

**FINAL AWARD ALLOWING COMPENSATION**  
(Affirming Award and Decision of Administrative Law Judge)

Injury No.: 05-144489

Employee: Howard Moreland  
Employer: Eagle Picher Technologies, LLC  
Insurer: Self-Insured

The above-entitled workers' compensation case is submitted to the Labor and Industrial Relations Commission (Commission) for review as provided by section 287.480 RSMo. Having reviewed the evidence and considered the whole record, the Commission finds that the award of the administrative law judge is supported by competent and substantial evidence and was made in accordance with the Missouri Workers' Compensation Law. Pursuant to section 286.090 RSMo, the Commission affirms the award and decision of the administrative law judge dated December 28, 2010. The award and decision of Administrative Law Judge Karen Wells Fisher, issued December 28, 2010, is attached and incorporated by this reference.

The Commission further approves and affirms the administrative law judge's allowance of attorney's fee herein as being fair and reasonable.

Any past due compensation shall bear interest as provided by law.

Given at Jefferson City, State of Missouri, this 20<sup>th</sup> day of October 2011.

LABOR AND INDUSTRIAL RELATIONS COMMISSION

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William F. Ringer, Chairman

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Alice A. Bartlett, Member

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VACANT  
Member

Attest:

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Secretary

## AWARD

Employee: Howard Moreland

Injury No. 05-144489

Dependents: N/A

Employer: Eagle Pitcher Technologies, LLC

Before the  
**DIVISION OF WORKERS'  
COMPENSATION**  
Department of Labor and Industrial  
Relations of Missouri  
Jefferson City, Missouri

Additional Party: N/A

Insurer: Self-Insured

Hearing Date: August 20, 2010

Checked by:

### FINDINGS OF FACT AND RULINGS OF LAW

1. Are any benefits awarded herein? YES
2. Was the injury or occupational disease compensable under Chapter 287? YES
3. Was there an accident or incident of occupational disease under the Law? YES
4. Date of accident or onset of occupational disease: JUNE 26, 2005
5. State location where accident occurred or occupational disease was contracted: JOPLIN, MO
6. Was above employee in employ of above employer at time of alleged accident or occupational disease? YES
7. Did employer receive proper notice? YES
8. Did accident or occupational disease arise out of and in the course of the employment? YES
9. Was claim for compensation filed within time required by Law? YES
10. Was employer insured by above insurer? SELF-INSURED
11. Describe work employee was doing and how accident occurred or occupational disease contracted:  
EXPOSURE TO BENZENE AND OTHER CHEMICALS
12. Did accident or occupational disease cause death? NO
13. Part(s) of body injured by accident or occupational disease: BLOOD SYSTEM, WRISTS, FEET, SKIN,  
KIDNEYS AND BODY AS A WHOLE
14. Nature and extent of any permanent disability: PERMANENT TOTAL DISABILITY
15. Compensation paid to-date for temporary disability: NONE
16. Value necessary medical aid paid to date by employer/insurer? NONE

17. Value necessary medical aid not furnished by employer/insurer? PAST MEDICAL EXPENSES AMOUNT TO \$734,586.49; AND TRAVEL EXPENSES AMOUNTING TO \$17,434.59: TOTAL \$752,021.08
18. Employee's average weekly wages: \$596.18
19. Weekly compensation rate: \$397.45
20. Method wages computation: STATUTORY

#### COMPENSATION PAYABLE

21. Amount of compensation payable: The employee is awarded permanent total disability benefits at a rate of \$397.45 per week from August 1, 2005 through the remainder of the lifetime of the employee.

Unpaid medical expenses: \$752,021.8.

Penalty: The employee is also awarded a penalty of fifteen percent (15%) of past permanent total disability benefits, of fifteen percent (15%) of the amount awarded pursuant to *Section 287.120.4*, based upon statutory violations of Chapters 292.300, 292.310 and 292.320. This penalty shall also apply to future permanent total disability benefits.

22. Second Injury Fund Liability: Dismissed

TOTAL: UNDETERMINED

23. Future requirements awarded: PERMANENT TOTAL DISABILITY BENEFITS

The employee is also awarded future medical treatment to cure and relieve the effects of the multiple myeloma. Further, the employee is awarded future medical treatment to cure and relieve the effects of complications from either multiple myeloma or medical treatment, primarily chemotherapy, to treat the multiple myeloma. These complications include, but are not limited to, entrapment neuropathies in the upper extremities, neuropathies in the lower extremities, skin cancer and chronic kidney disease. This award also specifically finds that further complications may result and the finding of the complications mentioned here do not exclude complications that may result in the future.

Said payments to begin AUGUST 1, 2005 and to be payable and be subject to modification and review as provided by law.

The compensation awarded to the claimant shall be subject to a lien in the amount of 25% of all payments hereunder in favor of the following attorney for necessary legal services rendered to the claimant:

Attorneys fees and expenses: Attorneys' fees of twenty-five percent (25%) of the amount recovered are awarded to the Law Firm of Neale & Newman, LLP and Patrick J. Platter. A lien is placed upon this award pursuant to *Section 287.260 R.S.Mo. 2000*.

## **FINDINGS OF FACT and RULINGS OF LAW:**

Employee:	Howard Moreland	Injury No.	05-144489
Dependents:	N/A		
Employer:	Eagle Pitcher Technologies, LLC		Before the <b>DIVISION OF WORKERS' COMPENSATION</b> Department of Labor and Industrial Relations of Missouri Jefferson City, Missouri
Additional Party:	N/A		
Insurer:	Self-Insured		
Hearing Date:	August 20, 2010	Checked by:	

### **AWARD**

This claim was the subject of a two-day hearing held on Friday, August 20, 2010 and Wednesday, September 1, 2010 at the Joplin office of Division of Workers' Compensation. The Claimant, Howard Moreland, appeared with his counsel, Patrick J. Platter. The Employer appeared by its legal counsel, Robert Gross and Safety Director William Ideker. Claimant dismissed the Second Injury Fund at the commencement of the hearing held on September 1.

The following award is a compilation of the proposed awards submitted by both parties in this matter with the Court's revisions.

### **STIPULATIONS**

The parties stipulated to the following:

(1) That the Claimant, Howard Moreland was an employee operating under and subject to the Missouri Workers' Compensation Law;

(2) That the Employer, Eagle Picher Technologies, Inc., was an employer operating under and subject to the Missouri Workers' Compensation Law;

(3) That Eagle Picher Technologies, Inc. was self-insured, at all times material, for liability under the Missouri Workers' Compensation Law;

(4) That the Claimant, Howard Moreland, was an employee of Eagle Picher Technologies, Inc;

(5) Venue was proper as the exposure occurred in Jasper County, Missouri;

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(6) That the compensation rate is \$397.45;

(7) That past medical expenses were \$734,586.49 and travel expenses were \$17,434.59 for a total of \$752,021.08.

### ISSUES

Howard Moreland seeks benefits due to an alleged occupational disease. Although employed with Eagle Picher since 1973 the claim is centered on his employment from 1984 until approximately 1994 at the Joplin plant located at C & Porter. He worked in a building known as Building #4 during that time. Moreland became ill in June 2005 and was definitely diagnosed with multiple myeloma in late July 2005. He claims that his exposure to chemicals such as, but not limited to, benzene, caused his multiple myeloma. Eagle Picher has denied the claim in its entirety. It argues that Moreland was not exposed to these chemicals and, further, that none of the chemicals alleged to have caused this disorder have been scientifically recognized to cause multiple myeloma.

Eagle Picher does not dispute that Moreland's various complications specified in this Award resulted from either Moreland's multiple myeloma, his stem cell transplants or chemotherapy.

The parties designated the following issues to be in dispute:

(1) Whether the Claimant, Howard Moreland, suffered an occupational disease as defined in *Section 287.067 R.S.Mo. 2000*;

(2) The liability for past medical expense;

(3) The liability for past temporary disability;

(4) The nature and extent of permanent disability;

(5) Future medical treatment;

(6) Whether the Claimant filed his claim within the applicable statute of limitations;

(7) Whether Eagle Picher Technologies, Inc. is liable for a statutory penalty pursuant to *Section 287.120.4 R.S.Mo. 2000*.<sup>1</sup>

### EVIDENCE

At the hearing Claimant testified on his own behalf. The following coworker witnesses also testified on behalf of Claimant: Kathy Ogden, Tom Betebenner, John Newberry, Leroy Christy, Julie Alford, and Donnie Smith.

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<sup>1</sup> Unless otherwise specified, all references to statutes shall be to RSMo. 2000.

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Patrick Platter, claimant's attorney, seeks an attorneys' fee of twenty-five percent (25%) of all amounts recovered pursuant to *Section 287.260*.

The following exhibits were admitted into evidence on behalf of the Claimant Howard Moreland:

- A. Deposition of Bernard Goldstein, MD and exhibits with DVD video
- B. Deposition of Allen Parmet, MD and exhibits
- C. Deposition of Mauricio Pinada-Roman, MD and exhibits
- D. Complete Medical Report of Leslie Hamlett, MD
- E. Deposition of Jeff Dermott and exhibits
- F. Deposition of William Ideker and exhibits
- G. MSDS Documents from Eagle Picher
- H. Medical Records – Irving LaFrancis, MD
- I. Medical Records – Samuel Carter, MD
- J. Medical Records – Freeman Hospital
- K. Medical Records – Freeman Radiation Oncology
- L. Medical Records – UAMS
- M. Medical Records – Leslie Hamlett, MD
- N. Medical Records – Derek Towery, MD
- O. Medical Records – John Ogden, MD
- P. Medical Records – Matthew Richins, MD
- Q. Medical Records – Michael Swann, MD
- R. Billing Records Summary
- S. Floor Plans – Eagle Picher facility
- S1 Floor Plans – Kathy Ogden
- S2 Floor Plans – John Newberry
- S3 Floor Plans – Donnie Smith
- S4 Floor Plans – Leroy Cristy
- S5 Floor Plans – Howard Moreland
- S6 Floor Plans – Harry Betebenner
- T Report of Injury
- U Environmental Covenant
- V Supplemental Deposition of Dr. Allen Parmet

Two witnesses testified at the hearing on behalf of the Employer: Bill Ideker, Director of Health, Safety and Environment and Dennis Chiappetti, Production Supervisor.

The following exhibits were admitted into evidence on behalf of the Employer, Eagle Picher Technologies, Inc.

- 1. Accident Reporting Guidelines
- 2. Analytical Procedures for Ni-H2
- 3. Hazardous Waste Disposal Reports
- 4. Health Safety & Environment Department Guidelines

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5. IH Monitoring Data
6. Dr. Parmet's 03APR09
7. Original claim filed by H. Moreland
8. Dr. Borak transcript & exhibits
9. Certificates of Compliance for Rubber
- 9a. H. Moreland's Application for Family or Medical Leave
10. MSDS for Rubber [NBR 6850]
11. MSDS for PS-18A Acrylic Adhesive
12. MSDS for PS-18B Acrylic Adhesive Catalyst
13. MSDS for PS18C Acrylic Adhesive Promoter
14. Transcript of the Deposition Testimony of Howard Moreland 12SEP08

### **FINDINGS OF FACT**

Howard Moreland is 57 years of age and resides in Webb City, Missouri with his wife, Cheryl Moreland. They have two sons. Howard has lived most of his life in the Joplin/Webb City area. He graduated from Webb City High School and attended one year of college at Missouri Southern State College-Joplin.

Moreland is a second generation employee of Eagle Picher. Moreland's father worked for Eagle Picher until his death due to a cerebral hemorrhage. There is no indication that a family history contributed to Moreland's disease.

Moreland does not smoke cigarettes, drink or take recreational drugs. He chewed tobacco for approximately twenty years. That personal habit did not contribute to the circumstances that underlie this claim. No physician attempted to connect Moreland's use of smokeless tobacco with any of his medical disorders.

