**IN THE WORKERS' COMPENSATION COURT OF THE STATE OF MONTANA**

**2005 MTWCC 34**

**WCC No. 2005-1292**

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**ELDON FLEMING**

**Petitioner**

**vs.**

**INTERNATIONAL PAPER COMPANY,**
**as successor-in-interest to**
**CHAMPION INTERNATIONAL COMPANY,**
**and**
**LIBERTY NORTHWEST INSURANCE CORPORATION**

**Respondents.**



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DECISION AND ORDER DENYING LIBERTY NORTHWEST'S MOTIONS TO DISMISS AND FOR SUMMARY JUDGMENT

**Summary:** The claimant alleges he suffers from asbestos-related lung disease as a result of his employment at a Libby, Montana, lumber mill from 1960 to May 28, 1998. The mill was owned by Champion International Company until November 1, 1993. It was thereafter owned by Stimson Lumber Company, which is insured by Liberty Northwest Insurance Corporation. Liberty moves to dismiss the petition, arguing that (1) the claimant failed to mediate his claim against it; (2) the petition is barred by the statute of limitations governing petitions to the Workers' Compensation Court; (3) the claimant is judicially estopped from pursuing a claim against Liberty; and (4) the latency period for the claimant's lung disease is so long that his disease cannot be legally attributed to his employment with Stimson.

**Held:** (1) The claimant filed for mediation but the mediation was derailed by Liberty. The claimant is entitled to complete the mediation process and the Court has jurisdiction to order the Department of Labor and Industry to do so and to retain jurisdiction over his petition pending such completion. (2) The statute of limitations, § 39-71-2905(2), MCA (1997-2003), was tolled by the claimant's filing for mediation and has not run. (3) The filing of a district court complaint against other parties who allegedly were responsible for the claimant's exposure to asbestos is not inconsistent with his claim that asbestos at his workplace contributed to or caused his asbestos lung disease; none of the elements for a judicial estoppel are met. (4) Liberty's evidence concerning the latency period for

asbestos lung disease does not demonstrate as an uncontroverted matter that the claimant was not injuriously exposed to asbestos during his employment with Stimson.

**Topics:**

**Mediation: Right to Mediation.** A claimant who files for mediation has a right to have his claim mediated by the Department of Labor and Industry. Since mediation is a prerequisite to filing a petition with the Workers' Compensation Court, the Court has jurisdiction to compel mediation.

**Mediation: Compelling Mediation.** A claimant who files for mediation has a right to have his claim mediated by the Department of Labor and Industry. Since mediation is a prerequisite to filing a petition with the Workers' Compensation Court, the Court has jurisdiction to compel mediation.

**Jurisdiction: Workers' Compensation Court: Scope.** The Workers' Compensation Court has inherent jurisdiction to assure access to the Court. Since mediation is a prerequisite to filing a petition with the Workers' Compensation Court, the Court has jurisdiction to compel mediation.

**Jurisdiction: Workers' Compensation Court: Mediation Requirement.** While mediation is required before a claimant may petition the Workers' Compensation Court for benefits, where a claimant has requested mediation and mediation is derailed through no fault of the claimant, the Court has jurisdiction to entertain a petition for benefits and order the Department of Labor and Industry to complete mediation so that the claimant may proceed with his petition.

**Limitations Periods: Workers' Compensation Court Petitions.** The statute requiring the claimant to petition the Workers' Compensation Court for benefits within two years of an insurer's denial of benefits, § 39-71-2905(2), MCA (1997-2003), is tolled during mediation. *See Preston v. Transportation Ins. Co.*, 2004 MT 339, 324 Mont. 225, 102 P.3d 527.

**Limitations Periods: Tolling.** The statute requiring the claimant to petition the Workers' Compensation Court for benefits within two years of an insurer's denial of benefits, § 39-71-2905(2), MCA (1997-2003), is tolled during mediation. *See Preston v. Transportation Ins. Co.*, 2004 MT 339, 324 Mont. 225, 102 P.3d 527.

**Constitutions, Statutes, Rules, and Regulations: Montana Code Annotated: 39-71-2905(2), MCA (1997-2003).** The statute requiring the claimant to petition the Workers' Compensation Court for benefits within two years of an insurer's denial of benefits, § 39-71-2905(2), MCA (1997-2003), is tolled during mediation. *See Preston v. Transportation Ins. Co.*, 2004 MT 339, 324 Mont. 225, 102 P.3d 527.

**Cases Discussed: *Preston v. Transportation Ins. Co.*, 2004 MT 339, 324 Mont. 225, 102 P.3d 527.** The statute requiring the claimant to petition the Workers' Compensation Court for benefits within two years of an insurer's denial of benefits, § 39-71-2905(2), MCA (1997-2003), is tolled during mediation.

**Limitations Periods: Statutes of Repose.** The statute requiring the claimant to file a petition within two years of a denial of benefits, § 39-71-2905(2), MCA (1997-2003), is a statute of limitations, not a statute of repose.

**Constitutions, Statutes, Rules, and Regulations: Montana Code Annotated: 39-71-2905(2), MCA (1997-2003).** The statute requiring a claimant to file a petition within two years of a denial of benefits, § 39-71-2905(2), MCA (1997-2003), is a statute of limitations, not a statute of repose.

**Limitations Periods: Statutes of Repose.** Use of the word "must" in a statute governing the time in which an action must be commenced does not make the statute one of repose rather than one of limitations. Only where the language of the statute indicates it overrides other limitations periods and/or unequivocally indicates that it cannot be tolled will it be held to be a statute of repose.

**Limitations Periods: Retroactivity.** Unless some other time is indicated, a statute adopting a new limitations period for bringing an action, or amending an existing statute of limitations, applies to all proceedings that are brought thereafter even though the cause of action arose prior to passage. Statutes of limitations are procedural and not subject to the rule precluding retroactive application of statutes which do not expressly provide for retroactivity. *See Fisher v. First Citizens Bank,* 2000 MT 314, 302 Mont. 473, 14 P.3d 1228.

**Estoppel and Waiver: Judicial Estoppel.** To judicially estop a party, four elements must typically be met. Those elements are: (1) the estopped party had knowledge of the facts at the time he or she took the original position; (2) the estopped party succeeded in maintaining the original position; (3) the

position presently taken is inconsistent with the original position; and (4) the original position misled the adverse party so that allowing the estopped party to change its position would injuriously affect the adverse party.

**Estoppel and Waiver: Judicial Estoppel.** Where a claimant may have been exposed to multiple sources of asbestos, some or all of which may have contributed to his asbestos-related disease, he is not judicially estopped from pursuing a petition for occupational disease benefits even though he is pursuing a district court action against non-employers allegedly responsible for some of his exposure. The Rules of Civil Procedure permit a party to join multiple defendants who are potentially liable for his injuries and to pursue his action in the alternative. Since the Workers' Compensation Court has exclusive jurisdiction over occupational disease claims, a claimant of necessity may be required to file both a district court action and a Workers' Compensation Court petition to achieve the same end.

**Occupational Disease: Last Injurious Exposure.** Where a claimant is exposed to asbestos which gives rise to lung disease, the exposure occurred over a period of years, and the exposure involved more than one employer, the insurer for the employment at which the claimant was "last injuriously exposed" is solely liable for his disease.

**Occupational Disease: Last Injurious Exposure.** The last injurious exposure rule applicable to sequential injuries or diseases is different from the last injurious exposure rule applicable where the claimant suffers a single disease from long-term exposure to fumes, dust, or chemicals. *Caekaert v. State Compensation Mut. Ins. Fund,* 268 Mont. 105, 111, 885 P.2d 495, 499 (1995) and *Liberty Northwest Ins. Corp. v. Champion Int'l. Corp.,* 285 Mont. 76, 945 P.2d 433 (1997), are distinguished.

**Occupational Disease: Last Injurious Exposure.** In applying the last injurious exposure rule, difficulty may arise in determining the degree of exposure necessary to find the exposure injurious. Montana courts have not addressed this problem and have not adopted a standard for determining the degree of exposure necessary. According to Larson's Workers' Compensation Law treatise, "[t]raditionally, courts applying the last injurious exposure rule have not gone on past the original finding of some exposure to weigh the relative amount or duration of exposure under various carriers and employers." § 153.02[7][a] at 153-19. However, some courts have adopted more stringent requirements.

**Summary Judgment: Disputed Facts.** The insurer is not entitled to summary judgment based on the fact that asbestos disease has a long latency period where the evidence upon which it relies does not show as an uncontroverted matter that the claimant's exposure to asbestos at the insured's place of employment was so short and trivial as to be wholly non-contributory to his disease. The insurer's proof is insufficient to entitle it to summary judgment under any of the standards identified in Larson's Workers' Compensation Law treatise as governing the degree of exposure necessary to impose liability under the last injurious exposure doctrine.

¶1 This is an asbestos case. The petitioner (claimant) has been diagnosed with asbestosis-related lung disease. In his petition he attributes his disease to exposure to asbestos while working at a lumber mill in Libby, Montana. He worked at the mill from 1960 to May 1998. From 1960 to November 1993, the mill was owned by Champion International Corporation (Champion). In November 1993, the mill was sold to Stimson Lumber Company (Stimson), which operated it thereafter. Sometime after the sale, Champion merged with or was acquired by International Paper Company (International Paper) but will be generally referred to hereinafter as "Champion" rather than International Paper.

¶2 Champion was self-insured during the claimant's employment, or at least it was at the time it sold the mill to Stimson. With respect to the present claim, Stimson is insured by Liberty Northwest Insurance Corporation (Liberty). The claimant is seeking, in the alternative, benefits from Champion or Liberty.

Liberty's Pending Motions

¶3 Liberty moves in the alternative to dismiss the petition and for summary judgment. (Liberty's Motion to Dismiss (Rule 12(b)(6)) and Motion for Summary Judgment and Supporting Brief.) In its motion, Liberty tenders four grounds in support of its request that the petition be dismissed. Those grounds, as restated, are:

¶ 3a The claim against it has not been mediated.

¶ 3b The claim is barred by the two-year statute of limitation set out in section 39-71-2905(2), MCA (1997-2003).

