she tested, she found no ***asbestos***.

Next was Sheldon Rabinovitz, a certified industrial hygienist and toxicologist. He calculated the total dose of ***asbestos*** that Donald could have experienced from Ebonite bowling balls containing HD-100. He calculated Donald would have been exposed to only .004 fibers per cubic centimeter per day and over the course of four years he would have had

.016 "fiber years" of exposure. By comparison the average person experiences .002 fiber years of exposure in very rural areas to

.1 fiber years of exposure in very industrialized areas. Dr. James Crapo, a medical doctor specializing in

pulmonary medicine, testified on both general and specific causation. Most cancers occur spontaneously, not as **[*31]** a result of exposure to any substance. He opined both that pericardial mesothelioma results only from spontaneous malignancy without

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any identified external cause and that ***asbestos*** causes all types of mesothelioma. He qualified his opinion by adding that pericardial mesothelioma is associated with amphibole and not with chrysotile ***asbestos*** because chrysotile does not have the durability to reach the pericardium. Then he opined there is insufficient evidence that inhalation of any type of ***asbestos*** fibers causes pericardial mesothelioma and that researchers did not find any cases of pericardial mesothelioma when studying groups of workers who were highly exposed to ***asbestos***. He also testified that there is no reasonable pathway for inhaled ***asbestos*** fibers to reach the pericardium and no evidence that such migration actually occurs. No one studying pericardial mesothelioma has ever found an ***asbestos*** fiber there. He also stated that pericardial plaques are not evidence of ***asbestos*** exposure because they develop whenever there is an infection or trauma to the area.

As for Donald's pericardial mesothelioma, Dr. Crapo opined it was not caused by exposure to chrysotile ***asbestos***; it was a spontaneous **[*32]** malignancy without any external known or identified cause, other than bad luck. He based his opinion on the dearth of sufficient evidence in the medical literature demonstrating an actual cause of this type of mesothelioma.

As to specific causation, Dr. Crapo opined that Donald's cumulative exposure to Bendix's HD-100 would be less than his lifetime exposure to background levels of ***asbestos*** and would not be sufficient to cause the disease. He also expressed his opinion that case reports cannot support an inference of causation because they do not include control groups. They simply report incidences of disease and exposure to toxic substances.

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Finally, Dr. Suresh Moolgavkar, a medical doctor and Ph.D. who conducts research on disease causation, testified. He also opined that most cases of cancer arise due to random cell mutation, not carcinogens in the environment. He acknowledged, however, that pleural mesothelioma is strongly associated with ***asbestos*** exposure in that 80 percent of all cases of pleural mesothelioma in men are attributable to high doses of amphibole ***asbestos***.

Dr. Moolgavkar dismissed case reports because they are just one physician's observations of one patient's disease. **[*33]**

Similarly, registry studies report on a number of cases of a specific disease but are useless with respect to causation without a proper epidemiological study. Dr.

Moolgavkar published a study in which he reviewed 237 papers on mesothelioma in the pericardium and scrotum and concluded there was absolutely no evidence that inhalation increased the risk of the disease. He testified that in one study of over 30,000 heavily exposed ***asbestos*** workers, one would expect to see between eight and 16 cases of pericardial mesothelioma, but in fact no cases were reported.

DISCUSSION

A. *Applicable Law*

Honeywell challenges the sufficiency of the evidence to

support the verdict. We review the sufficiency of the evidence under the substantial evidence standard of review. (*Izell v.Union Carbide Corp. (2014) 231 Cal.App.4th 962, 969*.) Under that standard, we consider all the evidence in the light most favorable to the prevailing party, giving it the benefit of every reasonable inference and resolving conflicts in support of the

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judgment. (*Howard v. Owens Corning (1999) 72 Cal.App.4th 621, 630*).

In an ***asbestos***-related injury case, causation involves two elements: exposure and substantial factor causation. A plaintiff

"may prove causation . . . by demonstrating that the plaintiff's exposure to defendant's ***asbestos***-containing **[*34]** product in reasonable medical probability was a substantial factor in contributing to the aggregate *dose* of ***asbestos*** the plaintiff or decedent inhaled or ingested, and hence to the *risk* of developing ***asbestos***-related cancer." (*Rutherford v. Owens-Illinois, Inc.*

(1997) 16 Cal.4th 953, 976-977, fn. omitted (*Rutherford*).) The contribution of the individual cause need only be "more than negligible or theoretical." (*Id. at p. 978*.) "Undue emphasis should not be placed on the term 'substantial.' " (*Id. at p. 969*.)