### **Employment History Eagle Picher**

Moreland worked for Eagle Picher from 1973 to 2005. Moreland worked at the Galena, Kansas location for approximately two and a half years, from 1973 through 1976. Moreland's work here primarily consisted of the manufacture and processing of materials for fertilizer. He transferred to the Eagle Picher location in Quapaw, Oklahoma, in 1976 and worked there from 1976 through 1984. He was initially an operator, then a foreman, in the manufacture and processing of boron.

Moreland then transferred from Quapaw, Oklahoma, to the C & Porter location in Joplin, Missouri. He worked in a particular location known as "Building 4" for ten years. This extended from 1984 through 1994. Moreland was primarily assigned to a work department in Building 4 that concerned the manufacture of nickel cadmium and nickel hydrogen components for battery cells. Moreland, however, would perform work in other work departments in Building 4 frequently. He initially worked in Building 4 as a tank operator and was promoted after about one year to working foreman.

Moreland transferred in 1994 to a different building known as "special products." This

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particular building, still at C & Porter, was located across the street from Building 4. He was first employed as a technician, then an engineering technician, then as a foreman/supervisor over construction.

Moreland's last day on the job for Eagle Picher was August 1, 2005.

### **Work Environment at Eagle Picher.**

The location where Moreland claims to have been exposed to the chemicals which caused his multiple myeloma was in Building 4 at C & Porter. There were several deficiencies at Building 4 from 1984 to approximately 1992 that concerned the exposure of employees to hazardous chemicals. The first deficiency concerned fumes emanating from open vats. Employees testified that they were responsible for plating components for batteries in vats that held hazardous chemicals. Employees wore no respiratory protection and ventilation hoods and ducts which extended to and over the vats would not carry the fumes to a safe location. The fumes would hang in the air causing people, especially unfamiliar with the work place, to become nauseous. Some of the chemicals, especially if splattered upon employees, would cause skin ulcerations. Plant personnel would place fans in Building 4 to circulate the air, though this would only carry fumes from one location to another. The effect was that employees would be exposed to hazardous chemicals that there were not directly working with. This was testified to by multiple witnesses.

The rubber room was responsible for cooking raw rubber in order that rubber could be processed and then placed to border components of the batteries. Raw rubber would be taken from an old-fashioned refrigerator, placed into an oven and "cooked" at a very high temperature. The temperature was high enough to create fumes which employees such as Moreland breathed.

According to witness testimony there was a plastic shop located immediately next to the nickel cadmium/nickel hydrogen work area that Moreland typically worked in. Benzene was in the glue that would adhere different pieces of plastic together. The heating of the glue while plastic was molded would create its own fumes and circulate throughout the building such as when fans were operating. Employees would cut these plastic parts throughout a work day. Cutting the plastic would create so much dust that it would resemble sawdust accumulating on a floor. So much plastic dust would fly that it would collect three to four inches deep on the work floor where Moreland worked in nickel cadmium/nickel hydrogen.

### **Coworker Witness Testimony**

Kathy Ogden was an employee of Eagle Picher from approximately 1985 to 2001. She worked in building number 4 for about seven to eight years. Ogden was very familiar with the environment in and around the plastic shop. The smell of glue was quite strong. Labels on containers used to make glue indicated that the material was flammable and a cancer-causing agent. Smelling the glue would leave one fuzzy-headed and with headaches. The glue also left a strange smell in one's nose. The smell also permeated clothes. She testified that although the "fans were always going" they were not effective in removing fumes and dust, but instead caused it to blow back onto you.

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She verified the means of exposure from chemicals to infiltrate the skin, eyes and breathing. Although employees wore aprons, boots, gloves and glasses, there was no respiratory protection. There was no medical monitoring. There were no warnings or training from the company concerning work habits about the hazardous chemicals.

Ogden knew of several employees who died from cancer while she worked there. They included Jim Morgan, Vonda Marks, Bob Johnson, J.R. Tinkle, Rob Leslie, Sam Bass, Ray Buening and Fred Divine.

Tom Betebenner worked for the company for 46 years. He worked in the plastic shop for fifteen (15) years. When he first started working in the plastic shop, it was originally located in building 11 and then was relocated to Building 4. The only personal protective equipment there was aprons and gloves, but they were not required. He stated that sawing plastic was an everyday occurrence. He verified the extent of plastic dust in the air. He verified, as Ogden, that union employees wore coveralls, though non-union employees were not provided this. The company provided no warnings concerning the use of chemicals until the late 1980s. He stated that material safety data sheets were available, but the company did not encourage their reference. He testified to the use of multiple solvents containing benzene.

John Newberry worked at Eagle Picher from 1953 to 1996. He worked at building number 4 in the nickel hydrogen and nickel cadmium processes with Moreland. He verified that the ventilation from the ducts before remodeling in approximately 1992 was quite poor. He stated that the fumes were present daily. He stated that the fumes would be so bad that visitors to the building would have to leave because of the extent of the fumes. The manufacturing process for nickel hydrogen, for example, left a yellow smoke from nitric acid fumes. When washing the negative plates there were fumes off the trichlorethylene and this process was done in a small space. He verified that everyone in the building was exposed to all of the chemicals because of the poor ventilation and movement of the fumes from one work area/department to another. He also verified that labels were removed from containers. He likewise verified that there was no respiratory protection for employees.

Leroy Christy worked in the nickel cadmium/nickel hydrogen work department with Moreland. He verified fumes from the plating. He stated that benzene was present during the cleaning process. Benzene was used as the stripper in the nickel hydrogen department. He also verified the fumes and dust from the plastic department. He stated that dust could be picked up with shovels. He likewise verified the poor quality of ventilation before remodeling. He stated that employees were exposed to either fumes or solvents from nitric acid, TCE, benzene and lithium. He likewise verified that employees would remove labels from containers.

Donnie Smith worked at Eagle Picher from 1988 to 2000. He started his employment in building number 4, working there from 1988 until 1992. Moreland was his shift leader in the nickel hydrogen work department. He also worked in the plastic shop and rubber shop. He remembered that the remodeling in Building 4 happened in the early 1990's, most likely 1992. He believed the remodel was done in part to get rid of the nickel cadmium contamination which they were phasing out. The chemicals with which he remembered being exposed, either through

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solvents or fumes, included nickel, phenyl thaline, cobalt, cadmium, platinum, sodium nitrate and lithium. He likewise verified that employees would remove labels on all containers. Employees would even cut the labels from the containers with a razor blade. Personal protective equipment included only gloves, goggles and plastic aprons. He was never provided respiratory protection. He knew of no air quality testing during this time.

Julie Alford worked for Eagle Picher from 1987 to 2006. She worked in a unit known as the "school house," then in the silver zinc department, in the snake pit, and then the plastic shop. She remembered that building 54 was destroyed by fire in June 1991. A bulldozer leveled the remnants of the building within a week of the fire. On one occasion she and a co-worker were told to go to a local chat pile to pick up labels that were blowing around the location. These labels were from containers that formerly contained hazardous chemicals at Eagle Picher and were at the site because that was "where the pit had been buried." Management at Eagle Picher instructed them to do this. She verified that no respirators were available for use. She likewise verified the extent of fumes from all chemicals and the extent of plastic dust generated by the plastic shop. The only medical monitoring she knew of regarded mercury levels and that was for a very limited time. She testified that every area was required to remove labels from any chemical container.

The claimant, Howard Moreland, detailed the processes for making nickel cadmium components for batteries and nickel hydrogen components for batteries. He also identified the process of a tank operator. He likewise verified the proximity of this work area to the plastics department. He testified that benzene was in the glue used on the plastics and was therefore in the sawdust. This dust would vary in volume from two to three inches to two feet deep. He also identified work processes in the rubber lab where he occasionally worked and discussed the volume of fumes during and after baking rubber. He stated that the most fumes were in the rubber lab, plastic shop, and nickel cadmium formation. Claimant also cleaned out a cabinet full of chemicals prior to a remodeling of Building 4. He testified that the chemicals in the cabinet included zinc, lead, carbon, benzene, nickel, and cadmium. He also testified that he ordered the raw rubber. On the certification or "cert" for the rubber was the "recipe" which showed benzene as an ingredient. He had seen this on multiple occasions. He identified the following chemicals to which he was exposed. Those included benzene, TCE, muriatic acid, cadmium, platinum and thenyl phaline. He stated that no respiratory protection was provided before 1994.

I find these witnesses to be credible and persuasive. Eagle Picher produced no evidence to rebut the testimony of the witnesses concerning atmosphere within Building 4. Eagle Picher denied the use of benzene in Building 4.

### **Onset of Illness.**

Moreland first presented to his primary physician, Dr. Samuel Carter, on June 14, 2005. Dr. Carter saw Moreland on June 14 and June 22 before an eventual hospitalization. Moreland reported that he had been more depressed, fatigued and listless for the past several weeks. He had frequently felt sleepy. Dr. Carter initially prescribed Wellbutrin. Moreland thought that he felt worse from Wellbutrin when he returned for a second office visit.

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### **Medical Treatment**

Dr. Carter decided on June 22 to admit Moreland for a hospitalization. The initial diagnosis upon admission was new onset diabetes with severe hypoglycemia, hypocalcaemia and dehydration. Lab studies conducted during this hospitalization indicated a urine protein electrophoresis that indicated a monoclonal band. A serum protein electrophoresis confirmed this. Dr. Carter suspected, based upon this testing, with a severe hypocalcaemia and worsened renal insufficiency that Moreland suffered from multiple myeloma. He then referred Moreland to oncologist, Dr. Irving LaFrancis.

Dr. LaFrancis conducted a bone marrow aspiration with biopsy on July 1, 2005 and confirmed the diagnosis. He conferred with Moreland on July 11, 2005. He referred Moreland to the University of Arkansas Medical Center, since a unit within the Center specialized in the treatment of multiple myeloma.

Moreland's first significant treatment at the University of Arkansas was during a hospitalization that lasted from July 26 through July 29, 2005. Dr. Roman Pineda was the attending oncologist. He conducted a repeat bone marrow biopsy and aspiration and a CT guided bone marrow biopsy. The latter confirmed the diagnosis of multiple myeloma. An MRI scan found a positive focal lesion in the sternum and a positive focal lesion in the seventh rib. A PET scan conducted on July 26, 2005 also indicated a large breakout lesion in the lateral seventh rib. Dr. Pineda outlined a treatment plan for Moreland on July 29, 2005. He offered what was called a total therapy III protocol. This consisted of a combination of stem cell transplants and chemotherapy. He stated that eighty percent (80%) of patients enjoyed a complete remission, based upon 150 patients currently enrolled.

Moreland then started consecutive rounds of chemotherapy on August 5 and September 6, 2005. He underwent his first stem cell transplant on October 20, 2005. He underwent his second stem cell transplant on January 11, 2006. He also underwent a round of chemotherapy on March 27, 2006. Dr. Pineda announced him to be in complete remission on September 21, 2006.

The chemotherapy, however, resulted in complications found by UAMS staff and they recommended that Moreland undergo treatment for these complications.

Moreland has suffered from bilateral carpal tunnel syndrome, cubital tunnel syndrome to the right wrist, trigger fingers, and a left ulnar nerve entrapment. He has underwent surgical releases for these disorders on November 1, 2007, December 7, 2007, May 15, 2008, and January 22, 2009. Dr. Timothy Ogden of Orthopedic Specialists of the Four States has been the attending orthopedic surgeon. Moreland first presented to Dr. Ogden on October 15, 2007. This initial evaluation concerned Moreland's right hand. Moreland complained of numbness in all fingers of the right hand, though more in the median distribution than ulnar. There was also numbness in the ulnar distribution of the left hand, particularly the ring and little fingers. Dr. Ogden noted that UAMS studies indicated peripheral neuropathy and right carpal tunnel syndrome, though the studies did not mention an ulnar nerve entrapment at the elbow. Dr. Ogden initially diagnosed

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carpal tunnel syndrome of the right wrist, right ulnar neuritis at the elbow and peripheral neuropathy.