¶ 3c Based on a district court action commenced against the State of Montana, Burlington Northern Santa Fe Railway Company, Robinson Insulation Company, John Swing, and unnamed "Does", the claimant is judicially estopped from claiming benefits on account of his work at the lumber mill.

¶ 3d The latency period for asbestosis is so long that the claimant's current disease could not be due to his exposure while working for Stimson.

Admitted and Uncontested Facts

¶4 The facts material to Liberty's motions are found in the non-controverted allegations of the petition; affidavits of Gary Schild, Cindy Brown Felton, and Ed Roberts, to which numerous exhibits are attached; a copy of a complaint filed on behalf of the claimant in the Montana Eighth Judicial District Court; and exhibits attached to Petitioner's Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment.[1] The uncontroverted facts are as follows:

¶ 4a The claimant was continuously employed at a lumber mill near Libby, Montana, from 1960 through May 28, 1998. (Petition for Hearing ¶ 1 and Liberty Northwest's Response to Petition for Hearing at 2.[2])

¶ 4b The lumber mill was owned and operated by Champion from 1960 until November 1, 1993. Champion has since been merged with or been acquired by International Paper.

¶ 4c On November 1, 1993, the Libby mill was purchased by Stimson. Stimson began operating the mill on November 5, 1993. (Affidavit of Ed Roberts at 1.)

¶ 4d Upon purchasing the Libby mill, Stimson rehired the claimant as its employee. The claimant continued working at the mill until he retired on May 28, 1998, a period of approximately four and a half years. (*Id.* at 2.)

¶ 4e On December 4, 2001, Liberty, which insures Stimson, received a written claim for compensation from the claimant. The claim was signed on

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[1]While not verified by affidavit, the authenticity of the documents has not been disputed by either of the respondents and in any event consist of correspondence and other documents associated with the claimant's request for mediation. The documents were generated by the parties' attorneys or are of the type the attorneys would have personal knowledge of.

[2]The allegation of employment and the time frame of employment are not denied by either of the respondents in their written responses to the petition.

November 26, 2001,[3] and stated that the claimant was suffering from "[l]ung disease caused by years of asbestos exposure" while working at Stimson. (Affidavit of Gary Schild, Ex. A.)

¶ 4f Liberty's claims adjuster initially requested medical records respecting the claim. (*Id.,* Exhibit B at 2.) Thereafter, on March 11, 2003, Liberty denied liability for the claim. (*Id.* at 1.)

¶ 4g On March 22, 2004, the claimant submitted a similar claim to Champion, alleging that his asbestos-related lung disease arose from his employment during the Champion years. Champion denied the claim on April 1, 2004. (Petition for Hearing ¶ IV.)[4]

¶ 4h On September 21, 2004, the claimant underwent a medical panel evaluation by Dr. Richard L. Sellman. In his report, Dr. Sellman opined that the claimant was suffering from "pleural thickening" caused by his exposure to asbestos during his employment; however, Dr. Sellman opined that the "pleural thickening is in no way responsible for his dyspnea on exertion, and this does not equate to the diagnosis of asbestosis."[5] (Ex. 3 to Petitioner's Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment at 3.)

¶ 4i Sometime prior to January 26, 2005, the claimant filed a request for mediation with respect to his claim against Champion. The request was filed with the Workers' Compensation Mediation Unit of the Department of Labor and Industry (Department). (*See* Ex. 5 to Petitioner's Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment.) Mediation as to Champion apparently took place but was unsuccessful.

¶ 4j On February 16, 2005, the claimant submitted a written request for mediation with respect to Liberty's denial of liability. (Affidavit of Gary Schild, Ex. C.) Mediation was scheduled for March 16, 2005. (Ex. 1 to Petitioner's

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[3]The Affidavit of Gary Schild, to which the claim is attached, states that the claimant's signature was dated November 29, 2001, however, I read the date as November 26, 2001.

[4]The allegation concerning submission of the claim to Champion and its denial of liability are not controverted by Champion in its response to the petition.

[5]This fact is set forth solely for historical purposes only and not to indicate that the doctor's opinions are undisputed.

Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment.)

¶ 4k On March 2, 2005, Liberty's attorney requested that mediation scheduled for March 16, 2005, be vacated until the claimant had undergone an occupational disease medical panel examination with respect to the claim against Liberty. In that letter, Liberty's attorney specifically noted that a panel evaluation was necessary under section 39-72-602, MCA, so that Liberty could "review the report and respond" to it.[6] (Affidavit of Gary Schild, Ex. D and Petitioner's Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment, Ex. 2.)

¶ 4l On March 4, 2005, the Mediation Unit vacated the scheduled mediation "until the Occupational Disease evaluation has been completed." (Affidavit of Gary Schild, Ex. E and Petitioner's Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment, Ex. 4.)

¶ 4m The claimant thereafter requested a medical panel examination;

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[6]The occupational disease panel provisions in effect at the time of the mediation request provided as follows:

**39-72-602. Insurer may accept liability -- procedure for medical examination when insurer has not accepted liability.** (1) An insurer may accept liability for a claim under this chapter based on information submitted to it by a claimant.

(2) In order to determine the compensability of claims under this chapter when an insurer has not accepted liability, the following procedure must be followed:

(a) The department shall direct the claimant to an evaluator on the list of physicians for an examination. The evaluator shall conduct an examination to determine whether the claimant is totally disabled and is suffering from an occupational disease. In the case of a fatality, the evaluator shall examine the records to determine if the death was caused by an occupational disease. The evaluator shall submit a report of the findings to the department.

(b) Within 7 working days of receipt, the department shall mail the report of the evaluator's findings to the insurer and claimant.

(c) Upon receipt of the report, if a dispute exists over initial compensability of an occupational disease, it is considered a dispute that, after mediation pursuant to department rule, is subject to the jurisdiction of the workers' compensation court.

however, the Department, which is responsible for arranging for such examinations, denied the request. In its denial, the Department indicated that the examination done in September 2004, satisfied the occupational disease panel requirements of section 39-72-602, MCA. The letter further stated:

> The role of the occupational disease panel is to determine if a claimant is totally disabled and is suffering from an occupational disease. The panel physician does not determine which employment is responsible for the occupational disease.
>
> Since the lung condition has been paneled and the information provided to the panel doctor covered the time from 1992 thru 2002, it appears the requirements set for the [sic] in Section 39-72-602, MCA.[sic], have been met by the Department. Therefore, an Occupational Disease panel Examination with [sic] not be scheduled.

(Affidavit of Gary Schild, Ex. F, and Petitioner's Response to Liberty NW's Motion to Dismiss and Motion for Summary Judgment, Ex. 7.)

¶ 4n On April 13, 2005, Liberty notified the claimant and the Department that it "continues to deny Mr. Fleming's claim against Stimson even in light of the OD evaluation that was done on the Champion claim." (Affidavit of Gary Schild, Ex. G.)

¶ 4o As of May 4, 2005, mediation with respect to the claim against Stimson was never rescheduled. (Affidavit of Cindy Brown Felton.) While mediation has not been completed, there is no evidence that the petition for mediation with respect to the claim against Liberty was dismissed.

¶ 4p Meanwhile, on April 14, 2005, the claimant filed his petition with this Court.

## DECISION

¶5 A motion to dismiss will be granted where the facts alleged in the petition show that no claim for relief can be stated under any legal theory, *Duffy v. Butte Teachers' Union*, No. 332, AFL-CIO, 168 Mont. 246, 253, 541 P.2d 1199, 1203 (1975), or where they affirmatively demonstrate that there is an insuperable bar to recovery, such as the statute of limitations, *Beckman v. Chamberlain*, 673 P.2d 480, 482 (Mont. 1983). While Liberty

captions its motion as a motion to dismiss and an alternative motion for summary judgment, the motion to dismiss is ultimately subsumed in the motion for summary judgment. I therefore apply summary judgment standards in disposing of the motions.

¶6 "Summary judgment is an extreme remedy and should never be substituted for trial if a material factual controversy exists." *Spinler v. Allen*, 1999 MT 160, ¶ 16, 295 Mont. 139, 983 P.2d 348 (1999). On the other hand, if the facts material to the motion are undisputed and entitle a party to summary judgment, then summary judgment is proper. *Mogan v. Cargill, Inc.*, 259 Mont. 400, 403, 856 P.2d 973, 975 (1993). What facts are material are determined by the substantive law applicable to the case. *DeVoe v. State*, 281 Mont. 356, 366, 935 P.2d 256, 263 (1997).

I. Failure to Mediate Defense

¶7 Liberty argues that the petition must be dismissed on account of the claimant's failure to mediate his claim against it. Mediation is mandatory, §§ 39-71-2408, -2905, MCA, and jurisdictional, *Peterson v. Montana Schools Group Ins. Auth.*, 2005 MTWCC 30.

¶8 The claimant attempted to comply with the mediation requirement by requesting mediation. His request was derailed at the insistence of Liberty and it is a bit disingenuous for Liberty to now attempt to derail the claimant's petition because mediation was never completed. Mediation should have proceeded.

¶9 As the facts set out earlier show, the claimant requested mediation on February 16, 2005. Liberty objected to the mediation, citing the claimant's failure to submit to an occupational disease panel evaluation required under section 39-72-602, MCA (2003), and earlier versions of that section.[7] Liberty did so despite the fact that the claimant had undergone a panel evaluation in connection with his claim against Stimson, an evaluation which found that the claimant was suffering from pleural lung thickening due to his exposure while working at the Libby lumber mill. Based on Liberty's objection, the scheduled mediation session was cancelled. The claimant then attempted to satisfy Liberty's objection by requesting a second panel evaluation but was rebuffed by the Department, which is responsible for arranging such evaluations, because he had already been examined. The Department reasoned that an evaluation with respect to the claim against Liberty was unnecessary in light of the fact that the evaluation previously done covered the claimant's long-term exposure at the mill, including his exposure when Champion owned the mill. (*See* ¶ 4.)

[7]The 2003 version of the section is set out in full in footnote 6.