Expert testimony that is based on factors that are speculative or conjectural does not constitute substantial evidence. (*Sargon Enterprises, Inc. v. University of SouthernCalifornia (2012) 55 Cal.4th 747, 771-772*; *Lockheed Martin Corp v. Superior Court (2003) 29 Cal.4th 1096, 1110*.) Because it is not the trial court's role to resolve scientific controversies, the jury must resolve conflicts between competing expert opinions. (*Sargon, at p. 772*; *Rutherford, supra, 16 Cal.4th at p. 984*.)

B. *Substantial Evidence Supports the Jury's Verdict that*

Donald was Exposed to ***Asbestos***

Honeywell argues the evidence was insufficient to establish

that Donald was exposed to the Bendix HD-100 ***asbestos***. It accurately cites *Collin v. CalPortland Co. (2014) 228 Cal.App.4th 582, 589 and LAOSD* ***Asbestos*** *Cases (2020) 44 Cal.App.5th 475, 488* for the proposition that if a plaintiff fails to prove exposure, there is no causation and no liability as a matter of law.

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Honeywell argues that the Vannis failed to carry their burden because at most they raised a "mere possibility" **[*35]** of exposure to ***asbestos***.

We disagree. The evidence was irrefutable that Bendix supplied its ***asbestos*** dust, HD-100, to Ebonite over at least a four-year period from 1968 to 1972 and that Ebonite used the HD-100 as an ingredient to fill its plastic bowling balls. Bendix's own paperwork established the supply chain. The testing experts, Dr. Compton for the Vannis and Renee Kalme for Honeywell, agreed that the tested Ebonite balls from the 1970's contained varying levels of ***asbestos*** dust. While Ebonite's former employee William Duncan testified that Ebonite had different formulas for its bowling ball filler, he confirmed that ***asbestos*** was one of the ingredients in the filler from the late 1960's to 1978 or 1979.

The evidence was also undisputed that Donald drilled the bowling balls in a small unventilated space for two and one-half to five hours per week with no mask or other breathing protection and that he did so while standing directly in front of and very close to the ball itself.

As set out above, the bowling ball testing by Dr. Compton established that Donald inhaled 10,000 times the background ambient air ***asbestos*** level through the drilling, sanding, and filing during this period. **[*36]** In addition, he inhaled more fibers through the sweeping and cleanup process. We conclude this is more than negligible or theoretical exposure to ***asbestos***.

That the Vannis could not present actual balls drilled by

Donald at his bowling alley to prove exposure is not dispositive. The jury was allowed to infer that the Ebonite bowling balls from

Donald's relevant occupational time period, randomly collected by

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Honeywell, tested by both experts and found to contain ***asbestos***, were similar to the Ebonite bowling balls Donald actually drilled which exposed him to ***asbestos*** fibers. This inference is further supported by the Bendix documents in 1970 touting that its HD-

100 made "excellent filler in molding the inner core of bowling balls."

In sum, we find the evidence at trial supports a finding that Ebonite plastic bowling balls were manufactured with some level of ***asbestos*** from the mid-to-late 1960's to 1978; Ebonite used the Bendix HD-100 ***asbestos*** product to fill its balls from 1968 to 1972; and Donald's drilling of the balls exposed him to ***asbestos*** fibers. This was not a case of negligible or theoretical exposure.