The first surgery on November 1, 2007 was a right carpal tunnel release and right ulnar nerve transposition. Moreland returned to Dr. Ogden on November 9, 2007. He informed Dr. Ogden that his left upper extremity was bothering him like his right and that he wanted to do the same for the left. Dr. Christopher Andrew confirmed that Moreland would benefit from a left ulnar nerve transposition and may also benefit from a median nerve release. Dr. Ogden performed these on December 7, 2007 after nerve conduction and EMG studies.

Moreland returned to Orthopedic Specialists on April 29, 2008 and informed the staff that he had locking of his index, middle and ring fingers. The staff initially suspected trigger fingers. Dr. Ogden performed release of the A-1 pulleys of the right index, middle and ring fingers on May 15, 2008.

Moreland returned with a reoccurrence of symptoms resembling nerve entrapment to the left wrist on December 9, 2008. EMG and nerve conduction studies were conducted and indicated a recurrent ulnar distribution of the left hand. Dr. Ogden believed that Moreland would benefit from a repeat ulnar nerve anterior transposition followed by decompression of the ulnar nerve at the left wrist. This was performed on January 22, 2009. Dr. Ogden's staff released Moreland upon a PRN basis on February 11, 2009.

Moreland has also suffered from neuropathies in his feet. Moreland commenced treatment with Dr. Matthew Richins, a podiatrist affiliated with Four States Foot Clinic, on March 20, 2008. Dr. Richins measured Moreland for shoes and inserts. He also conducted an extremity echography and recommended orthotics. Dr. Richins recommended three pairs of custom molded orthotics and shoes due to bilateral pronation, which affected Moreland's ability to perform daily activities. Moreland periodically during 2008 followed with Dr. Richins concerning his shoe wear.

Moreland returned on March 9, 2009 complaining of burning, numbness and tingling in both feet. Dr. Richins, on August 17, 2009, recommended prescriptions for new orthotics and noted that Moreland had symptoms consistent with tarsal tunnel syndrome. Moreland saw Dr. Richins on December 1, 2009 and Dr. Richins instructed him to gradually increase the time for wearing the orthotic shoes.

Moreland has also undergone treatment for skin cancers. Dr. Derek Towery, an oncologist, and Dr. Michael Swann, a plastic surgeon, have treated these skin cancers. Dr. Towery, it should be noted, diagnosed an ulcerated basal cell carcinoma on August 5, 2003. Dr. Towery conducted a lab skin check on July 19, 2005 and recommended an excision. Moreland was at UAMS in Little Rock at the time for his multiple myeloma. Dr. Towery has seen Moreland approximately twenty-five times between February 19, 2007 and March 15, 2010 for recurrent skin cancers. The procedure would be diagnosis, biopsy, treatment and/or removal of the skin cancers. The surgical procedure would either be for an office procedure with Dr. Towery to excise them or, instead, to refer Moreland to Dr. Swann for a more involved surgical excision.

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Moreland has also started treatment for chronic kidney disease. This treatment shall be specified in the section of the findings of fact that concern the opinions of Dr. Leslie Hamlett.

### **Notice to Employer/Insurer's Policy of Reporting Injuries**

Jeffrey Dermott testified by way of deposition. Mr. Dermott is a Project Engineer at Eagle Picher's C and Porter Street facility, and he testified that he had known Mr. Moreland for over twenty years. Mr. Dermott also testified that he was Mr. Moreland's supervisor from 2003 to July, 2005, which was when Mr. Moreland ceased working at Eagle Picher. Mr. Dermott testified that after Mr. Moreland began treatment for multiple myeloma in Little Rock, Arkansas, Mr. Moreland called him to keep him apprised of his treatment progress. Mr. Dermott testified that he had notified Eagle Picher's Human Resources when he learned that Mr. Moreland had been diagnosed with multiple myeloma so that Human Resources would be aware of Mr. Moreland's absence from the workplace. Mr. Dermott also testified that Mr. Moreland did not tell him that he multiple myeloma diagnosis was related to Mr. Moreland's work at Eagle Picher.

Mr. Moreland testified that once he started missing work at Eagle Picher in late-June 2005, he kept his Eagle Picher supervisor, Jeff Dermott, apprised of his medical condition. The telephone number for the Eagle Picher plant at C & Porter is 417-623-8000. Cell phone records of Moreland, an exhibit to the Dermott deposition admitted into evidence, reflect that Moreland frequently called the Eagle Picher plant. Those days included July 5, July 13, July 21, July 22, July 26, July 27, July 28 and July 29.

The cell phone records document a noteworthy cell phone call that happened on July 29 at 3:05 p.m. Moreland testified that he called his supervisor, Dermott, on that day and that time. July 29 was a Friday. This was the day that Dr. Pineda informed Moreland of the final diagnosis of multiple myeloma and that the cause was exposure to chemicals at Eagle Picher. Moreland called the plant, while driving home, and spoke with Dermott during this particular phone call. Moreland informed Dermott of his diagnosis (multiple myeloma) and the cause of that disease (exposure to chemicals at work). Moreland testified that he informed Dermott because Dermott asked him.

I find the testimony of claimant, Howard Moreland, to be credible and most persuasive. I find and conclude Howard Moreland notified Jeff Dermott of his diagnosis of multiple myeloma and how it was related to his exposure to chemicals at Eagle Picher when he called Dermott from his cell phone on July 29, 2005 at approximately 3:05 p.m.

The parties stipulated that Eagle Picher did not file a Report of Injury with the Division of Workers' Compensation until after Moreland filed his original Claim for Compensation which was filed December 17, 2007. Thus, Eagle Picher failed to timely file a Report of Injury as required by §287.380, RSMo.

### **Present Condition**

Moreland still suffers from fatigue, much as he did when he started his medical treatment

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in 2005. He also suffers from pain and tingling in his feet which prevents him from walking normally. His medications include Dexamethasone. He takes this medication three weeks on and one week off at a four mg. doze. This medication is part of his oncology protocol. A medication for his kidneys includes Lisinopril, 40 mg. daily. These are but two, but prominent, medications that he takes either for his underlying cancer or for complications.

Dr. Parmet testified that Moreland reached maximum medical improvement because Moreland was taking maintenance chemotherapy. He believed, however, that Moreland would require future medical treatment.

“I would prefer to defer that to the treating oncologist because the protocols that they use sometimes change with time and a new development will show that the same drugs, different drugs, different versions and administration of drugs become the better pathway for him. So rather than somebody like myself who doesn’t treat myeloma, I’d rather defer to the treating physicians here.”

Regardless of the specific protocol, however, Dr. Parmet testified to a reasonable degree of certainty that a protocol should be followed:

“If he doesn’t follow this protocol, he is either going to have a high risk of relapse, recurrence of his myeloma, he could get what’s called graft v. host which is where the -- the stem cell transplant he got actually rejects him. And that – that can cause death.

Or he’ll get a super infection from some opportunistic agent and that would kill him. So even he doesn’t stay on protocol and continue to follow with his treating physicians, he is at grave risk.”

Moreland testified that Dr. Hamlett has advised him that, with his kidney function at approximately thirty (30) to forty percent (40%), that he is a potential candidate for a kidney transplant.

### **Testimony of Dr. Roman Pineda**

Dr. Roman Pineda testified on behalf of Moreland. He was, at the time of his deposition, an assistant Professor of Medicine at the University of Arkansas for Medical Services. He practiced at the Myeloma Institute for Research and Therapy. He had been located there since the summer of 2004. He was board certified in hematology in 2005 and in medical oncology in 2004. He conducted his fellowship in hematology/oncology from 2001 through 2004 at the University of South Carolina.

The basic mission of the Institute is to find a cure for multiple myeloma or, barring that, to find optimum treatment. The Institute is both a research and treatment institution. It has a worldwide population that is based upon total therapy protocol. Dr. Pineda described multiple myeloma. It is a cancer of the type of white cells known as plasma cells. These plasma cells are located inside the bone marrow. These white plasma cells constitute five percent (5%) of the

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bone marrow. These cells produce a protein called an antibody, which is part of a person's immunity. These cells recognize any foreign antigen of a substance and create antibodies approaching that will attack that vein of bacteria, virus or other element. These plasma cells become cancer when multiple myeloma happens. Just as in any other cancer, it is originated from a normal counterpart (the 5%), but then expands to anywhere from twenty (20) to ninety percent (90%). Complications of multiple myeloma include bone damage from the cancer cells which are located inside the bone because these usually remain inside the bone marrow and that, in turn, is inside every bone. This damages bones such as ribs, spine and mainly the central skeleton. Damage to the kidney is another complication from multiple myeloma because those plasma cells that are produced in excess get filtered through the blood into the kidneys. This can cause kidney inflammation and decreased kidney function.

The primary clinical feature to multiple myeloma is anemia. A clinical manifestation of multiple myeloma is the production of excess protein. The antibodies previously mentioned are proteins that can overwhelm the ability of the kidney and body to clear those from the system. The proteins will go out into the urine and cause kidney damage.

There are stages of multiple myeloma. The International Staging System is the new classification for these stages. The stages are based upon the levels of albumin and beta-2 microglobulin. If both readings are normal, the development of multiple myeloma is stage 1. If the beta-2 microglobulin is over 3.5, then the disease is in stage 3. Readings in between are stage 2.

Patterns of involvement include an abnormal MRI which indicates bone defects and an abnormal bone marrow biopsy. Very rare patterns also include situations where cancer cells proceed outside the bone marrow, which can be seen on PET scans. There are also some patients who produce only one smaller piece of abnormal protein. This means that normal plasma cells are able to produce normal antibodies. This has two parts, a heavy chain and a light chain. When the plasma cells become cancer plasma cells, this will produce an excessive amount of protein antibody; but it can appear normal looking. This is known as a "free light chain." These free light chains can cause other complications besides kidney damage. This is known as "light chain deposition disease."

A typical range of age for the onset of the disease is the mid-sixties.

The accepted criterion for the diagnosis is a monoclonal protein, which is more than two (2) grams. There must be a percentage of plasma cells in the bone marrow that exceeds twenty percent (20%) which detects clumps of plasma cells elsewhere in the body.

Howard Moreland had a type of multiple myeloma known as IgA Myeloma. "Ig" stands for immunoglobulin. This describes the type of protein that plasma cells normally produce. The "A" stands for the chemical type of the heavy chain. Most myelomas are the IgG type. The second most frequent group is the IgA type.

Patients undergo a bone marrow aspirate and biopsy. This is essentially a needle placed into a specific location of the pelvic bone from the back. The aspirate will extract a liquid. A

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syringe will aspirate several syringes of liquid of bone marrow. This will come out mixed with blood, be smeared, put under a microscope and analyzed. The biopsy consists of the same procedure. The pathologist will determine if there is excessive fat in the bone marrow and then look at the cells in between the fat and determine if those cells look normal or abnormal. If the cells are abnormal and, if there are abnormal plasma cells, one will then determine the percentage. If the percentage ranges from, for example, thirty (30) to ninety percent (90%), then the case for multiple myeloma is established.

The physician then reviews the MRI and PET scans. The MRI scan will determine the extent of damage within the bones, meaning the skeletal system. The PET scans will look for the progression of myeloma outside the bones. This is rare, but extremely dangerous because this means that the cancer cells have lost the ability to stay in the bone marrow. There are also different blood chemistries and blood counts to look for anemia, hemoglobin and platelet count. This is important because a platelet count indicates the ability to collect healthy stem cells despite having lots of myeloma cells inside the bone marrow.

An evaluation for myeloma patients also includes an echocardiogram. This is conducted to determine whether the "light chains" previously mentioned have gone into the tissues of the heart. One also looks at pulmonary function tests to determine if the patient will be able to tolerate an intensive treatment such as a stem cell transplant.