¶10 The Department's determination that a second evaluation was unnecessary was clearly correct. However, for whatever reason, a mediation hearing was never rescheduled with respect to the claimant's February 16, 2005 mediation request and mediation has never been completed. That failure deprived the claimant of his statutory right to mediate his claim and prevented him from satisfying the requirement that a claim be mediated before petitioning the Workers' Compensation Court.

¶11 The failure of the Department to proceed with mediation gives this Court jurisdiction to order completion of the mediation necessary to enable it to adjudicate the merits of the claim. "Jurisdiction as applied to courts is the power or capacity *given by law* to a court to entertain, hear and determine the particular case or matter." *State ex rel. Johnson v. District Court of Eighteenth Judicial Dist.*, 147 Mont. 263, 267, 410 P.2d 933, 935 (1966) (quoting from *State ex rel. Bennett v. Bonner,* 123 Mont. 414, 425, 214 P.2d 747, 753 (1950). "Whenever jurisdiction is conferred, all the means necessary to carry the same into effect are provided." *State ex rel. Eisenhauer v. Second Judicial Dist. Court*, 54 Mont 172, 168 P. 522, 523 (1917). Based on those jurisdictional principles, this Court may issue such orders as necessary to preserve its jurisdiction over workers' compensation and occupational disease disputes and to assure that its jurisdiction over such suits is not frustrated by a failure or refusal of a party or agency to act.

¶12 Subsequent to the Court's drafting the above determination concerning the mediation defense, the Court received a Case Status Report from claimant's counsel. That report states that mediation as to Liberty has now been completed. In that light, it is unnecessary to order the Department to complete mediation. In light of the interruption and delay of the mediation proceeding, the Court had jurisdiction over the petition when it was filed. Since mediation is now complete, this Court has full jurisdiction to adjudicate the merits of the claim.

## II. Statute of Limitations

¶13 Liberty next urges that the claim against it must be dismissed in any event on account of the claimant's failure to bring his petition within two years of Liberty's denial of his claim.

¶14 The limitations period invoked by Liberty is found in section 39-71-2905(2), MCA (1997-2003), which provides:

> (2) A petition for hearing before the workers' compensation judge must be filed within 2 years after benefits are denied.

This provision was enacted in 1997 and became effective on July 1, 1997. 1997 Montana Laws, ch. 276, §§ 29 and 34(2). It was applicable to "injuries occurring on or after" the

effective date.

¶15 The claimant was allegedly subjected to asbestos exposure at his workplace until May 1998, which was after the limitations period became effective. However, he suffers from an alleged occupational disease rather than an injury. Since the applicability section for the 1997 amendments mentions "injury," the question arises as to whether the limitations period applies to the occupational disease claim in this case.

¶16 In *Penrod v. Hoskinson*, 170 Mont. 277, 552 P.2d 325 (1976), the Supreme Court held that a specific statute of limitations applicable to medical malpractice actions which was enacted by the 1971 legislature did not apply to malpractice which occurred prior to the effective date of the statute. In so finding, the Supreme Court relied on the general rule that statutes are not "retroactive unless expressly so declared." 170 Mont. 277, 281. Since the legislature had not expressly provided that the new statute be applied retroactively, the Court held that the longer, general statute of limitations for torts which were in effect at the time of the malpractice governed the claim.

¶17 However, in the more recent case of *Fisher v. First Citizens Bank,* 2000 MT 314, 302 Mont. 473, 14 P.3d 1228, the Court held that statutes of limitation are procedural and that unless the legislature expressly provides otherwise, they should be applied to actions brought after the time they are effective, irrespective of when the actions accrue:

> ¶ 14 Statutes of limitations are generally considered laws of procedure. If the legislature passes a new statute of limitations, all rights of action are to be enforced under the new procedure regardless of when the cause of action accrued unless there is an explicit savings clause set forth in the statute. [Citations omitted.]

In *Fisher,* the legislature had enacted a savings clause expressly providing that it did not affect rights and duties that had matured or proceedings that had begun.

¶18 *Fisher* effectively overrules *Penrod.* The reference in *Fisher* to a "new statute of limitations" does not distinguish the decision in *Fisher* from that in *Penrod*; the statute in *Penrod* was a "new" and distinct statute for malpractice claims. Moreover, the holding in *Fisher* is based on the Court's characterization of statutes of limitation as "procedural." A statute affecting procedure may be applied to causes of action arising prior to its enactment and such application does not constitute a retroactive application subject to section 1-2-109, MCA, which provides that statutes are not retroactive unless the legislature expressly provides for retroactive application. *Haugen v. Blaine Bank of Montana*, 279 Mont. 1, 8-9, 926 P.2d 1364, 1368 (1996).

¶19 There is no savings clause in the 1997 amendments, hence they apply to claims filed after the effective dates of the amendments. The 1999 amendments to section 39-72-403, MCA, did not change the limitations period adopted in 1995.

¶20 Therefore, if the legislature's reference to "injury" when making the new limitations inapplicable to injuries occurring prior to the effective date of the 1995 amendments does not encompass occupational diseases, the amendments apply to all occupational diseases irrespective of the date they arose or were diagnosed. If the reference does apply, then the section still applies since the occupational disease claim was not made until after July 1, 1997.

¶21 Liberty denied the claim against it on March 11, 2003. The petition in this case was filed on April 14, 2005, which is more than two years after the denial. However, in *Preston v. Transportation Ins. Co.*, 2004 MT 339, 324 Mont. 225, 102 P.3d 527, the Montana Supreme Court held that mediation proceedings toll the statute of limitations. While the statute of limitations involved in that case was the statute applicable to rescinding a contract based on mistake of fact, section 27-2-203, MCA, the Court found that tolling arises out of the mandatory nature of the mediation statutes:

> ¶ 36 As § 39-71-2408(1), MCA, states, mediation is mandatory under the Workers' Compensation Act before a party can even petition the Workers' Compensation Court for relief. In addition, the Workers' Compensation Court does not have jurisdiction during the pendency of a statutorily-mandated mediation, given that a claimant may only petition the Workers' Compensation Court "after satisfying dispute resolution requirements otherwise provided" in the Workers' Compensation Act--such as mandatory mediation.
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> ¶ 37 Given these clear statutory constructs, we hold that the statute of limitations tolled during the pendency of Preston's mediation.

*Preston,* ¶¶s 36-37. The rationale of the Court requires the same tolling conclusion with respect to section 39-71-2905(2), MCA (1997-2003), unless, as Liberty argues, the two-year limitation period is a statute of repose rather than a statute of limitations.

¶22 Statutes of repose provide time limits which are absolute and which cannot be tolled. That is because they extinguish the underlying right giving rise to the cause of action. *Hardgrove v. Transportation Ins. Co.,* 2004 MT 340, ¶ 10, 324 Mont. 238, 103 P.3d 999. For a court to characterize a limitations period as a statute of repose rather than a statute of limitation, the language of the statute must clearly indicate legislative intent to extinguish the right of action after the stated period. That was the case in *Hardgrove*

where subsections (1) and (2) of the 1983 version of section 39-72-403, MCA,[8] established basic limitations periods – true statutes of limitation – for filing occupational disease claims, but went on to provide in subsection (3):

> (3) Notwithstanding the provisions of subsections (1) and (2) of this section, no claim to recover benefits under this chapter may be maintained unless the claim is properly filed within 3 years after the last day upon which the claimant or the deceased employee actually worked for the employer against whom compensation is claimed.

Similarly, in *Joyce v. Garnaas,* 1999 MT 170, 295 Mont. 198, 983 P.2d 369, the Supreme Court held that a requirement that any legal malpractice action be commenced within ten years of the malpractice was a statute of repose. In that case, as in *Hardgrove*, the statute established a basic limitations period – three years after discovery of the malpractice in the case of legal malpractice – but the legislature then expressly overrode that basic limitation with an absolute limitations period, providing that "in **no** case may the action be commenced after 10 years from the date of the act, error, or omission." *Id.* at ¶ 12, emphasis added.

¶23 The provision at issue in this case contains no similar, extraordinary or overriding provision. The use of the word "must" in the section does not change the provision into one of repose. Indeed, the word "must" was used in the basic statute of limitations period

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[8]Subsections (1) and (2) of 39-72-403, MCA (1983), provided:

> (1) When a claimant seeks benefits under this chapter, his claims for benefits must be presented in writing to the employer, the employer's insurer, or the division within 1 year from the date the claimant knew or should have known that his total disability condition resulted from an occupational disease. When a beneficiary seeks benefits under this chapter, his claims for death benefits must be presented in writing to the employer, the employer's insurer, or the division within 1 year from the date the beneficiaries knew or should have known that the decedent's death was related to an occupational disease.
>
> (2) The division may, upon a reasonable showing by the claimant or a decedent's beneficiaries that the claimant or the beneficiaries could not have known that the claimant's condition or the employee's death was related to an occupational disease, waive the claim time requirement up to an additional 2 years.

of the section considered in *Hardgrove* – section 39-72-403(1) and (2), MCA (1983) – and was also used in the basic limitations provision of the statute considered in *Joyce*.

¶24 I therefore conclude that section 39-71-2905(2), MCA (1997-2003) is a statute of limitations which may be tolled in accordance with *Preston*. The tolling period is calculated from the date of the request for mediation through the deadline for both parties to respond to the mediator's recommendation. *Preston,* ¶¶s 35 and 37.

¶25 Liberty argues that the there is "no tolling" in this case "[b]ecause the mediation petition was dismissed and the mediation procedure was never completed. . . ." (Motion to Dismiss (Rule 12 (b)(6)) and Motion for Summary Judgment and Supporting Brief at 4.) Its argument is without merit. Absent mediation, the statute of limitations would have run on March 11, 2005, however, the claimant filed for mediation on February 16, 2005. Contrary to Liberty's statement that the mediation petition was dismissed, there is no evidence to support that assertion. The only evidence is that the original date scheduled for mediation was vacated at Liberty's request. Moreover, it would have been an error to dismiss the proceeding in light of the fact that the claimant had complied with the occupational disease panel requirement.[9] As it stands, the petition for mediation is pending and the claimant is entitled to complete the mediation process. The limitations period therefore began tolling on February 16, 2005, and continues to be tolled. Even if this Court dismissed the petition because mediation is incomplete, the number of days from February 16, 2005, until completion of mediation would be added to the two years, *Preston,* ¶¶s 35 and 37; thus there would still be time for the claimant to file another petition.