Honeywell relies on *Berg v. Colgate-Palmolive Co. (2019) 42 Cal.App.5th 630* as support for its argument that the evidence of exposure **[*37]** is insufficient. In *Berg*, plaintiff alleged he was exposed to ***asbestos*** from 1959 to 1962 when he used defendant's talc product, which was allegedly contaminated by ***asbestos***. The trial court granted summary judgment on the ground that plaintiff could not prove exposure because the products he actually tested were not proven to be from the relevant time period, a particularly important fact because the ***asbestos*** were a contaminant, not an intended ingredient. (*Id. at pp. 632-633, 636-637*.) *Berg* is inapplicable where, as here, the ***asbestos*** in Ebonite's bowling balls was an intended ingredient and the tested balls were manufactured in the relevant time period. The Vannis proved at trial that ***asbestos*** was among the ingredients of the filler material in Ebonite's plastic bowling balls from the mid-to-late 1960's to 1978; Bendix supplied more than 232,000 pounds of HD-100 to Ebonite from 1967 to 1972; Ebonite used the HD-100 filler in the core of its bowling balls; and Bendix was

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Ebonite's exclusive supplier of ***asbestos*** filler for its bowling balls from 1968-1972. This is more than negligible evidence that the balls Donald drilled contained ***asbestos*** to which he was exposed by the drilling process.

C. *Substantial **[*38]** Evidence Supports the Jury's Verdict that Donald's Exposure to **Asbestos** Was a Substantial Factor in*

Increasing His Risk for Mesothelioma.

Honeywell next argues that the Vannis' expert opinions on causation are insufficient evidence because they rest on unfounded speculation. Specifically, Honeywell argues 1) many of the articles Dr. Horn and Dr. Brodkin relied upon expressly contradicted their opinions; 2) neither expert relied on epidemiological studies and instead relied on registry studies or case reports or the Helsinki Criteria; 3) studies of the most heavily exposed ***asbestos*** workers report no cases of pericardial mesothelioma; 4) there is no data indicating that pericardial mesothelioma increased in proportion to the use of ***asbestos***.

We reject Honeywell's arguments. First, under the substantial evidence standard of review, we look at the evidence in the light most favorable to the prevailing party, even if there is contradictory evidence in the record. (*Shirvanyan v. Los AngelesCommunity College Dist. (2020) 59 Cal.App.5th 82, 89, fn. 2*.) That there may be articles or passages in articles that arguably contradict the Vannis' expert opinions is not dispositive of the appeal in favor of Honeywell. Dr. Brodkin, for instance, edited a textbook on occupational medicine **[*39]** which included opinions that contradicted his own opinions, observations, and experience of

30 years. He explained reasonably that he edited the textbook so that there would be a full and complete discussion of the medical issues presented in the book. He believed each chapter author

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presented a fair and even discussion of the issues, whether he, as editor agreed with their opinions. Both experts backed up their opinions with relevant literature in the field.

This brings us to Honeywell's second objection that neither

Dr. Horn nor Dr. Brodkin relied on epidemiological studies, but relied instead on case reports, national registry studies, and the Helsinki Criteria. The Helsinki Criteria suffice to support the result here. The Helsinki Criteria for Diagnosis and Attribution is a compendium

of information on mesothelioma and its causation compiled by the world's 20 most prominent scientists and doctors in the field. This consensus report, updated in 2015, reported that ***asbestos*** causes all different forms of mesothelioma.

This expert report is substantial evidence that Donald's pericardial mesothelioma was caused by Honeywell's ***asbestos***.

Honeywell also points out that many large studies of **[*40]** heavily exposed workers do not show that pericardial mesothelioma is caused by ***asbestos*** and, in particular, chrysotile ***asbestos***. In that same vein, Honeywell argues that the absence of studies showing that the risk of pericardial mesothelioma increases as the exposure to ***asbestos*** increases is dispositive in its favor. This, again, ignores the standard of review which requires us to view the evidence in the light most favorable to the Vannis, notwithstanding evidence contradicting the verdict. We find that the Helsinki Criteria, upon which the experts relied and whose significance they explained at length to the jury, constitute substantial evidence that exposure to ***asbestos*** increases the risk of developing this rare type of mesothelioma.

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DISPOSITION

The judgment is affirmed. Respondents are awarded costs on appeal.

NOT TO BE PUBLISHED IN THE OFFICIAL REPORTS

STRATTON, Acting P. J.

We concur:

WILEY, J

OHTA, J.*****

***** Judge of the Los Angeles Superior Court, assigned by the

Chief Justice pursuant to *article VI, section 6 of the California Constitution*.

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