Moreland underwent the toral therapy III protocol. Toral therapy was started in the late-1980s. Toral therapy III started in 2004. Total therapy means to give as many active drugs against the disease as early on as possible. The rationale is to kill as many cancer cells very early in the process so they do not have the chance to continue growing. Multiple myeloma, like other cancers, can mutate and acquire resistance to a drug. The point is to treat the cancer as early as possible before the cells acquire a resistance. The Institute based this treatment similar to that found in childhood leukemia.

The treatment process is initially one chemotherapy cycle using seven drugs, known as ZDTPACE. Each of these initials stands for a different drug. Patients will initially receive four drugs by infusion in which they carry a pump with them for four days. Patients are seen every day in the chemotherapy room to have their blood count checked and to make sure that the drugs are being infused properly. The drugs and the pump are changed once a day and drugs are rotated twice a week for two weeks in this cycle.

The objective is to see the blood count eventually rise, which means that the bone marrow starts making healthy cells again. This is when the stem cells are collected. The process, from beginning to end, is about two and a half to three weeks. This means that there is one week to get the drugs in; one week for low counts; and another week for the counts to recover and the stem cells to be collected. The patient then gets a break of a week or two and then goes through another cycle just like the first. Some patients, during the second cycle, collect additional stem cells, but most patients collect all the stem cells they need in the first cycle. There is then another one to two week break after the second cycle. After that second break, the patient then returns for a stem cell transplant. The stem cell transplant is, essentially, a rescue. It is a two-step process. The first step is to give a medication called Melphlan. The next day the Melphlan is

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extracted out of the blood and the patient gets stem cells injected similar to a blood transfusion. There is a second transplant with a one to two month break in between. This is followed by two consolidation chemotherapy treatments because the same drugs that were given at the very beginning are then given two to three months apart. Patients will, however, stay two and a half weeks in town for the consolidated chemotherapy treatments to be conducted.

Maintenance then follows. There are three drugs typically used during the maintenance phase. These include medications by mouth and injection. Maintenance is normally conducted while the patients are at home. The staff will, of course, monitor the patient's progress during that time. Patients can donate their specimens either to the local primary care physician or oncologist who will then ship them to the Institute.

Moreland underwent this treatment protocol at the Institute, which included the maintenance program conducted near Moreland's residence. The records concerning the first stem cell transplant are reflected in Exhibits 12-22 of the Pineda deposition. Records concerning the second stem cell transplant concern records from Exhibits 23-36.

Mr. Moreland has also undergone what is known as PICC line placements. This stands for peripherally inserted central catheter. This means that a catheter is placed in a vein and reflects a long line that enters from the arm and goes all the way into the axillary subclavian vein for chemotherapy infusions. Moreland has also undergone a series of bone marrow biopsies in order to monitor his condition.

Moreland has suffered various complications from his multiple myeloma. These have included damage to the kidney for the reasons previously mentioned. He has also suffered neuropathy, which is a complication from myeloma and its treatment. Thalidomide and Velcade are the two medications that can cause neuropathy. This is present in both his hands and feet. He has also suffered from fatigue and a disturbance of balance, as well.

Multiple myeloma can fairly be considered an old person's disease. As people get older, the immune system will react to an infection. The mutation of the genetic material has more chance of falling into a mistake. The mistake is that the mutation does not create a variety of antibodies, but the mutation happens elsewhere in the DNA, inside the genetic material of the cell, and this happens to have an effect in a growth factor, which can make the cell become a cancer cell. As one grows older, the chances of this happening are higher. This process is not necessarily true of a younger person, who is for example 51 years of age at the time that the disease is diagnosed. Dr. Pineda identified the median age for onset of multiple myeloma as age 65.

Dr. Pineda identified the cause of Moreland's myeloma. He believed that Moreland's exposure to chemicals at his employment was the cause of his myeloma, because he was 51 years old. Other patients typically present with multiple myeloma in their sixties. This is more than ten years younger than the median age of a myeloma diagnosis. There is also a typical pattern of patients diagnosed with this disorder who have been employed in the oil, painting, automotive or hairdressing industry and who typically use dyes, agriculture chemicals and pesticides. This is very typical of patient exposure even if they come from outside the United States or from other

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geographical areas different from the South and Mid-south. Dr. Pineda identified primarily the benzene as the causative agent. He also identified lithium as causing kidney damage as having an extra contribution. He also testified that the increase in frequency of skin cancers had resulted from the myeloma.

Moreland, at the time of the deposition, had been in complete remission for approximately three years. The survival rate was sixty (60) to eighty percent (80 percent) for a survival rate of ten (10) years.

### **Testimony of Dr. Allen Parmet**

Dr. Allen Parmet examined Moreland at the referral of Moreland's legal counsel. He testified by deposition that four chemicals contributed to the development of multiple myeloma. They were benzene, cadmium, nickel and trichloroethylene. He is a specialist in occupational medicine. He graduated from the United States Air Force Academy with a bachelor's degree in chemical engineering and later graduated from the University of Kansas Medical School in 1976. He had two stages in his residency. He received a bachelor's degree in public health in 1981 from the University of Texas School of Public Health. He completed a second stage of his residency from the Air Force School of Aerospace Medicine in San Antonio, Texas in 1982. He attended a fellowship in Space Medicine at NASA. He has completed the didactic portion of a PhD at the University of Kansas in Toxicology. Dr. Parmet is board-certified by the American Board of Graduate Medical Education and the American Board of Preventative Medicine in the specialties of aerospace medicine and occupational medicine.

Approximately twenty-five percent (25%) of his time is spent in accepting workers' compensation referrals. In the relatively recent past, forty-seven percent (47%) of the referrals were at the appointment of plaintiffs or claimants and forty-two percent (42%) of the time was for defendants or the employer. The remainders were neutral examinations, in which he was either jointly appointed or requested by an Administrative Law Judge to evaluate the case.

Dr. Parmet defined multiple myeloma as a cancer of one of the cell lines involved in producing white blood cells of the bone marrow. It is called a hematologic malignancy. The most common are leukemias. Multiple myeloma is much more rare, but falls within the same group. The diagnosis depends upon an examination of cell types and the tendency of these cells toward immaturity and immortality of the cells. They keep reproducing. They do not function as a normal cell does and will invade other parts of the body both locally and remotely from their normal positions.

Dr. Parmet understood that Moreland, during his years working in proximity to the chemicals of most concern, did not have respiratory protection. This time would have been during Moreland's work at the Joplin plant buildings. Dr. Parmet particularly focused upon the chemicals of benzene, cadmium, nickel and trichloroethylene (TCE). The OSHA regulation for benzene exposure is one part per million upon an eight hour time-weighted average. If one has an open tank with a liquid, some of the liquid will simply escape into the atmosphere as a vapor and unless one does something to prevent that either by covering the tank or using a very special kind of surface ventilation system, it will escape into the atmosphere and enter the workers'

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breathing space.

Dr. Parmet testified regarding the four chemicals of which he had particular concern. Benzene is an organic chemical that is a carbon chemical of the primary aromatic ring structure. It is a very common chemical, but has a long history of being a suspect carcinogen. While suspected for being a carcinogen for seventy to eighty years, it was finally determined that benzene, once metabolized, eventually produces a carcinogen. Cadmium is a basic element of a metal that is extremely toxic as of itself. It is simply very, very poisonous. It is also associated with and felt to be causal for a number of kinds of cancer, particularly lung, prostate and testicular cancers. Nickel is a metal and another element. Nickel has a very unusual toxicity curve. Nickel participates with certain enzymatic reactions within the body so it is essential for normal health. It is important to have enough nickel so that certain enzyme systems within the body break down, but an excess of nickel will also cause direct toxicity and carcinogenesis, producing a dose response curve that is sometimes called a j-curve or a u-curve. Trichloroethylene is a widespread solvent and is controversial to whether it is toxic or not.

Dr. Parmet believed that these airborne chemicals were a substantial factor, more likely than not, in contributing to Moreland's multiple myeloma. Cancer is a complex disease requiring mutation after damage to the human DNA. Physicians know these agents will cause that and some of them are absolutely known to be carcinogenic. Benzene is known to be a hematogenic cancer agent, particularly for leukemia lines for the bone marrow where plasma cells produce the myeloma. Although the risk of benzene was known to be extremely high, physicians could not find the mechanism for that for many, many years and even then, some people believe that it is controversial. He, however, believes that the mainstream has accepted this. The statistics behind it were overwhelming and regulation was primarily based upon the statistical risk of benzene. This includes everything from ordering the reduction of benzene in gasoline to restricting sales of benzene to the general public.

Dr. Parmet identified factors in epidemiology regularly recognized by physicians when considering whether there is a causal connection between an agent and a disease. These are known as the Hill criteria and Dr. Parmet applied them as follows:

Temporal relationship is a factor. This means that one has to have an exposure to an agent before the development of the disease. With cancer, one expects a latent period which is a period of time between when the exposure begins and when the disease is actually present. Not only do mutations have to occur, but there must then be unregulated cell growth of the cancer that has to occur until it becomes present. The latency period here of (20) twenty years was appropriate to find a positive temporal relationship.

Strength of association is another factor. This is a statistical analysis and it has been controversial for myeloma. Benzene has been regulated for over twenty years and this effective regulation, therefore, reduces human exposure and, therefore, actual exposure. The numbers of cases have decreased and myeloma is a rare cancer anyway when compared to leukemias.

Consistency of association is another factor. Here, one looks for multiple studies which show similar trends. They may not show the exact trend, depending upon the popular selected,

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but the general trending of studies goes in the same direction. The studies that would show an association of benzene with myeloma are consistent with there being an increased risk. This is true for the majority of studies although the studies are not absolutely irrefutable as they are for leukemia.

Another factor, biological plausibility, is present here. Plausibility means that one may understand the process that is going on. The criterion of biologic plausibility is a strong criterion in favor of finding causal connection between benzene exposure and myeloma. We know that benzene is metabolized eventually in the body to a potent carcinogen and it is produced by metabolism in the bone marrow. One has production of a known carcinogen in the presence of stem cells that become plasma cells. We also know this simultaneously affects white blood cells and red blood cells, giving us different kinds of leukemias. Thus, the plausibility of causing a cancer in the same stem cell location by the same chemical agent becomes very readily appreciated because it is the same mutation. All one needs to effect is a different stem cell line, although a much rarer one. Dr. Parmet, in fact, indicated on diagrams from known toxicology texts how this metabolism would work.

Specificity of association is another factor. This deals with an exposure to a specific agent which causes a specific outcome or group of outcomes. There is an exposure geared to certain chemicals that are known carcinogens in general. They are known to cause bone marrow problems and there is therefore consistency here. The relative rarity of myeloma makes it difficult to get sufficient statistical evidence beyond that to make an association extremely strong.

A dose response relationship is another criteria. Simply put, the dose makes the poison. This can be translated into calculations such as parts per million (the former OSHA regulation of one part per million). A drop of benzene or a very small amount of cadmium can rapidly contaminate a workplace far above these levels. Dr. Parmet, in fact, has personal experience in conducting surveys in the workplace. For example, readjusting a break or performing spot welding on electronics is sufficient to surpass permissible cadmium levels.

The last criteria is the lack of an alternative relationship. Dr. Parmet specifically addressed the issue of whether obesity causes multiple myeloma and he refuted this proposition submitted by Dr. Borak based upon the Hill criteria. Dr. Parmet noted that there was no biologic plausibility for this proposition. The number of obese people in the United States, for example, has tripled in the past twenty-five years. The number of morbidly obese people has gone up a factor of six hundred percent. Under the Hill criteria, if there is an increased exposure to obesity, there should be an increased outcome. While there is clearly an increase of obesity, and, for example, an increase in obesity-related diseases such as diabetes, there has been no increase in the cases of multiple myeloma. Dr. Parmet showed this with charts (Exhibits 60 and 61). Dr. Parmet likewise did not believe that there was a biological plausibility to this proposition. He also found no scientific information which recognizes a temporal relationship between the onset of obesity or morbid obesity with the onset of myeloma.