## III. Judicial Estoppel

¶26 Liberty asserts that the claimant is judicially estopped from claiming occupational disease benefits. The alleged estoppel arises from a civil action filed by the claimant in state district court. That action named the State of Montana, Burlington Northern Santa Fe Railway Co., Robinson Insulation Company, John Swing, and unnamed "Does" as defendants. In his district court complaint, the claimant alleges that his asbestos-related lung disease was caused by exposure to asbestos present in Libby, Montana, as a result of W. R. Grace mining activities; to asbestos present on BNSF property located nearby the claimant's residence in Libby; and to asbestos in insulation used in Libby and supplied by Robinson Insulation Company. He further alleges that the State of Montana was negligent

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[9]If the occupational disease panel requirement had not been satisfied, another issue arises, that being whether the panel provisions toll the running of the limitations period. As with the mediation provisions, the panel requirement is mandatory and delays the ability of the claimant to seek legal redress.

in failing to warn him of the danger of the asbestos exposure in Libby. Liberty argues that the district court complaint is incompatible with the claimant's present claim for workers' compensation benefits and that he is therefore estopped from seeking occupational disease benefits.

¶27 Contrary to Liberty's contention, the claimant's petition for occupational disease benefits is **not** inconsistent or incompatible with his district court complaint. Read together, the district court complaint and the petition in the present case simply allege that the claimant was exposed to multiple sources of asbestos in the Libby area, including asbestos at his workplace. Any or all of those sources could have caused or contributed to his asbestos-related lung disease. Under such circumstances, the Rules of Civil Procedure permit pleading in the alternative, as well as joinder of multiple defendants potentially liable to the claimant even though it may ultimately be determined that one or more of them is in fact not liable. Rule 20(a) of the Rules provides in relevant part:

> All persons may be joined in one action as defendants if there is asserted against them jointly, severally, or in the alternative, any right to relief in respect of or arising out of the same transaction, occurrence or series of transactions or occurrences and if any question of law or fact common to all defendants will arise in the action. A plaintiff or defendant need not be interested in obtaining or defending against all the relief demanded. Judgment may be given for one or more of the plaintiffs according to their respective rights to relief, and against one or more defendants according to their respective liabilities.

However, the claimant could not have named Liberty, Stimson, or Champion in his district court complaint because district courts do not have jurisdiction over occupational disease claims – that jurisdiction lies with this Court, §§ 39-71-2905, 39-72-305, MCA (2003). Thus, he was required to split his action between the district court and this court.

¶28 The splitting of claims as between the two courts does not give rise to judicial estoppel. The purpose of judicial estoppel is summarized in *Kauffman-Harmon v. Kauffman,* 2001 MT 238, 307 Mont. 45, 36 P.3d 408 at paragraph 15:

> ¶ 15 The fundamental purpose of judicial estoppel is to protect the integrity of the judicial system and thus to estop a party from playing "fast and loose" with the court system. Hence, the doctrine of judicial estoppel binds a party to his or her judicial declarations, and precludes a party from taking a position inconsistent with previously made declarations in a subsequent action or proceeding. Although judicial estoppel may be regarded as a form of estoppel, "it is not strictly one of estoppel, but partakes rather of positive rules of procedure based on manifest justice and, to a

> greater or lesser degree, on considerations of the orderliness, regularity, and expedition of litigation", and "those elements such as reliance and injury, or prejudice to the individual, which are generally essential to the operation of equitable estoppel, may not enter into judicial estoppel, at least not to the same extent". [Citations omitted.]

That purpose is not undermined where claimants exposed to Libby asbestos sue all of the entities possibly responsible for the exposure and ask the courts to determine which entities, if any, are liable for the harm caused by the exposure.

¶29 Moreover, as set forth in *Kauffman-Harmon,* four elements must be satisfied in order to judicially estop a party:

> ¶ 16 A party claiming that judicial estoppel bars another party from re-litigating an issue must show that: (1) the estopped party had knowledge of the facts at the time he or she took the original position; (2) the estopped party succeeded in maintaining the original position; (3) the position presently taken is inconsistent with the original position; and (4) the original position misled the adverse party so that allowing the estopped party to change its position would injuriously affect the adverse party.

*Id.* at ¶ 16 (citations omitted). Liberty argues that the four elements need not be satisfied in every case and that there is a second type of judicial estoppel under which the party asserting the estoppel is relieved of the fourth element, which requires detriment to the party asserting the estoppel. Liberty cites *Brown v. Small,* 251 Mont. 414, 825 P.2d 1209 (1992), which was a legal malpractice case.

¶30 The plaintiff in *Brown* had been represented by two attorneys. Those attorneys brought an action on his behalf against the insurer of property owned by Brown which had been destroyed by fire. The attorneys recovered a $315,000 settlement on his behalf. Thereafter, a dispute arose between Brown and his attorneys over attorney fees. Brown retained a third attorney and sued the first two. During a settlement conference in that case, the two original attorneys indicated they had information which could lead to an additional recovery against the insurer. Brown then entered into an agreement providing that if the attorneys recovered an additional $20,000 he would dismiss the lawsuit over the fees. The attorneys filed a second action against the insurer in which they alleged, on Brown's behalf, that the insurer had fraudulently concealed the fact that additional coverage was in effect. They recovered an additional $112,500. Brown then dismissed his lawsuit for fees but then had second thoughts and filed a second action against the attorneys, alleging that the attorneys had negligently failed to discover the additional coverage when pursuing the first action against the insurer. He asked that his first lawsuit against the attorneys be reinstated. The district court dismissed that action and the

Supreme Court affirmed, finding that Brown was judicially estopped from alleging negligence against the attorneys since he had recovered $112,500 in the second action based on his allegation that the insurer had fraudulently concealed the fact of the additional coverage.

¶31 The decision in *Brown* does not drop the fourth prong – detrimental reliance – of the four-part test for judicial estoppel; indeed, the decision does not discuss the fourth element at all, or even articulate the four elements. Analysis of the facts shows that there was in fact detrimental reliance on the part of the attorneys. They commenced and pursued the action asserting the claim of fraud on the part of the insurer in exchange for Brown's promise to drop his first action against them.

¶32 There is, however, language in *Brown* which indicates that strict adherence to the four-part test may not always be required. Quoting from *Rowland v. Klies,* 223 Mont. 360, 726 P.2d 310 (1986), which in turn was quoting from *LaChance v. McKown,* 649 S.W.2d 658, 660 (Tex. Ct. App. 1983), the Court in *Brown* said:

> Judicial estoppel may arise when a person has taken a position or asserted a fact under oath in a judicial proceeding contrary to the position he is taking in the present litigation ... The rule's purpose is to suppress fraud and prevent abuse of the judicial process *by deliberate shifting of positions to suit the exigencies of a particular action*, and it will not be applied when the previous act or statement is uncertain or based on undetermined facts, but only when it is clear and certain. [Citations omitted.] [Emphasis added.]

*Brown* at 418, 825 P.2d at 1212 (parenthetical and bracketed material in original). The Court went on to say:

> Judicial estoppel is equally applicable to a party like Brown who seeks to take a position contrary to his pleadings in an earlier judicial proceeding. *Fey v. A.A. Oil Corp.* (1955), 129 Mont. 300, 323, 285 P.2d 578, 590. The doctrine applies with additional force here because Brown's allegation in the second complaint against the insurer resulted in a net recovery by him of almost $75,000. After accepting the benefits of that allegation, Brown cannot now change his position and allege that negligence by Small and Doubek was the real reason they did not discover the mid-term endorsement sooner.

*Id.* at 418-19, 825 P.2d at 1212. Moreover, in discussing the four-prong test in *Kauffman-Harmon*, the Court specifically indicated that "those elements such as reliance and injury, or prejudice to the individual, which are generally essential to the operation of equitable estoppel, may not enter into judicial estoppel, at least not to the same extent." *Kauffman-Harmon Supra,* ¶ 15 (quoted in ¶ 28 above).

¶33 Eliminating the detrimental reliance requirement is important to Liberty because it cannot prove detrimental reliance. There is no evidence or indication that Liberty took any action on account of the representations made by the claimant in the district court case, or that it was adversely affected in any way by allegations in the district court complaint. Moreover, Liberty could actually benefit if the claimant succeeds in the prosecution of his district court action, either through subrogation or by a finding that one or more of the defendants in that action are solely responsible for the claimant's asbestos disease.

¶34 However, even if the fourth factor is eliminated, the other factors are not satisfied. Element two – requiring that the party to be estopped succeeded in maintaining his original position – is not satisfied. I have reviewed the docket sheet for the district court case and take judicial notice of it. The case is in its early stages and no determinations have been made on the merits of the action. A copy of the docket entry sheet is attached.

¶35 Similarly, element three – inconsistency in positions – is not met. An occupational disease is compensable even though non-occupational factors contributed to the disease. *See* § 39-72-706, MCA (1997-2003) (allowing for apportionment between occupational and non-occupational factors contributing to the disease). Further, since the claimant may have been exposed to multiple sources of asbestos while living and working in Libby, any or all of those sources may have contributed to his lung disease. He is entitled to sort out liability among those sources.

¶36 Finally, element one – knowledge – is not met. Given the multiple potential sources of asbestosis exposure and the difficulty in sorting out causation and contribution among those sources, it can hardly be said that in bringing the district court action the claimant had knowledge of facts inconsistent with his position in this case.

¶37 In sum, Liberty has failed to establish a basis for judicially estopping the claimant from pursuing his present petition.

## IV. Latency Period as Precluding Any Claim Against Liberty

¶38 Liberty urges that the claim against it should be summarily dismissed in light of the long latency period alleged in the petition and Dr. Whitehouse's general affidavit on file in *Johnson v. International Paper Co. and Liberty Northwest Ins. Corp.*, WCC No. 2004-1092. A copy of Dr. Whitehouse's affidavit was attached to Liberty's motions in this case.

### A. Dr. Whitehouse's Opinions

¶39 Dr. Whitehouse is a pulmonologist who treats many Libby, Montana, workers and residents who suffer from asbestos-related lung disease. *See Paul v. Transportation Ins. Co.*, 2004 MTWCC 69, ¶ 15; *Doubek v. CNA Ins. Co.*, 2004 MTWCC 76, ¶ 13. He has

testified previously in this Court, *id.* and *see also Fellenberg v. Transportation Ins. Co.*, 2004 MTWCC 29. His experience is summarized in *Doubek* as follows:

> ¶ 13 Dr. Whitehouse is a board certified pulmonologist who has been treating Libby asbestosis cases for approximately three decades. . . . Dr. Whitehouse has evaluated approximately 500 patients from Libby and maintains and tracks data concerning those patients. He has also treated asbestosis patients from the Hanford, Washington, nuclear facility.

*Doubek*, ¶ 13.

¶40 In the prior cases, as well as in his general affidavit, Dr. Whitehouse described the nature of Libby asbestos and lung disease arising from that asbestos. In *Fellenberg,* I summarized as follows:

> [Libby asbestos lung] disease is caused by "tremolite" asbestos fibers. Tremolite fibers are needle like and penetrate the lung more deeply than other, more common types of asbestos fibers which have a serpentine structure. They are too small to be expelled, therefore they lodge in the inner surfaces of the lung and slowly migrate outward until they reach the pleura, which is the thin membrane which covers the outside of the lungs. Dr. Whitehouse described the pleura as an "expansible" membrane much like a balloon. As tremolite fibers penetrate and impregnate the pleura, the pleura thickens and takes on an orange-rind appearance. The thickening causes the pleura to be less elastic and expansive, thus limiting inspiration (inhaling).

*Fellenberg*, ¶ 16.

¶41 The petitioner in *Johnson v. International Paper Co.,* WCC No. 2004-1092, filed a General Affidavit of Dr. Alan C. Whitehouse. In that affidavit, Dr. Whitehouse sets out a great deal of information about the nature of Libby asbestos disease, including the latency period of the disease. The latency issue is raised by Liberty in that case as well as in this case. Liberty argues the latency period described by Dr. Whitehouse is so long that the claimant's current lung disease could not be the result of his exposure while employed by Stimson.

¶42 In his affidavit, Dr. Whitehouse provides the following information and opinions concerning the nature of Libby asbestos:

> 11. Asbestos is a mineral fiber. There are two kinds, serpentine and amphibole. Serpentine asbestos, or chrysotile asbestos, is the kind used commercially in building products. Serpentine asbestos is more curly,

or more club-like, whereas amphibole asbestos is like tiny needles or spears. The Libby asbestos is an amphibole. It is generally referred to as tremolite, and variously referred to as winchite, richterite or tremolite-actinolite, all of which are amphiboles. I will refer to it as tremolite.

(Motion to Dismiss (Rule 12(b)(6)) and Motion for Summary Judgment and Supporting Brief, Ex. 2[10] at 3.) He goes on to describe tremolite fibers, the effect of inhaling the fibers, and the disease they produce:

> 12. A tremolite fiber is shown on Exhibit 4. The fibers are long and sharp, like needles. The fibers are microscopic, as are the tiny air sacs (alveoli) in the lungs. The fibers when breathed in lodge in the tiny air sacs, and are too small to be expelled. With each breath, they poke and scar the air sacs and the lung tissue structure around the air sacs (the interstitia). Scarring in the interstitia is interstitial disease. When the interstitia are significantly scarred, they can no long [sic] expand or contract, and breathing is restricted.
>
> 13. The asbestos fibers also migrate through the air sacs to the outside portion of the lung, where they scar and inflame the pleura (the lung lining) and cause pleural disease. *See* Frazer and Pare, p.2809.
>
> 14. The normal pleura is actually thinner than a blown up balloon. It is a very thin membrane, and it can expand like a balloon. Asbestos fiber scarring causes the pleura to look much like the orange portion of an orange rind, and can be just as thick. When surgeons peal it off the pleura, they call it a rind. When the lung lining becomes as thick as an orange rind, it can no longer expand freely and breathing is restricted. Asbestos disease is restrictive lung disease.

(*Id.* at 4.)

¶43 According to Dr. Whitehouse, asbestosis, a diagnosis in which he includes pleural thickening, is progressive:

> 38. In most patients with asbestosis (including asbestos pleural disease) from exposure to amphibole asbestos, the asbestosis is

---

[10]The General Affidavit of Dr. Alan C. Whitehouse which is attached to Liberty's Motion to Dismiss (Rule 12(b)(6)) and Motion for Summary Judgment and Supporting Brief as Ex. 2, will hereinafter be referred to as Whitehouse Affidavit.

progressive. In the words of one author, "it appears that once a dose of asbestos sufficient to initiate the disease has been retained, it is inexorably progressive." Sluis-Cremer (1989) "Progression of Irregular Opacities in Asbestos Miners," British Journal of Industrial Medicine, 46:846.

(*Id.* at 9.)

¶44 Finally, Dr. Whitehouse discusses the latency period between exposure and the time when the disease becomes identifiable and symptomatic:

> 42. There is a latency period between exposure [to asbestos] and the first appearance of asbestos disease on chest x-ray or CT. During the latency period, microscopic asbestos fibers are working at a microscopic level, until they become detectible on chest x-ray or CT. The average latency period is said to be 20 years. Rosenstock (1994), p. 256. ATS (2000) uses a period of 15 years. With tremolite asbestos, the range appears to be about 5-50 years, with an average latency period of about 30 years from first exposure to diagnosis.

(*Id.* at 10.) I attach a complete copy of Dr. Whitehouse's affidavit for further information about his opinions concerning Libby asbestosis disease.

¶45 In considering Liberty's motion with respect to latency, I treat Dr. Whitehouse's affidavit, and the evidence tendered therein, as uncontroverted. I do so because Liberty is relying on it; because the information is generally applicable to Libby claimants treated by Dr. Whitehouse, including the claimant in this case; because the claimant does not dispute the opinions set forth therein; and, because no contrary opinions or information are proffered in connection with Liberty's motion. I recognize that the claimant in this case has not specifically addressed the latency issue;[11] however, his counsel has addressed the argument in the *Johnson* case (representing claimant Johnson), and the matter was argued to the Court in *Johnson*. In any event, the issue is resolved in the claimant's favor.

---

[11]The claimant addressed the mediation issue and requested that the present proceedings be stayed, reserving argument concerning the other issues for a later time. However, in light of my holding that this Court has jurisdiction, the other issues are ripe for resolution. I resolve them without further argument from the claimant since my determinations herein favor him.

B. The "Last Injurious Exposure" Rule

¶46 Liability as between or among employers of a claimant exposed to asbestos in the workplace is governed by section 39-72-303(1), MCA (1993-2003). The subsection has not been substantively changed since 1993,[12] and presently provides:

> **39-72-303. Which employer liable.** (1) Where compensation is payable for an occupational disease, the only employer liable is the employer in whose employment the employee was last injuriously exposed to the hazard of the disease.[13]

While the claimant was employed at the same facility from 1960 until his retirement in May 1998, the facility changed ownership. Thus, he had two different employers over those years. That being the case, subsection (1) of 39-72-303 – the last injurious exposure rule – applies in determining which of those employers is potentially liable for his claim.

¶47 The "last injurious exposure" rule at issue here is not the same rule as applied in *Caekaert v. State Compensation Mut. Ins. Fund,* 268 Mont. 105, 111, 885 P.2d 495, 499 (1994); *Liberty Northwest Ins. Corp. v. Champion Int'l. Corp.,* 285 Mont. 76, 945 P.2d 433 (1997); and *Montana State Fund v. Murray,* 2005 MT 97. Those cases involved allegations of aggravations suffered on account of second, subsequent occupational diseases or aggravations arising after an earlier injury or a previously diagnosed occupational disease. In this case, a single disease has been diagnosed and it was diagnosed subsequent to the claimant's retirement.

---

[12]The 1993 legislature made minor changes in style. 1993 Montana Laws, ch. 619, § 5.

[13]In 1993 the legislature added subsection (2) to the statute. That subsection governs liability where there is one employer but multiple insurers, providing:

> (2) When there is more than one insurer and only one employer at the time the employee was injuriously exposed to the hazard of the disease, the liability rests with the insurer providing coverage at the earlier of:
>
> (a) the time the occupational disease was first diagnosed by a treating physician or medical panel; or
>
> (b) the time the employee knew or should have known that the condition was the result of an occupational disease.

§ 39-72-303(2), MCA (1993-2003) (enacted by 1993 Montana Laws. ch. 619, § 5.)

¶48 Larson's treatise on workers' compensation law reports that the last injurious exposure rule has particular application to diseases arising from the inhalation of fumes and chemicals, *including asbestosis*. 9 Arthur Larson and Lex K. Larson, Larson's Workers' Compensation Law, § 153.02[5]. He notes that the rule is "particularly useful for allocating liability in occupational disease cases, which often involve a number of insurers," and cites asbestosis cases as an example. *Id.* The rule imposes liability for cumulative exposures solely on the insurer at risk during the claimant's last injurious exposure to the fumes, chemicals, or substances giving rise to the disease. *Id.*

¶49 Larson identifies two problems in applying the last injurious exposure rule. The first is in "determining who was the 'last' insurer at risk during claimant's exposure to the disease. . . ." *Id.* The second is "determining the degree of exposure that should be held to be 'injurious.'" *Id.*

¶50 The first problem is not an issue in this case, at least at the present time, since Liberty is not presently asserting (for purposes of its motion) that the claimant was **not** exposed to asbestos during his employment by Stimson. It is the second problem which is raised by Liberty.