Dr. Parmet found the open tanks around which Moreland worked to be quite significant. Open top tanks vaporize and these often have stirring and splattering, so one either has vapor or aerosols coming from the tanks themselves. The contents of the tanks enter the breathing space

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of workers and workers can inhale them. The absence of respiratory protection is significant. The liquid escapes into the atmosphere as a vapor. Unless something is done to prevent it, either by covering the tank or a special surface ventilation system, it will escape into the atmosphere and enter a worker's breathing space. Eagle Picher did not document the amount of vapors emanating from tanks. Dr. Parmet, with this, stated that there was no confidence that the employees were, therefore, working at safe levels.

### **Testimony of Dr. Bernard Goldstein**

Dr. Bernard Goldstein reviewed case material at the referral of Moreland's legal counsel. He testified that there is now credible evidence researched and identified within the past twenty years which has led him to believe that benzene is a chemical agent which causes multiple myeloma. He testified that he a ninety percent (90%) degree of confidence which, to his understanding, far surpasses what is necessary for a reasonable medical certainty.

Dr. Bernard Goldstein is a professor of medicine at the University of Pittsburg Graduate School of Public Health. He is also a professor of medicine at the University of Pittsburg School of Medicine. Dr. Goldstein is a physician, toxicologist and hematologist. He has worked at the University of Pittsburg approximately ten (10) and was, until recently, the dean of the Graduate School of Public Health. He was located at Rutgers University for approximately ten (10) years, serving as the founding director of the Environmental and Occupational Health Services Institute. This is a joint program of Rutgers University and the Robert Wood Johnson Medical School in New Jersey.

Dr. Goldstein has studied benzene toxicity and published upon the subject for approximately twenty (20) years. He has researched and published close to one hundred papers or reviews concerning benzene toxicity.

Dr. Goldstein has also specially published and instructed members of the federal judiciary upon issues that concern toxicology and, in particular, whether specific chemical agents should be deemed to have caused or contributed to the development of disease.

Dr. Goldstein testified that benzene was medically probable to be a cause of multiple myeloma. He testified that this is based upon epidemicalogical data, bioassays (experiments on laboratory animals) and mechanistic data. Epidemicalogical data concerns statistical studies in which researchers attempt to document past exposures of a chemical agent upon a class of individuals. Mechanistic data refers to accepted medical principals confirmed by medical research.

According to Dr. Goldstein benzene is a well-known potent hematological toxin. It was originally noted to cause human aplastic anemia which is a failure of production of blood cells by the bone marrow. This has been known since 1897. Scientific literature then reported that benzene was a cause of human leukemia. It was not until the late 1970s, however, with a now famous study, that the scientific community finally and fully accepted benzene as a cause of adult leukemia. The same workers who were the subject of this study also had an increase in multiple myeloma. The author of the original study then extensively reviewed the earlier study and, in a

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combined analysis of seven (7) pertinent studies, reported a statistically significant association between benzene and multiple myeloma. Other studies, if not all of them, have likewise found elevated risks of multiple myeloma associated with benzene exposure. Dr. Goldstein identified a meta-analysis authored by a Dr. Infante and others which was a follow up of this famous study from the late 70s. He found the Infante analysis reliable. Infante and others reviewed existing literature and chose studies that they believed to reflect benzene exposure and to reflect the workforce that was really exposed to benzene. Dr. Infante correctly, Goldstein believes, rejected certain studies which discounted any benzene myeloma connection. He rejected studies in which large groups of workers (e.g., 250,000) were not exposed to benzene exposure because there was no increased risk of the signature cancer. Likewise, some studies would not evaluate “contract labor” that were the traditional dirtier jobs in which those workers were exposed to benzene exposure but were not studied. This had the affect of analyzing sedentary desk-type workers from oil companies, but not analyzing the contract labor. Dr. Goldstein found the Infante approach to be reasonable. In addition, one researcher who originally performed a meta-analysis and found no connection (Dr. Sonoda) then took his own suggestion, performed a specific case study, and found a statistical association.

Dr. Goldstein noted that the medical community now considers multiple myeloma to be a form of non-Hodgkin’s lymphoma. The World Health Organization recognizes this classification. This is a cancer that has been clearly demonstrated to occur in benzene-exposed laboratory animals. There is also ample evidence of this in humans. Lymphatic and myelocitic cell lines both arise from the same bone marrow precursor cell. This is the same precursor cell that is at risk for mutation as a result of benzene exposure. It is the same precursor cell that is at risk for exposure with adult leukemia (known as acute myelogenous leukemia). Dr. Goldstein also testified that benzene- induce mutations are not specific, but produce a family of different mutations.

The mechanistic evidence in support of connecting benzene exposure to multiple myeloma has also grown stronger over the past twenty years. Benzene exposure clearly causes chromosomal damage to human lymphocytes which can be observed in circulating lymphocytes, including the V-lymphocyte of which plasma cells are a sub-type. This is evident in studies of exposed workers as well as laboratory animals. In fact, among all cells, the lymphocyte is particularly known to be at risk from benzene toxicity. There is no question, therefore, that exposure to benzene causes damage to human lymphocyte chromosomes, including a variety of genetic abnormalities that are known to increase cancer risks. Alterations of chromosomal genetic components leading to mutation are at the basis of virtually human cancers. This is in keeping with the role for benzene, known to cause chromosomal abnormalities of lymphocytic cells, in causing cancer of plasma cells.

Dr. Goldstein also stated the status of mechanistic data thusly:

“Well, again, it gets to mechanistic issues. There is no question whatsoever from a scientific point of view that benzene causes a cancer in the bone marrow. None whatsoever. There is no question that benzene causes chromosomal abnormalities that is manifest not only in the stem cell, but that you can see in the lymphocytes of circulating blood. There is no question that the

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plasma cell, -- that the myeloma cell is a lymphocytic series cell. Myeloma used to be considered separate from non-Hodgkin's lymphoma from the broad lymphoma series. Now it is considered to be part of it because we understand that these are really very related, that plasma cells are a form of lymphocytes. So here we have benzene causing a cancer in the bone marrow. It does it through acting on stem cells, which can differentiate lots of different directions and a multiple of all of these stem cells are responsible for causing the diseases that include all the non-Hodgkin's lymphoma, all of the leukemias and multiple myeloma. So we have what is mechanistically a very sound growingly strong story that benzene ought to be causing multiple myeloma based upon how we understand these things to occur."

Scientists can confirm this, where they could not, for example, twenty years ago, because there are now molecular markers which can identify these stem cells. These molecular markers can now track the progression of the stem cells.

Multiple myeloma is a cancer of plasma cells that usually appears in the bone marrow. These plasma cells are specialized lymphocytes that normally function to produce antibodies to foreign substances. Each plasma cell appears to produce just one antibody protein. Detection of a relatively large amount of a single serum protein, known as monoclonal gammopathy is a usual diagnostic finding when diagnosing multiple myeloma. The presence of a single specific protein being produced by all of the cancer cells demonstrates that multiple myeloma, like other cancers, begins as a mutation of just one plasma cell precursor. This single cell continues to double in number until there is a clinically significant case to diagnose multiple myeloma.

Dr. Goldstein disagreed with employer's expert, Dr. Borak, whose contention was that obesity was a cause of multiple myeloma. Dr. Goldstein stated that Moreland's obesity, if anything, explained the causal connection between benzene toxicity and multiple myeloma.

Occupational physicians have long considered body fat to be more susceptible to benzene toxicity, which would also explain an apparent higher risk among females. There is a great solubility in fat to benzene. There is also longstanding strong evidence that it is not benzene itself that is responsible for hematological toxicity, but rather the metabolites of benzene that are formed within the body. About one-half of benzene that enters the body is metabolized and the rest is exhaled harmlessly. Any factor that speeds up the metabolism of benzene will increase its toxicity because less will be exhaled.

The metabolic process that explains the formation of toxic benzene products is known to be dependent upon cytochrome P450 2E1 (also known as CYP2E1). The extent of benzene toxicity highly depends upon levels of CYP2E1. This was confirmed in a study of benzene-exposed Chinese workers in whom the level of bone marrow toxicity was related to the rate of metabolism of administered chlorzoxazone. Obesity likewise leads to an increase of CYP2E1 activity in humans because there is an increased metabolism of chlorzoxazone and directly in liver tissue.

Thus, there is a reasonable explanation consistent with Dr. Borak's point that obese

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individuals have a higher rate of multiple myeloma. The cause of a higher risk of multiple myeloma in obese individuals is external chemicals for which the CYP2E1-dependent metabolize are a source of toxicity, and benzene, which is a hematological poison, is a leading candidate.

The counsel for Eagle Picher, at the conclusion of Dr. Goldstein's direct examination, interposed an objection to the testimony of Dr. Goldstein at the end of cross-examination:

“Let me read an objection to this testimony for the record just so we have a record of it for legal purposes. I object to the introduction of the testimony of Dr. Goldstein in this work comp case and move that it be stricken on the grounds that his testimony does not meet the standard required of expert testimony by Section 490.065 of the Missouri Revised Statutes, nor does it meet the standards set for expert testimony in Bill 509 U.S. 579 nor does it meet the standard set forth for expert testimony in *Frye*, 293 F.1013 nor does it meet the standard for expert testimony set forth by the Supreme Court of Missouri in *The State Board of Registration for Healing Arts v. McDonagh*, 123 S.W.3d 146. Dr. Goldstein's testimony is not based on medical certainty but rather is based solely on educated speculation that amounts to nothing more than a possibility that Mr. Moreland's multiple myeloma was caused by benzene or any other chemical. Dr. Goldstein's testimony is not based on any medical or scientific facts that are reasonably relied upon by experts in the field of diagnosis or treatment of multiple myeloma, nor the facts and data upon which – on which Dr. Goldstein bases his opinion otherwise reasonably reliable as required by Section 490.065.3 of the Revised Missouri Statutes.”

First, Section 490.065, not the *Frye* test, is the sole basis for considering the admissibility of expert testimony in either civil actions or administrative proceedings pending before Missouri State Courts or administrative agencies. *State ex rel. Board of Registration of Healing Arts v. McDonagh*, 123 S.W.3d 146 (Mo. banc 2003); *McGuire v. Seltsam*, 138 S.W.3d 718 (Mo. 2004) expressly states that the *Frye* test used by courts outside of Missouri does not apply to Missouri judicial or administrative proceedings.

§490.065 RSMo. provides as follows:

1. In any civil action, if scientific, technical or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education may testify thereto in the form of an opinion or otherwise.
2. Testimony by such an expert witness in the form of an opinion or inference otherwise admissible is not objectionable because it embraces an ultimate issue to be decided by the trier of fact.
3. The facts or data in a particular case upon which an expert bases an opinion or inference may be those perceived by or made known to him at or before the

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hearing and must be of a type reasonably relied upon by experts in the field in forming opinions or inferences upon the subject and must be otherwise reasonably reliable.

4. If a reasonable foundation is laid, an expert may testify in terms of opinion or inference and give the reasons therefor without the use of hypothetical questions, unless the court believes the use of a hypothetical question will make the expert's opinion more understandable or of greater assistance to the jury due to the particular facts of the case.<sup>3</sup>

I find the testimony of Dr. Goldstein meets the standard required of expert testimony. Certainly Dr. Goldstein qualified as an expert by his knowledge, skill, experience, training, and education. Dr. Goldstein thoroughly documented his reasoning with articles or studies and closely explained why a collection of epidemiological studies, bioassays, and mechanist data (accepted medical principal) should all be considered, and to be taken as a whole, when considering whether a chemical agent causes a disease. This approach is sanctioned by the International Agency on Research for Cancer. The facts and data upon which he based his opinions are of a type reasonably relied upon by experts in the field in forming opinions or inferences upon the subject and are otherwise reasonably reliable. The objection is overruled.