¶51 Larson addresses the degree of injurious exposure required to hold an employer liable, as follows:

> **[7] Degree of Injurious Exposure Required**
>
> **[a] Determining How Much Exposure Is "Injurious"**
>
> It goes without saying that, before the last-injurious-exposure rule can be applied, there must have been *some* exposure of a kind contributing to the condition. So, if a silicosis claimant had been transferred to outside work or to work in a place where dust conditions were not harmful, the carrier on the risk during the later period will not be held liable. However, once the requirement of some contributing exposure has been met the question remains: Was this enough of an exposure to be deemed "injurious"?
>
> Traditionally, courts applying the last injurious exposure rule have not gone on past the original finding of some exposure to weigh the relative amount or duration of exposure under various carriers and employers. As long as there was some exposure of a kind that could have caused the disease, the last insurer at risk is liable for all disability from that disease. Thus, insurers or employers who have been at risk for relatively brief periods have nevertheless been charged with full liability for a condition that could only have developed over a number of years. In one instance, the carrier

had the misfortune to assume coverage at midnight during the last 11:00 P.M. to 7:00 A.M. shift worked by an employee who subsequently filed a claim for disability caused by anthracosilicosis. The insurer was held liable for the entire amount of the claimant's benefits despite its only being on the risk for seven hours.

In contrast to this traditional rule, however, are decisions such as that in *Busse v. Quality Insulation,* in which the Minnesota Supreme Court took notice of medical testimony to the effect that there is a "lag time" of five to ten years between exposure to asbestos and the development of asbestosis. The court accepted this testimony in support of a conclusion that the claimant's exposure under the last insurer, who had been at risk for only two months, was not a "substantial contributing cause" of death. Other courts have also held that in order to impose liability on the insurer who was last at risk, the exposure during its period of risk must have been of such length or degree that it could have *actually* caused the disease.

9 Arthur Larson and Lex K. Larson, Larson's Workers' Compensation Law, § 153.02[7][a] at 153-19-20 (2004)(footnotes omitted). Under the traditional rule discussed in Larson's, it is likely that Liberty is the insurer at risk for the claimant's asbestosis-related lung disease.

¶52 Even under the decisions cited in the last quoted paragraph of Larson's, Liberty is still not entitled to summary judgment. The latency period described by Dr. Whitehouse in his affidavit does **not** exclude the claimant's exposure during his Stimson employment as contributing to the claimant's pleural thickening. Dr. Whitehouse indicates that the latency period may be as short as five years. (Whitehouse Affidavit at ¶ 42.) The claimant's initial claim was in 2001, some eight years after Stimson became his employer. Moreover, in this case the claimant's exposure during his employment with Stimson occurred over a four and a half year period, which is far longer than the two-month period considered insignificant in *Busse (see* last paragraph of the Larson's quote above). Dr. Whitehouse's affidavit also indicates that whether or not the four and a half year exposure during the claimant's employment by Stimson is in fact the cause or a contributor to the claimant's condition in 2001, it may have been sufficient to cause future disease independently of prior exposure. He cites a study indicating that two years of exposure to amphibole fibers is sufficient to cause asbestosis. (Whitehouse Affidavit at ¶ 35.) Finally, Dr. Whitehouse's affidavit does not rule out some contribution of exposure during later years to the claimant's current disease; he simply does not address the contribution.

¶53 Under any of the lines of cases cited and discussed in Larson's, Liberty has failed to provide uncontroverted evidence demonstrating that the claimant was not significantly and injuriously exposed to asbestos during his employment with Stimson. Thus, it is not

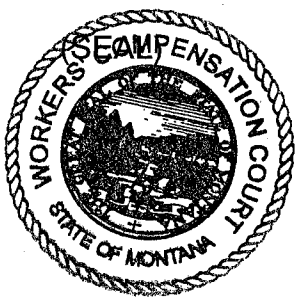
entitled to judgment as a matter of law. The nature and extent of any contribution of asbestos exposure during the Stimson years must be ascertained at trial. Liberty may renew its latency argument at that time. Based on the facts found following the trial, this Court will either determine which line of cases to follow in applying the last injurious exposure rule or craft a new, different rule for Montana.

ORDER

¶54 Liberty's motions to dismiss and for summary judgment are **denied**.

¶55 A new scheduling order will be separately issued.

DATED in Helena, Montana, this 5th day of July, 2005.




JUDGE

c: Ms. Laurie Wallace
Mr. Jon L. Heberling
Mr. Leo S. Ward
Mr. Larry W. Jones
Mr. Charles E. McNeil
Attachments: General Affidavit of Dr. Alan C. Whitehouse and Cascade County docket sheet, Case No. ADV 04-176.
Submitted: June 6, 2005

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Attorneys for Petitioner

**IN THE WORKERS' COMPENSATION COURT OF THE STATE OF MONTANA**
**IN AND FOR THE AREA OF KALISPELL**
**BEFORE THE WORKERS' COMPENSATION JUDGE**

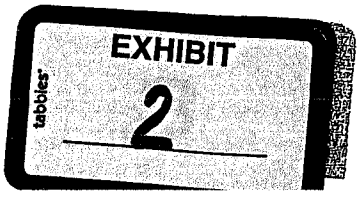
| | | |
|---|---|---|
| RAYMOND JOHNSON,<br><br>Petitioner,<br>v.<br><br>INTERNATIONAL PAPER CO.; as successor-in-interest to CHAMPION INTERNATIONAL CO; and LIBERTY NW INS. CORP.,<br><br>Respondents/Insurer | ) | WCC No. 2004-1092<br><br>**GENERAL AFFIDAVIT OF DR. ALAN C. WHITEHOUSE** |

STATE OF MONTANA)
:ss
County of Lincoln )

DR. ALAN C. WHITEHOUSE, being first duly sworn upon oath, deposes and states as follows:

1. Qualifications.

1. I am Dr. Alan C. Whitehouse. My address is 1507 East Eloika Road, Deer Park, WA 90066.



2. I am licensed in Montana. I currently practice chest medicine at the Center for Asbestos Related Disease in Libby, Montana where we have about 1,500 active cases of asbestos disease from exposure to Libby tremolite asbestos.

3. My curriculum vitae is attached as Exhibit 1.

4. In addition, I have been an invited speaker on the subject of Libby tremolite asbestos disease at various locations across the country.

| | | |
|---|---|---|
| 1998 | Libby, MT | Presentation to local doctors at St. John's Hospital |
| 1/2/00 | Bellingham, WA | America College of Occupational and Environmental Medicine |
| 3/00 | Cincinnati, OH | Center for Disease Control, meeting on tremolite asbestos disease |
| 5/10/00 | Washington D.C. | NIOSH/CDC meeting on tremolite asbestos disease |
| 10/00 | Kalispell, MT | Grand rounds at Kalispell Regional Hospital |
| 2001 | Washington D.C. | Senate Committee Panel |
| 6/24/02 | Missoula, MT | Conference on Asbestos Disease 2002 New Directions and Needs in Asbestos Research |

5. Since 1980 I have evaluated or treated over 500 patients for asbestos disease from Libby tremolite. Since about 1980 patient data has been tracked on a data base. Since 1980 I have also evaluated or treated over 500 patients for chrysotile asbestos disease. I am in a position to compare asbestos disease from Libby tremolite to asbestos disease from chrysotile asbestos. Chrysotile asbestos is the ordinary form of asbestos used in building materials in the United States, accounting for about 95% of the total asbestos used in the United States. Fraser and Pare's Diagnosis of Diseases of the Chest, 4th Ed. (1999), p.2420.

6. A listing of medical literature and texts which I consider authoritative on pulmonary medicine and asbestos disease has been delivered to counsel.

7. I am Board Certified in internal medicine and pulmonary disease. I treat the entire range of pulmonary disease. In my practice in Spokane in the years 1994-2004, the majority of my time, probably about 90%, was

related to general chest disease, including asthma, emphysema, lung cancer and hospital care. About 5-10% of my time was spent on asbestos related issues and other pneumoconioses. Probably about 10% of my time was related to industrial disease. Currently I spend a small amount of time on legal matters, but for the most part, my time is devoted to patient care.

8. In 30 years of practice I have probably testified at trial 8-12 times, about half for the plaintiff and half for the defendant. I testified in three asbestos trials relating to exposure from the W.R. Grace mine and mill near Libby, and one trial on the same subject in Missoula, Montana. These trials related to asbestos disease from Libby tremolite. In addition, my deposition has been taken on the subject of asbestos disease probably 25-30 times. I have testified in three Libby asbestos cases before the Montana Workers' Compensation Court.

9. I have published a paper on asbestos disease in Libby, titled "Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana," Am J Ind Med 46:219-225 (2004). A copy of the paper is attached as Exhibit 2. 123 patients were followed for an average of 35 months. Lung function was measured in terms of total lung capacity, forced vital capacity and diffusion capacity. The range of loss was between two and four percent per year for each of these functions. This means that in 10 years such a patient would lose 20 to 40% lung function.

10. Over the last three decades I have practiced occupational medicine. I have performed studies for companies, done screenings for companies and done disability exams for companies. In the 1980s I was involved in multiple screening programs for asbestos disease. I have also done independent medical examinations for the State of Washington, Department of Labor and Industry for decades.

2. The mechanism for asbestos disease.

11. Asbestos is a mineral fiber. There are two kinds, serpentine and amphibole. Serpentine asbestos, or chrysotile asbestos, is the kind used commercially in building products. Serpentine asbestos is more curly, or more club-like, whereas amphibole asbestos is like tiny needles or spears. The Libby asbestos is an amphibole. It is generally referred to as tremolite, and variously referred to as winchite, richterite or tremolite-actinolite, all of which are amphiboles. I will refer to it as tremolite.

12. A tremolite fiber is shown on Exhibit 4. The fibers are long and sharp, like needles. The fibers are microscopic, as are the tiny air sacs (alveoli) in the lungs. The fibers when breathed in lodge in the tiny air sacs, and are too small to be expelled. With each breath, they poke and scar the air sacs and the lung tissue structure around the air sacs (the interstitia). Scarring in the interstitia is interstitial disease. When the interstitia are significantly scarred, they can no long expand or contract, and breathing is restricted.

13. The asbestos fibers also migrate through the air sacs to the outside portion of the lung, where they scar and inflame the pleura (the lung lining) and cause pleural disease. See Frazer and Pare, p.2809.