#### **Testimony of Dr. Leslie Hamlett**

Dr. Leslie Hamlett is a nephrologist who practices with Freeman Health System in Joplin. Her first office visit with Moreland was on September 24, 2009. The staff at UMAS suspected that Moreland suffered complications of kidney disease as a result of chemotherapy. Dr. Hamlett directed a kidney biopsy on December 2, 2009. Dr. Hamlett not only was concerned about chronic kidney disease, but also a recurrence of multiple myeloma, although Moreland's serum parameters did not suggest a reoccurrence.

The biopsy indicated focal segmental and global glomerular sclerosis (also known as FSGS). This condition was severe. The biopsy also showed sub-acute thrombotic microangiopathy which included the glomeruli. It also indicated mild diabetic glomerular sclerosis.

Dr. Hamlett noted that there are multiple cases cited in medical literature which document the association between kidney disease with stem cell transplants and chemotherapy. There was no evidence on Moreland's biopsy to suggest that he had a reoccurrence of multiple myeloma. His proteinuria dramatically improved after the initial stem cell transplant and chemotherapy, and it then worsened over time. The most likely cause of this decline in renal function and worsening proteinuria was secondary to the stem cell transplant and chemotherapy. Moreland had other diseases that could result in proteinuria. The sub-acute thrombotic microangiopathy was related to high blood pressure. Moreland also had mild damage to the kidney related to the diabetes. The most prominent lesion in the kidney, however, was focal segmental glomerular sclerosis. Dr. Hamlett believed this was due to the stem cell transplant and chemotherapy.

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### **Testimony of Dr. Jonathan Borak**

Dr. Borak reviewed case materials at the referral of Eagle Picher's legal counsel. He testified that it has not been established through epidemiological studies that benzene is a chemical cause of multiple myeloma. He also disputed whether other chemicals identified by Dr. Pineda and Dr. Parmet were causes of multiple myeloma. His focus exclusively concerned epidemiological studies, and did not rely upon other criteria identified by Dr. Parmet (the other Hill criteria) or sources of evidence recognized by the International Agency on Research on Cancer (identified by Dr. Goldstein) which both physicians said must be considered when considering the issue of causal connection.

Dr. Jonathan Borak is a physician and toxicologist. He is the owner of a consulting firm, Jonathan Borak and Company, Inc. He is an associate professor in public health and medicine at Yale University. The subject matter of this claim falls within one of his specialties. The subjects that he teaches include toxicology and risk assessment.

Dr. Borak criticized the analysis of the three physicians who testified on behalf of Moreland.

Dr. Borak testified that he had reviewed the June 11, 2008, opinion letter of Mr. Moreland's multiple myeloma specialist in Little Rock, Dr. Mauricio Pineda-Roman [Exhibit 2 to Employer's Exhibit 8] in which Dr. Pineda-Roman had opined that Mr. Moreland's exposure to chemicals during his employment at Eagle Picher had significantly contributed to Mr. Moreland's multiple myeloma and skin cancers. Dr. Borak testified that he disagreed with Dr. Pineda-Roman's opinion since Dr. Pineda-Roman did not have any independent knowledge of the level of dosage or duration of exposure to any chemical to which Mr. Moreland claimed to have been exposed, and that without this vital information, a medical diagnosis of the causation of any disease is impossible [Employer's Exhibit 8, p. 8].

Dr. Borak also testified about the opinions rendered on behalf of Claimant by Dr. Allen Parmet. Dr. Borak testified that he had reviewed the April 3, 2009 Report that was authored by Dr. Parmet in which Dr. Parmet opined that Mr. Moreland's multiple myeloma is causally related to occupational exposures to multiple chemicals, specifically benzene. [Employer's Exhibit 8, pp.10-11] Dr. Borak testified that Dr. Parmet's opinion that Mr. Moreland had developed multiple myeloma as a result of exposure to benzene or other chemicals is incorrect for multiple reasons.

In his Report, Dr. Parmet's cited a 1992 case study by the Agency for Toxic Substances and Disease Registry [ATSDR], which is a division of the Department of Health and Human Services' Centers For Disease Control. [Employer's Exhibit 8, pp.12-13] Dr. Borak pointed out that the ATSDR report that Dr. Parmet cited contained the statement that there is no scientific proof of a causal relationship between exposure to benzene and multiple myeloma. [Employer's Exhibit 8, p.13] Dr. Borak also testified that ATSDR studies that were conducted since the 1992

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study cited by Dr. Parmet, including a 2007 study, have also failed to find that benzene causes multiple myeloma. [Employer's Exhibit 8, pp.13-14]

Dr. Borak also testified about another source that Dr. Parmet cited for the link between benzene and multiple myeloma, which is a chapter from a medical textbook by Harvison. Dr. Borak testified that the cited chapter from the Harvison textbook actually states that while there is evidence that benzene causes *leukemia*, evidence that benzene might cause *multiple myeloma* is not strong, and that the Harvison book chapter Dr. Parmet cited also states that the chronic effects of benzene depend on the level of exposure, a critical fact that is unknown in Mr. Moreland's case. [Emphasis added] [Employer's Exhibit 8, p.14]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a chapter from a leading toxicology textbook by Casarett and Doull. Dr. Borak pointed out that Dr. Parmet referenced an edition of the textbook that has since been revised twice, and that the more recent editions of the chapter cited by Dr. Parmet do not even mention multiple myeloma, but that the newer editions do state that investigators have concluded that there is no scientific evidence to support a causal relationship between benzene exposure and multiple myeloma. [Employer's Exhibit 8, pp.14-15]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a study, authored by a researcher named Aksoy, of approximately 28,000 Turkish leather workers who were exposed to benzene in the course of their leatherworking jobs. Among that group of workers, Aksoy identified 34 cases of acute myelogenous leukemia [AML], but only one case of multiple myeloma. Dr. Borak testified that Aksoy's findings clearly show no proof of association between benzene and multiple myeloma. [Employer's Exhibit 8, pp.15-16]

To confirm that multiple myeloma occurs more frequently than AML in the general population, Dr. Borak pointed out that the incidence rate of multiple myeloma and acute myelogenous leukemia [AML] among white American males of all ages has been tracked by the National Cancer Institute since 1975, and that the chart of that tracking, which is entitled "Surveillance Epidemiology and End Results" [SEER] shows that over that period of time, the incidence rate for multiple myeloma has been greater than it has been for AML, and that the corresponding mortality rate for multiple myeloma was also greater for multiple myeloma than for AML. [Employer's Exhibit 8, pp.63-64, and Exhibits 38 and 39 thereto]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a paper by Pyatt that is a review of the link between benzene and hematopoietic malignancies, i.e., cancers involving blood cells, that described multiple myeloma as one of five hematopoietic malignancies for which there are "insufficient data for causal link". Dr. Borak testified that Pyatt's review clearly does not support Dr. Parmet's opinion that benzene causes multiple myeloma. [Employer's Exhibit 8, pp.16-17]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a meta-analysis of case-control studies, by a researcher named Sonoda, of the relationship between multiple myeloma and engine exhaust. Dr. Borak pointed out that the individuals studied by Sonoda had, according to Sonoda,

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“probable exposure to organic solvents or petroleum”, but not to benzene. [Employer’s Exhibit 8, p.62] Sonoda’s study found that there was actually a significantly *decreased* association between multiple myeloma and exposure to petroleum, and that the Sonoda study revealed no association between multiple myeloma and exposure to benzene, organic solvents, or petroleum products, meaning that Sonoda’s study showed that benzene exposure is not a risk factor for multiple myeloma. [Emphasis added] [Employer’s Exhibit 8, pp.17-18]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is an analysis of a case-controlled study of thirteen hospital or population-based control studies of multiple myeloma that was published in a paper by a researcher named Bezabeh. In his testimony, Dr. Borak pointed out that Bezabeh’s findings do not support Dr. Parmet’s opinion on causation, since Bezabeh concluded that, “Benzene exposure is unlikely to be a causal agent for multiple myeloma. The current published case control literature is not ambivalent and does not indicate that benzene exposure is a risk factor for multiple myeloma”. [Employer’s Exhibit 8, p.18]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a case-control study by a researcher named Costantini that reviewed risks for acute myelogenous leukemia [AML] and multiple myeloma. Dr. Borak testified that Costantini’s study does not support Dr. Parmet’s opinion because Costantini reported that his study found an insignificant increase in risk for myeloma, and no increase in risk for AML. Dr. Borak pointed out that this finding causes the results of Costantini’s entire study to be questionable because there is a known link between benzene and the incidence of AML, so that if Costantini found no increase in the incidence of AML, the results of the entire study are unreliable. [Employer’s Exhibit 8, pp.18-19]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a paper by a researcher named Lynge. Dr. Borak pointed out that the Lynge paper discusses solvents in general, and does not even mention multiple myeloma, and does not therefore support Dr. Parmet’s opinion regarding a link between benzene and multiple myeloma. [Employer’s Exhibit 8, pp.19-20]

Dr. Borak next testified about another source that Dr. Parmet cited in support of an alleged link between benzene and multiple myeloma, which is a meta-analysis by a researcher named Infante. Dr. Borak testified that the Infante study is fatally flawed because Infante did not follow the standards of practice for such studies. Dr. Borak pointed out that Infante improperly utilized “post-hoc” analysis to reach a conclusion from his meta-analysis. Dr. Borak pointed out that Infante knew in advance that he was choosing for inclusion in the study the only group that would achieve the pre-determined result that Infante wanted, because Infante had been involved with studies involving this same group over a twenty year period. Dr. Borak testified that other studies involving this same control group showed results that Infante did not wish to find, so Infante merely ignored those studies.

Dr. Borak also testified that much of what is known about the effects of benzene on workers in America comes from the study of the Pliofilm cohort, a group of approximately 1,200 workers who were exposed to benzene while making a rubberized material during World War Two. [Employer’s Exhibit 8, p.24] Dr. Borak testified that there have been a number of follow-

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up studies of these workers over the years, and that the information obtained by the study of these workers has shed light on the effect exposure to benzene has had on the workers who were included in this study.

Dr. Borak testified that among the Pliofilm cohort, there was a 5-fold increase risk of acute myelogenous leukemia [AML], but that there were only four cases of multiple myeloma among the cohort. [Employer's Exhibit 8, p.24] Dr. Borak testified that the Pliofilm cohort was studied by various researchers through the years, and that studies conducted between 1994 and 1996 showed that there had been no more cases of myeloma among the group since a 1987 study, leading the researchers to the conclusion that there was no statistical significance between exposure to benzene and multiple myeloma, i.e., the rate of incidence between benzene and multiple myeloma in the Pliofilm workers was not greater than the incidence of multiple myeloma in the general population. A later analysis of the study of the Pliofilm workers was conducted by Rinsky and published in 2002, and it also confirmed that the relationship between benzene and multiply myeloma was not statistically significant. [Employer's Exhibit 8, p.25] Dr. Borak pointed out that when Infante did his meta-analysis study in 2006, he chose to use the data from the 1981 study rather than the data from the 1994 study, which Dr. Borak pointed out confirmed that Infante had cherrypicked the data in an attempt to prove his hypothesis. [Employer's Exhibit 8, pp.25-26]

Dr. Borak also testified that of the five total cases of multiple myeloma reported in the Pliofilm cohort, four of the cases were in the group that had the lowest exposure to benzene, and that one of those four individuals had been employed in the Pliofilm plant for only four days. Dr. Borak testified that scientists who have studied the link between chemical exposure and disease have correctly insisted that the level of dose of a chemical is an important determinant of effect, a precept that is consistent with the basic principles of toxicology, and a fact that studies of the Pliofilm workers has confirmed. Dr. Borak pointed out, however, that Infante's finding – that there was no dose-response effect – is completely at odds with the basic principles of toxicology. [Employer's Exhibit 8, pp.26-27]

Dr. Borak also testified that none of the chemicals other than benzene to which Mr. Moreland claimed to have been exposed has been shown to be linked to multiple myeloma. [Employer's Exhibit 8, p.27]

Dr. Borak, testifying on behalf of Eagle Picher, explained that the theory of 2E1 up-regulation - whether excessive exposure to benzene causes a change in human body metabolism that enhances the development of multiple myeloma - is indeed merely a theory that cannot be substantiated by medical proof.