14. The normal pleura is actually thinner than a blown up balloon. It is a very thin membrane, and it can expand like a balloon. Asbestos fiber scarring causes the pleura to look much like the orange portion of an orange rind, and can be just as thick. When surgeons peal it off the pleura, they call it a rind. When the lung lining becomes as thick as an orange rind, it can no longer expand freely and breathing is restricted. Asbestos disease is restrictive lung disease.

**3. Diagnosis of asbestos disease.**

15. For the diagnosis of asbestos disease, I use American Thoracic Society (2004), "Diagnosis and Initial Management of Non-Malignant Diseases Related to Asbestos," Am J Respir Crit Care Med, Vol. 170: 691-715 (2004). ATS (2004) defines asbestosis as "asbestos induced pulmonary parenchymal fibrosis, with or without pleural thickening." Rosenstock (1994), Clinical, Occupational and Environmental Medicine, "Asbestosis and Asbestos-Related Pleural Disease," p.261 states: "Some investigators have used the term asbestosis to encompass non-malignant asbestos-related pleural abnormalities." I agree with this statement. Asbestos interstitial disease is due to scarring in the lung structure around the alveoli (air sacs) from the poking and inflammation from asbestos fibers. Asbestos pleural disease is due to the scarring and inflamation in the pleura (the lung lining) from asbestos fibers. Asbestos pleural disease and asbestos interstitial disease are essentially the same disease process.

16. The diagnosis of asbestos disease generally requires at a minimum, a history of exposure to asbestos and a 15 year latency period. ATS (2004) states the diagnostic criteria as follows:

Evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology.

Evidence of causation by asbestos as documented by the occupational and environmental history, markers of exposure (usually pleural plaques), recovery of asbestos bodies, or other means.

Exclusion of alternative plausible causes for the findings.

17. I have taken hundreds of histories of work exposure at the W.R. Grace mine and mill, and am familiar with conditions in the various jobs there.

18. Asbestos disease causes a restrictive defect. The amount of air breathed in is restricted. The physical examination includes determinations of chest restriction, the presence of rales (the crackling sound of scarred air sacs reopening), and an evaluation of shortness of breath. While chest x-rays occasionally show abnormalities not seen on CT scan, chest x-rays generally miss about one-third of parenchymal abnormalities of asbestosis, and miss even higher percentage of pleural abnormalities, as compared to CT scans. See Frazer and Pare, pp. 2440 and 2431, respectively. Subpleural interstitial fibrosis is often not seen on chest x-ray, but is seen on CT scans, and may play a significant role in the severity of the disease process. See Schwarz and King, Interstitial Lung Disease, 4th Ed. 2003, p.422.

19. At our clinic, lung function tests are performed in accordance with ATS criteria. We use Knudson norms for vital capacity (spirometry), Intermountain Thoracic Society for lung volumes, and Miller for diffusion capacity.

20. The functions measured in lung function tests are shown on Exhibit 3. Normal or quiet inspiration/expiration is the tidal volume in Green. Maximum inspiration/expiration is in pink. The residual volume (orange) is the amount of air in the lung that cannot be expelled. The vital capacity (black) plus the residual volume (orange) is the total lung capacity. Of all lung function tests, the three most important in asbestos disease are forced vital capacity (FVC), total lung capacity (TLC) and diffusion capacity (DLCO).

Fishman's Pulmonary Diseases and Disorders, 3d Ed. (1998), p.883, states "The characteristic pulmonary function changes of asbestosis are a restrictive impairment with a reduction in lung volumes (especially FVC and

total lung capacity) decreased diffusion capacity, and arterial hypoxemia."

21. There are three components to pulmonary function tests. First is the spirometry, which measures the amount of volume of the lung and the rapidity of inhalation, which gives an index of air flow and lung volumes. We usually do this before and after brochodilator. If there is improvement with brochodilator, this suggests asthma. There is often an asthmatic effect with asbestos disease from exposure to Libby tremolite asbestos.

Second, we do lung volumes in what is called a body box, or plethysmograph, where we measure very small changes in air flow, pressure and volume, with a shutter and a closed system. Using Boyle's law, one can calculate the volume of the lung.

Third, we measure diffusion capacity, by having the patient breathe a small percentage of carbon monoxide, using very tiny tracer amounts of methane, which is not absorbed, and we measure what comes out of the lungs. We measure the methane, measure the carbon monoxide, and the differential uptake gives us the carbon monoxide diffusion capacity. Diffusion capacity is the efficiency of the lungs in transferring oxygen into the blood stream.

4. **Smoking.**

22. Smoking causes emphysema and chronic bronchitis.

23. Fishman, p. 684, states:

> The diagnosis of **emphysema** is based on pathologic, rather than clinical criteria. The ATS defines **emphysema** as air space enlargement distal to the terminal bronchioles and destruction of the alveolar wall.

24. Fishman, p. 683-684 states:

> The ATS defines **chronic bronchitis** as the persistence of cough and excessive mucus secretions on most days over a three month period for at least two successive years.

25. Fishman, p. 649, states:

> The ATS defines "chronic obstructive pulmonary disease (COPD) as a disease state characterized by the presence of airflow

obstruction due to chronic bronchitis or emphysema;"

26. ATS (1995), "Standards for the Diagnosis and Care of Patients with Chronic Obstructive Pulmonary Disease," Am J Respir Crit Care Med, Vol. 152, p.79, states: "Only about 15% of cigarette smokers develop clinically significant COPD."

27. Smoking disease is an obstructive disease. It obstructs what is breathed out. With emphysema, the lung tissue acts like an overexpanded balloon. It does not constrict back to its natural form. Hence exhalation is obstructed.

28. Asbestos disease is generally a restrictive disease. It restricts what is breathed in. The scarring in the lung lining and the lung air sacs and structure restricts the lungs' ability to expand on inhalation.

29. Generally the differences between obstructive disease due to smoking and restrictive disease due to asbestos can be sorted out on pulmonary function tests. This is somewhat complicated by the fact that asbestos disease often causes airway obstruction, or obstructive disease. See Fishman, p.884; Frazer and Pare, p.2445. Also, there is evidence that smoking increases the attack rate of asbestos disease. Frazer and Pare, p.2423.

Fishman, p.568, states:

> The hallmark of the obstructive pattern is a reduction in the FEV1/FVC percentage . . . Typically, all three lung volumes - residual volume, functional residual capacity, and total lung capacity are increased.

Normal for FEV1/FVC is 70 or higher. For hyperinflation in obstructive disease, TLC or RV must be over 120. Fishman, p.569.

5. Tremolite asbestos is highly toxic.

30. Amphibole asbestos in general and tremolite asbestos in particular are far more carcinogenic and fibrogenic (productive of asbestosis) than is chrysotile asbestos. Greenberg (1997), Occupational, Industrial and Environmental Toxicology, p.480 states:

> Several studies have also shown that worker cohorts exposed to higher concentrations of amphibole fibers have higher lung cancer rates than those exposed to similar concentrations of chrysotile asbestos. . . . This pattern of increased toxicity of amphiboles also holds true for all the other asbestos-related lung diseases (asbestosis, pleural disease, and mesothelioma).

31. Fraser and Pare (1999), supra p.1075, states "exposure to amphibole fibers . . . is associated with a significantly greater risk of carcinoma compared to chrysotile exposure."

32. Case (1991), "Health Effects of Tremolite," Annals of NY Academy of Sciences, 491, p.494, states:

> Significantly, the tremolite fibers were amongst the most carcinogenic tested, with actual incidence of 75% and "percent tumor probability" of 100%.

33. American Thoracic Society (1990), "Health Effects of Tremolite," Am Rev Resp Dis 142:1453, p.1456, states:

> Asbestiform varieties of tremolite are highly carcinogenic.

34. It has been estimated that tremolite asbestos is roughly ten times as carcinogenic as chrysotile asbestos. See McDonald (1997) "Chrysotile, Tremolite and Carcinogenicity" Annals of Occupational Hygiene, 41:699. See also, Antman (1993) "Natural History and Epidemiology of Malignant Mesothelioma," Chest 1993, p.373S, "Amphiboles are about 10x as carcinogenic as chrysotile."

35. It has been estimated that tremolite asbestos is roughly five to ten times as fibrogenic as chrysotile asbestos. See McDonald (1999) "Chrysotile, Tremolite and Fibrogenicity," Annals of Occupational Hygiene, 43:439. Compare Sluis-Cremer (1990) "Evidence for an Amphibole Asbestos Threshold Exposure for Asbestosis," Annals of Occupational Hygiene 34:443 with Ontario Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, (1984) Ontario Ministry of the Attorney General, and Doll and Peto (1985), "Asbestos: Effects on Health of Exposure to Asbestos," London: Her Majesty's Stationery Office.

The following summarizes the above studies' findings re the minimum number of fiber years of exposure for asbestosis:

| | |
|---|---|
| Sluis Cremer (1990) | min 2 fiber years (amphibole) |
| Doll & Peto (1985) | min 25 fiber years (chrysotile) |
| Ontario (1984) | min 25 fiber years (chrysotile) |
| Huang (1990) | min 22 fiber years (chrysotile) |

36. The results of the Libby asbestos screening include the following, for pleural abnormalities, two of three B readers concurring:

| | |
|---|---|
| All participants over 18 (n=6668) | 18% |
| Ever worked for W.R. Grace (n=365) | 51% |
| Lived with W.R. Grace workers (n=1376) | 26% |
| Vermiculite insulation in homes (n=2819) | 21% |

Peipins, et al (2003) "Environmental Health Perspectives," 111:14, pp.1753-59. These results clearly indicate that Libby tremolite asbestos is of high toxicity.

37. Amphibole asbestos is more than twice as likely to produce asbestosis and asbestos pleural disease which is progressive than is chrysotile asbestos. Compare Jones (1989) "Progression of Asbestos Effects," British Journal of Industrial Medicine, Gregor (1979) "Radiographic Progression of Asbestosis: Preliminary Report," Annals of the NY Academy of Sciences, and Becklake (1979) "Radiological Changes After Withdrawal From Asbestos Exposure," British Journal of Industrial Medicine, on chrysotile asbestos, with Sluis-Cremer (1989) "Progression of Irregular Opacities in Asbestos Miners," British Journal of Industrial Medicine, Cookson (1986) "The Natural History of Asbestosis in Former Crocidolite Workers of the Wittenom Gorge," American Review of Respiratory Disease, Ehrlich (1992) "Long Term Radiological Effects of Third Term Exposure to Amosite Asbestos Among Factory Workers," British Journal of Industrial Medicine, and McDonald (1999) "Chrysotile, Tremolite and Fibrogenicity" Annals Occupational Hygiene, on amphibole asbestos.