Dr. Borak pointed out that if 2E1 up-regulation actually existed, it would have manifested in the numerous studies of the Pliofilm cohort. Dr. Borak pointed out that the opposite actually occurred. Multiple myeloma appeared in Pliofilm workers who had only minimal exposure to benzene, which Dr. Borak testified confirms that there is no proof that exposure to high doses of benzene increases the incidence rate of multiple myeloma. [Employer's Exhibit 8, p.47] Dr. Borak also testified that a study of 75,000 Chinese workers who were exposed to very high levels of benzene also did not report an increase of multiple myeloma among those workers greater than the general population. [Employer's Exhibit 8, pp.47-48]

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Dr. Borak also testified that the hypothesis of whether 2E1 up-regulation contributes to metabolic changes ignores the fact that a second enzyme, known as NQ01, works in combination with the 2E1 enzyme to produce metabolic changes. Dr. Borak testified that as such, the discussion of possible metabolic change due to benzene exposure is incomplete when both of these factors are not considered. [Employer's Exhibit 8, pp.49-52]

To support his contention that focusing only on the 2E1 enzyme as the factor that determines metabolic change in the presence of benzene, Dr. Borak pointed out that alcohol is known to elevate 2E1 by up to 400%, meaning that if elevated 2E1 were the sole factor in triggering the alleged metabolic change that leads to multiple myeloma, it could be expected that alcoholics would have a much higher rate of multiple myeloma than non-alcoholics. Dr. Borak testified that this is not the case. Instead, the rate of multiple myeloma among alcoholics is 80% lower than the general population.

Dr. Borak also testified that the theory of metabolic changes caused by enzymatic alteration is based on the premise that in order to increase the risk of developing multiple myeloma, an increase of 2E1 must be accompanied by a decrease of NQ01. Dr. Borak testified that medical tests have shown that obesity increases both 2E1 and NQ01, thereby increasing the risk that obese persons are more likely to develop multiple myeloma, a scientific finding that refutes the suggestion that 2E1 up-regulation occurring alone increases the risk of developing multiple myeloma. [Employer's Exhibit 8, pp.52-54]

Dr. Borak concluded that, given the scientific evidence that has been produced by numerous studies over several decades, benzene does not cause multiple myeloma, nor does exposure to benzene trigger any metabolic changes that lead to multiple myeloma. [Employer's Exhibit 8, p.59]

Dr. Borak, in a supplemental report, criticized the conclusions of Dr. Allen Parmet. He contended that seven of the ten studies cited by Dr. Parmet did not support the proposition that multiple chemicals could lead to the development of multiple myeloma. Of the three remaining articles, he considered one study to lead to speculative conclusions; another to lead to an association of no statistical significance; and a meta-analysis to be flawed.

Dr. Borak had several criticisms for Dr. Goldstein that concerned benzene toxicity. He first stated that references cited by Dr. Goldstein did not support this proposition. This included studies by Dr. Kirekilit, et al., Dr. Constantini, et al., Joshi and Yng, et al.

Dr. Borak also criticized Goldstein concerning Goldstein's viewpoint upon mechanistic evidence. He stated that this has been Dr. Goldstein's "own editorial since 1990." He stated that this was not based upon epidemiology.

Dr. Borak was also critical concerning Goldstein's viewpoint that obesity would increase the metabolism for benzene and therefore subject Moreland to a higher risk of multiple myeloma. He first testified that this was at odds with the opinions of Dr. Pineda and Dr. Parmet. He also found a lack of scientific support for this proposition based upon epidemiological studies. The studies that concern this found an increased metabolism in low dose patients, where he would

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expect to see it in “high dose” cases. He also testified that Goldstein’s theory was inconsistent with Goldstein’s own book chapter (exhibit 8 from the Goldstein deposition). More specifically, Dr. Borak stated, it was deficiency of NQ01 with the increase of 2E1 that would be important, not merely the increase of 2E1 that would increase metabolism. He stated that Dr. Goldstein ignored the presence of NQ01 in this theory for purposes of testimony in this particular case. Also, obesity is not the only factor that incites 2E1. Alcohol also incites 2E1 “a bit.” One, under this theory would expect multiple myeloma in alcoholics, though the incidences of alcohol are both reduced in cases of acute myelogenous leukemia and multiple myeloma. The presence of obesity increases the risk of acute myelogenous leukemia and multiple myeloma. One would need, he stated, an increase in 2E1 and a decrease in NQ01 for the Goldstein theory to be correct.

**Investigation by Eagle Picher. Testimony of Dennis Chiappetti, Production Supervisor and Bill Ideker, Directory of Health, Safety and Environment**

William Ideker testified on behalf of Employer. Mr. Ideker testified that he has been the Director of Health, Safety and Environment for Eagle Picher Technologies in Joplin since 1992, and that his department is responsible for the monitoring of worker safety at the C and Porter Street facility where Mr. Moreland was employed. Mr. Ideker testified that when he became aware that Mr. Moreland had claimed that his multiple myeloma was caused by his exposure to benzene, he had conducted a review of the chemicals that were used in Building 4 where Mr. Moreland worked, and that his review of those chemicals revealed that no benzene was used during Mr. Moreland’s work history in Building 4, nor was benzene used in any other work area where Mr. Moreland worked at the C and Porter Street Eagle Picher facility. Mr. Ideker compiled two document records that showed that benzene was not utilized in Mr. Moreland’s workplaces.

A document entitled Hazardous Waste Disposal records was submitted as Employer’s Exhibit 3, which showed that from 1987 through 2005 the disposal of benzene had been recorded only three times, twice in 1999 and once in 2004. Mr. Ideker testified that the benzene that had been disposed of, as indicated in the Hazardous Waste Disposal Records, was utilized in small quantities by engineers in a building in which Mr. Moreland had not worked.

As a result of the contents of these records, Mr. Ideker testified that Mr. Moreland was not exposed to benzene during his employment at Eagle Picher in Joplin.

Mr. Ideker testified that despite the fact that he knew Mr. Moreland well enough that they addressed one another by their nicknames, he was not aware until Mr. Moreland filed his Claim for Compensation in December 2007 that Mr. Moreland claimed that his multiple myeloma was caused by his exposure to benzene during his employment at Eagle Picher.

The Eagle Picher safety director, William Ideker, requested that chemist, Dennis Chiappetti, conduct the investigation concerning the work environment of Howard Moreland in Building 4. The investigation consisted of the retrieval of various business documents retained by Eagle Picher over the years, or prepared specifically for this claim.

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The first and most significant portion of Chiappetti's investigation was the retrieval of an Analytical Procedures Handbook, employer's exhibit 2, which concerned the components of nickel hydrogen batteries. The lab analysis was no longer available. Chiappetti identified various chemicals identified in the analytical process. They included potassium hydroxide; nitric acid; hydrochloric acid; ammonium chloride; EDTA; magnesium sulfate; arochrome black tea; acetic acid; hydrozene sulfate; sodium nitrate; phenylthalline; methyl orange; and sulfuric acid. Phenylthalline is a benzene-related chemical. Chiappetti did not produce the procedures or analysis for nickel cadmium which was contained in Building 4.

Chiappetti identified three material safety data sheets (employer's exhibits 11, 12, and 13), which reflected chemicals used in the glue used in the plastic shop. Chiappetti testified that none of the glue included benzene. However, Exhibit 13 shows the presence of diamethylamine which is a benzene-related chemical that has a benzene ring. This chemical is used as an accelerator to "set up" the glue.

Chiappetti also identified a certification for raw rubber and material safety data sheets for raw rubber (employer's exhibits 9 and 10). He did not find benzene as a chemical or a benzene-related chemical within these documents.

A fire destroyed building 54 (adjacent to Building 4) in September 1991. This was significant because building 54 contained the Document Control Center. The Document Control Center would have included the analytical procedure for nickel cadmium, nickel hydrogen, material safety data sheets, certificate of manufacture for raw rubber and material safety data sheets for raw rubber. The Eagle Picher witnesses could not verify that these documents were either complete, or that there were other documents that would bear upon material issues.

Next, Eagle Picher could not produce a manufacturing process for either nickel hydrogen batteries or nickel cadmium batteries.

In addition, Eagle Picher produced no field studies or lab results to indicate the level of particulates from hazardous chemicals during the time in which Moreland worked in building number 4.

## **RULINGS OF LAW**

### **Statute of Limitations**

According to Missouri Workers' law an employer must file a report of injury within thirty (30) days from the date of injury.

"Every employer or his insurer in this state, whether he has accepted or rejected the provisions of this chapter, shall within thirty days after knowledge of the injury, file with the division under such rules and regulations and in such form and detail as the division may require, a full and complete report of every injury or death to any employee for which the employer would be liable to furnish medical aid, other than immediate first aid which does not result in further medical

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treatment or lost time from work, or compensation hereunder had he accepted this chapter, and every employer or insurer shall also furnish the division with such supplemental reports in regard thereto as the division may require.” Section 287.380.1.

The statute of limitations is determined as follows:

“Except for a claim for recovery filed against the second injury fund, no proceedings for compensation under this chapter shall be maintained unless a claim therefor is filed with the division within two years after the date of injury or death, or the last payment made under this chapter on account of the injury or death, except that if the report of the injury or the death is not filed by the employer as required by Section 287.380, the claim for compensation may be filed three years after the date of injury, death or last payment made under this chapter on account of the injury or death.” *Section 287.430*

This limitation applies to claims for an occupational disease:

“The statute of limitation referred to in Section 287.430 shall not begin to run in cases of occupational disease until it becomes reasonably discoverable and apparent that an injury has been sustained related to such exposure.” Section 287.063.3

I have stated my finding that Mr. Moreland informed his supervisor, Jeff Dermott, on July 29, 2005, that he suffered multiple myeloma and that it was due to his exposure to chemicals at work. The parties stipulated that Eagle Picher did not file a Report of Injury until after Moreland filed his Claim for Compensation on December 17, 2007.

The Missouri Court of Appeals-Eastern District has held that a statute of limitation is three (3) years when the employer fails to file a Report of Injury. This applies to occupational diseases. *Gillam v. General Motors Corporation*, 913 S.W.2d 81 (Mo.App.E.D. 1995). The court in *Gillam* noted the statutes previously cited here and then ruled that the statute of limitations was three (3) years rather than two (2) while stating the following:

“Here, employer acknowledges it never filed an injury report. On appeal, employer claims this was due to employee’s failure to give notice of a work related disease. However, the parties stipulated at the administrative hearing that notice of work related disease was not an issue. Employer has waived this point on appeal. Further, employer does not claim any prejudice from lack of notice. Moreover, employer had reasonable notice of employee’s condition. Employee told his foreman about his foot pain and went to the medical dispensary of the company, but nothing was done. In 1988 and 1989, he told his foreman he “was having a lot of trouble” and he needed surgery. He had three foot surgeries in 1989 and missed about ten months of work. Employee’s dispensary records included references to these foot problems. Employer had notice and opportunity

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to investigate the cause of disability, but failed to do so. The three year time limitation is applicable since employer failed to file an injury report. The statute began to run September 1989 and would have expired in September 1992. Employee timely filed his claim on January 14, 1992.” *Gillam* at page 83.

I find that claimant timely filed his claim for compensation in this case.

### **Occupational Disease**

The Missouri Workers’ Compensation law underwent a significant amendment on August 28, 2005. Here, however, this claim arose on July 29, 2005. The legislation in effect on that date, which is substantive in nature, and not procedural, governs this claim. *Lawson v. Ford Motor Company*, 217 S.W.3d 345 (Mo.App.E.D. 2007). Several familiar principles, therefore, deserve mention.

The fundamental purpose of the Missouri Workers’ Compensation law is to place upon industry the losses suffered by employee resulting from injuries arising out of and in the course of employment. The law is to be broadly and liberally interpreted and is intended to extend its benefits to the largest possible class. Any question as to the right of an employee to compensation must be resolved in favor of the injured employee. *Cherry v. Powdered Coatings*, 897 S.W.2d 64 (Mo.App.E.D. 1995); *Wolfgeher v. Wagner Cartage Services, Inc.*, 646 S.W.2d 781 (Mo. banc 1983). A liberal construction cannot be applied in order to excuse a lacking element. *Johnson v. City of Kirksville*, 855 S.W.2d 396 (Mo.App.W.D. 1983).

The claimant, however, need not establish the elements of his claim on the basis of absolute certainty. It is sufficient if the claimant shows them to a reasonable probability. “Probable,” for the purpose of determining whether a workers’ compensation claimant has shown the elements by reasonable probability, means founded on reason and experience, which inclines the mind to believe, but leaves room for doubt. See, for example, *Cook v. St. Mary’s Hospital*, 939 S.W.2d 934 (Mo.App.W.D. 1997); *White v. Henderson Implement Company*, 879 S.W.2d 575 (Mo.App.W.D. 1994); and *Downing v. Willamette Industries, Inc.*, 895 S.W.2d 650 (Mo.App.W.D. 1995). All doubts must be resolved in favor of the employee and in favor of coverage. *Johnson v. City of Kirksville* at page 398.

The elements for an occupational disease are set forth in Section 287.067.1:

“In this chapter the term “occupational disease” is hereby defined to mean, unless a different meaning is clearly indicated by the context, an identifiable disease arising with or without human fault out of and in the course of the employment. Ordinary diseases of life to which the general public is exposed outside of the employment shall not be compensable, except where the diseases follow as an incident of an occupational disease as defined in this section. The disease need not to have been foreseen or expected but after its contraction it must appear to have had its origin in a risk connected with the employment and to have flowed from that source as a rational consequence.”

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A claimant seeking benefits for an occupational disease need only, as with other claims, submit medical evidence which establishes a probability that the working condition caused the disease. *Jacobs v. City of Jefferson*, 991 S.W.2d 693 (Mo.App.W.D. 1999). A claimant is required to prove that there was an exposure to a disease which was greater than or different from that which affected the general public and that there was a recognizable link between the disease and some distinctive feature of the job which was common to all jobs of that sort. *Causey v. McCord*, 763 S.W.2 155 (Mo.App.S.D. 1998); *Sellers v. Transworld Airlines, Inc.*, 752 S.W.2d 413 (Mo.App.W.D. 1988); *Estes v. Noranda Aluminum, Inc.*, 574 S.W.2d 34 (Mo.App. 1978). The first element is straightforward. Multiple myeloma, as defined by Drs. Pineda, Parmet and Goldstein, is an identifiable disease based upon their respective definitions of this disease in their depositions. Dr. Borak pointed out multiple discrepancies that he believed were contained in the opinions and sources relied upon by Dr. Pineda-Roman, Dr. Parmet, and Dr. Goldstein. However, after reviewing the evidence and testimony involved I find that the opinions of these doctors are credible and persuasive and certainly provide substantial and competent evidence to meet claimant's burden of proof of proving the probability that claimant's work exposure caused the disease. Dr. Borak bolstered claimant's case by pointing out that these studies, while not able to prove with medical and scientific certainty that these chemicals and particularly benzene caused multiple myeloma, they do show that exposure to these chemicals, including benzene, could cause multiple myeloma and this satisfies the burden of proof required of claimant in this case.

The second question is whether there was an exposure to the disease which was greater than or different from that which affected the public generally. A claimant need only prove an exposure to a chemical agent in a claim for occupational disease based upon "reasonable probability." *Barr v. Vickers, Inc.*, 648 S.W.2d 577 (Mo.App.S.D. 1983). This was established by the testimony of the former Eagle Picher employees and Moreland. I find the testimony of these individuals to be both credible and persuasive. There was testimony and evidence (Exhibit 13) that benzene was included in the glue used for plastics. Moreland testified that there was also benzene mentioned on the menu for raw rubber. Benzene-related compounds, such as phenyl thaline were even identified in the analytical process for nickel hydrogen. These chemicals would emanate into fumes and dust from the plastic shop that would then infiltrate the atmosphere throughout Building Number 4 where the Claimant worked. Witnesses identified chemicals used that would emanate from open vats or the rubber room to include benzene, nickel and cadmium. Trichloroethylene was also used as a widespread solvent. These former employees of Eagle Picher and Moreland testified to this and Eagle Picher was not able to produce witnesses or persuasive documentary evidence to counter this testimony. At Bill Ideker's deposition Eagle Picher presented an MSDS for benzene and coworkers testified to it being used in various processes in Building 4.

This exposure is clearly greater than which affects the public generally. Eagle Picher argues it is necessary that Claimant must prove through airborne particulate testing the amount of these chemicals to which employees were exposed. However in a case recently decided by the Commission no "testing" was required to prove exposure. *Bennett v. Kansas City Power and Light*. Nevertheless, Dr. Parmet testified that even the smallest amount of liquid, such as a drop, can, mixed with other solid solutions, create fumes or gases that immediately exceed permissible

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levels.

The third question is whether there is a recognizable link between the disease and some distinctive feature of the claimant's job which was common to all jobs of that sort. That is true here. Moreland, as other employees, worked for approximately 10 years in Building 4 where he was exposed to fumes and dust that contained benzene and other toxic chemicals. He worked, as other employees, in the nickel cadmium/nickel hydrogen area and all were exposed to fumes that came from open vats holding hazardous chemicals. Indeed, a co-worker named Jim Morgan passed away due to multiple myeloma.

Dr. Borak was not persuasive in his proposition that obesity, by itself, contributes to the development of multiple myeloma.

I find and conclude that Howard Moreland suffered an occupational disease as defined in *Section 287.067.1*.

### **Past Medical Expense**

The parties have stipulated to the amount of past medical. I find the employee's claim compensable and therefore I hereby award past medical expenses in the amount of \$752,021.08 and order that this amount be paid to Claimant.

### **Nature and Extent of Permanent Disability**

Dr. Parmet stated in his report that Mr. Moreland is permanently and totally disabled due to his myeloma and its treatment. I find that Howard Moreland is permanently and totally disabled as a result of the chemical exposure at Eagle Pitcher and resulting multiple myeloma and its treatment. I order the employer/insurer to pay Howard Moreland the amount of \$397.45 per week for the remainder of his lifetime pursuant to §287.200. I also order the employer/insurer to begin these payments as of August 1, 2005, which is the last day Claimant worked. As a result of this finding the issue of temporary total disability is moot.

### **Future Medical Treatment**

I find Howard Moreland is entitled to medical treatment which cures or relieves the effects of the myeloma, chronic kidney disease, neuropathies in his hands, neuropathies in his feet and skin cancers. This is based upon the testimony of Dr. Pineda, Dr. Parmet and Dr. Hamlett. These physicians testified without contradiction concerning the connection of either myeloma, chemotherapy or stem cell transplant to these various disorders. I order the employer/insurer, Eagle Pitcher, to pay for this future medical care pursuant to §287.140.

### **Statutory Penalty**

I find that a penalty of fifteen percent (15%) of all past medical expenses, past permanent total disability benefits and future permanent total disability benefits be awarded pursuant to *Section 287.120.4*, based upon violations of *Sections 292.300, 292.310 and 292.320*.

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The Division has jurisdiction to impose a penalty of fifteen percent (15%) of compensation awarded upon Section 287.120.4:

“Where the injury is caused by the failure of the employer to comply with any statute in this state or any lawful order of the division or the commission, the compensation and death benefit provided for under this chapter shall be increased fifteen percent.”

Three statutes are at issue here, Section 292.300, 292.310 and 292.320. They state as follows:

“That every employer of labor in this state engaged in carrying on any work, trade or process which may produce any illness or disease peculiar to the work or process carried on, or which subjects the employee to the danger or illness or disease incident to such work, trade or process to which employees are exposed shall for the protection of all employees engaged in such work, trade or process adopt and provide approved and affective devices, means or methods for the prevention of such industrial or occupational diseases are incident to such work, trade or process.” Section 292.300.

“The carrying on of any process or manufacture or labor in this state in which antimony, arsenic, brass, copper, lead, mercury, phosphorus, zinc, alloys or salts or any poisonous chemicals, minerals, acids, fumes, vapors, gases or other substances are generated or used, employed or handled by the employees in harmful quantities or under harmful conditions or come into contact with in a harmful way are hereby declared to be especially dangerous to the health of the employee.” Section 292.310

“Every employer in this state to which sections 292.300 to 292.440 apply shall provide for and place at the disposal of the employees so engaged, and shall maintain in good condition without cost to the employees, working clothes to be kept and used exclusively by such employees while at work and all employees therein shall be required at all times while they are at work to use and wear such clothing; and in all processes of manufacture or labor referred to in this section which are productive of noxious or poisonous dust, adequate and approved respirators shall be furnished and maintained by the employer in good condition and without cost to the employees, and such employees shall use such respirators at all times while engaged in any work productive of noxious or poisonous dust.” Section 292.320

These three statutes from Chapter 292 are a portion of the Occupational Disease Act §§ 292.300 to 292.400. The statutes are not constitutionally indefinite to require safety devices and are not void for vagueness. *Boll v. Condie-Bray Glass and Paint Company*, 11 S.W.2d 48 (Mo. 1928). The definition of what constitutes an approved and adequate device to protect employees against occupational diseases is not necessary to render these statutes enforceable. See *Boll*,

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*supra*. Only one penalty may be imposed even though more than statute may have been violated. *Akers v. Warson Garden Apartments*, 961 S.W.2d 50 (Mo. 1998). The penalty applies to disability benefits and the cost of medical treatment. *Martin v. Star Cooler Corp.*, 484 S.W.2d 32 (Mo.App. 1972). These three statutes were the subject of *McGhee v. W.R. Grace and Company*, 312 S.W.3d 447 (Mo.App.S.D.2010).

Evidence of the work conditions in Building 4 was uncontroverted. Six employees testified that no employees wore respirators or any other kind of respiratory protection in Building 4 from 1984 until 1994. The evidence was uncontradicted that ventilation was quite poor until approximately 1992. It was so poor that the vents over the open vats were not even capable of picking up a feather. Large fans used to create a breeze merely pushed fumes that hovered over one vat to another. This had the effect of putting employees at risk with chemicals with which they were not even directly working.

Eagle Picher presented no evidence that its employees were protected from hazardous materials, as required in the three statutes quoted above, from 1984 to the early 1990s. It, instead, argued that employees were not exposed to the hazards of these chemicals, though it has no documented business records to indicate such. Further, Eagle Picher even produced business records which indicated that employees worked in the proximity of benzene-related chemicals which are more dangerous than benzene itself (e.g., benzene[a] pyrene and benzene[b] pyrene). Further, it is obvious from the testimony of the Eagle Picher employees and the testimony of Dr. Parmet that the limit of one part per million was significantly violated. Visitors to the plant, upon smelling these fumes, became so ill that they could not stay.

I hereby order the Employer/Insurer pay an additional 15 percent of all benefits awarded herein to Claimant as a result of safety violations pursuant to §287.120.4

I also order attorney's fees of 25 percent of all amounts awarded herein to Patrick Platter. This shall constitute a lien upon this award.

Date: December 28, 2010

Made by: /s/ Karen Wells Fisher  
Karen Wells Fisher  
Administrative Law Judge  
Division of Workers' Compensation

A true copy: Attest:

/s/ Naomi Pearson  
Naomi Pearson  
Division of Workers' Compensation