38. In most patients with asbestosis (including asbestos pleural disease) from exposure to amphibole asbestos, the asbestosis is progressive. In the words of one author, "it appears that once a dose of asbestos sufficient to initiate the disease has been retained, it is inexorably progressive." Sluis-Cremer (1989) "Progression of Irregular Opacities in Asbestos Miners," British Journal of Industrial Medicine, 46:846.

39. Cookson (1986) "The Natural History of Asbestosis in Former Crocidolite Workers of Wittenoom George," American Journal of Respiratory Disease 133:994-998, presents Fig. I, a chart showing that 34 years after first exposure approximately 97% of workers progressed to mild disease, 77% to moderate disease and 65% to severe disease. Crocidolite, like tremolite asbestos is an amphibole. Based on my experience, I believe the numbers for the Libby workers would be similar, perhaps with a longer lag time.

40. The paper I have published (see ¶ 9 above) demonstrates that Libby tremolite asbestos is highly toxic, and causes highly progressive lung disease.

41. There generally appears to be a distinct pattern for Libby tremolite asbestos disease. The disease appears to be predominately pleural, for the large portion of the time that people have the disease. Interstitial disease occurs rather late in the process, and frequently is only a minor factor. Frequently, we see subpleural interstitial fibrosis on CT scans. The pleural disease is highly progressive leading to restrictive defect and shortness of breath. Very often there is an obstructive component. Several patients have died of pleural disease, with no significant interstitial disease. Aspects of this pattern find support in Lockey (1984), "Pulmonary Changes after Exposure to Vermiculite Contaminated with Fibrous Tremolite," p.956; and in animal studies, Vorwald (1951), "Experimental Studies of Asbestosis," p.32 and Schepers (1955), "An Experimental Study of the Effects of Talc Dust on Animal Tissue," p.322. Other investigators as well have found significant restrictive disease due to pleural thickening. See Rom (1992), "Accelerated Loss of Lung Function and Alveolitis in a Longitudinal Study of Non-Smoking Individuals with Occupational Exposure to Asbestos," American Journal of Industrial Medicine, p.843.

7. Latency period.

42. There is a latency period between exposure and the first appearance of asbestos disease on chest x-ray or CT. During the latency period, microscopic asbestos fibers are working at a microscopic level, until they become detectible on chest x-ray or CT. The average latency period is said to be 20 years. Rosenstock (1994), p.256. ATS (2000) uses a period of 15 years. With tremolite asbestos, the range appears to be about 5-50 years, with an average latency period of about 30 years from first exposure to diagnosis.

8. Course of the disease.

43. When asbestos disease due to Libby tremolite exposure is first diagnosable, there usually are no symptoms, only positive findings on chest x-ray or CT. The disease may take decades to progress to a point of severity. Severe disease may include shortness of breath, chest pain, rales, clubbing of the fingernails, hypoxia cor pulmonale, pleural effusions, and oxygen dependency. See ATS (2004). At the end stage, the patient is bedridden, oxygen dependent, and generally the hypoxia will lead to organ malfunction and death.

9. Workers dead from asbestos disease.

44. In 2000, I performed an evaluation of death certificates and some medical records, and identified 100 workers from the W.R. Grace mine and mill who had died of asbestos disease. A copy of this study has been delivered to counsel as Exh. 225. Of the 100, 49 died of asbestos lung cancer, 11 died of mesothelioma and 40 died of asbestosis (including asbestos pleural disease). With chrysotile asbestos disease, about 50% of patients with asbestosis develop lung cancer. Frazer and Pare, p.1075. Due to the higher toxicity of tremolite asbestos, the 60% rate of death by asbestos lung cancer and mesothelioma is not surprising.

10. Impairment generally.

45. For Montana cases, I use the AMA Guides to the Evaluation of Permanent Impairment (5th Ed.). I am familiar with it as to lung and heart disease, and recognize it as authoritative. The Guides, p.88, states:

> The purpose of respiratory impairment assessment is (1) to determine if a permanent respiratory impairment exists, (2) quantify its severity, (3) assess its impact on the ability to perform activities of daily living, and, if possible, (4) identify the cause of the abnormality and (5) recommend measures to prevent further impairment and insure proper function. (Numbers added.)

Evaluation of pulmonary function tests is the best objective tool in assessing severity of disease. The symptoms suffered in severe disease may include shortness of breath, fatigue, chest pain and cor pulmonale (right sided heart failure).

46. Guides, p.89, presents Table 5-1 "Impairment Classification of Dyspnea (shortness of breath)."

| Severity | Definition and Question |
|---|---|
| Mild | Do you have to walk more slowly on the level than people of your age because of breathlessness? |
| Moderate | Do you have to stop for breath when walking at your own pace on the level? |
| Severe | Do you ever have to stop for breath after walking about 100 yards or for a few minutes on the level? |
| Very severe | Are you too breathless to leave the house, or breathless on dressing or undressing? |

47. It is also useful to inquire about shortness of breath upon climbing one flight of stairs. Shortness of breath is a key producer of limitations on physical activities. Often we do an oxygen saturation test by placing an oximeter on the patient's finger and have the patient walk a measured distance or climb a flight of stairs. Normal oxygen saturation at the altitude of 2,000 feet is 93 to 94%, P02 greater than 65, based upon Julius Comroe, Physics of Respiration, p.161. "Desaturation" means an oxygen saturation rate of under 90%. Medicare pays for oxygen at 88% oxygen saturation and below. Desaturation is consistent with severe asbestos disease.

48. Guides, p.89, 5.2 states:

> The significance of respiratory symptoms is better understood when integrated with findings from more objective means, such as physical exam, radiography, lung function and lab studies.

All the above assist in evaluating impairment. Clinical judgment is important in doing impairment ratings. The Guides, p.11, states that "clinical judgment, combining both the "art" and "science" of medicine, constitutes the essence of medical practice."

49. For impairment ratings, the Guides generally rely on criteria presented at page 107, Table 5-12. The Table attempts to apply to many different respiratory disorders, and does not provide a good fit for the

restrictive defect found in asbestos disease. As stated above, there are three key pulmonary function test measures for restrictive disease: forced vital capacity, total lung capacity and diffusion capacity. Table 5-12 omits total lung capacity. Total lung capacity can be the most important measure in restrictive disease. The American Thoracic Society, "Lung Function Testing: Selection of Reference Values and Interpretive Strategies," Am Rev Resp Dis 1991; 144:1202-1218, states:

> A restrictive ventilatory defect is characterized physiologically by a reduction in total lung capacity . . . if there is a contradiction between vital capacity and total lung capacity in defining restriction, the classification should be based on total lung capacity.

Interestingly, although the Guides, Table 5-12, omit total lung capacity from the impairment criteria, Table 5-13 includes lung volumes in Respiratory Impairment Evaluation Summary, for restrictive disorders.

50. Lung function test results vary with the individual. Total lung capacity (TLC) may be in the severe range, whereas forced vital capacity (FVC) and diffusion capacity (DLCO) may not, yet the patient may have severe impairment of function. In such cases, the Guides, p.107, call for the use of clinical judgment:

> It is recognized that pulmonary impairment can occur that does not significantly impact pulmonary function and exercise test results but that does impact the ability to perform activities of daily living, such as with bronchiectasis.
>
> In these limited cases, the physician may assign an impairment rating based on the extent and severity of pulmonary dysfunction and the inability to perform activities of daily living (see Table 1-2).

51. We further note that the Guides, Table 5-12, also permit the use of FEV1, as a sole measure of impairment in an asbestos disease evaluation. This is inappropriate, since FEV1 is not a measure of restrictive disease.

52. For forced vital capacity, the Guides do not require use of a brochodiolator. They appear to use the pre-brochodiolator result. I concur with doctors Paul Loehnen and Dana Headapohl of Missoula, Montana, on this point.

53. In addition, Guides, Table 5-12 requires that FVC be in the 40s or DLCO be in the 30s, before the individual is considered impaired greater than 50%. In my experience with patients with asbestos disease from Libby tremolite asbestos exposure, many are dead before they reach this point.

**12. Obesity.**

54. Fishman, p.1555, states:

> Obesity is considered mild when . . . BMI (body mass index) lies between 28 and 40 Kg/M2.

Fishman, p.1556, states:

> The effects of obesity on lung volumes have been extensively studied. Patients with simple obesity may have either a mild restrictive ventilatory pattern or normal lung volumes. Generally, with simple obesity there is no restrictive ventilatory defect unless the body mass index is greater than 60 Kg/M2. In this circumstance, VC may be reduced by 25%, but TLC and FRC can still be within the range of normal. When TLC is reduced in patients with a BMI less then 60 Kg/M2, other explanations of the restrictive process should be sought.

DATED this 21 day of February, 2005.

Dr. Alan C. Whitehouse

SUBSCRIBED AND SWORN to before me this 21st day of February, 2005.

(SEAL)

Notary Public for the State of Montana

Residing at: Libby, MT

My Commission Expires: 5/17/08

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| | |
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1

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# Asbestos-Related Pleural Disease Due to Tremolite Associated With Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana

Alan C. Whitehouse, MD, FCCP[1,2]*

**Background** *The community of Libby, Montana has recently been the focus of national attention secondary to widespread amphibole contamination associated with vermiculite mining and processing.*

**Methods** *Patients who had occupational and non-occupational exposure to amphibole asbestos in Libby, Montana were evaluated for progressive loss of pulmonary function.*

**Results** *Of the 123 patients evaluated, 94 demonstrated average age-corrected accelerated loss per year of vital capacity at 3.2%, total lung capacity (TLC) 2.3%, and DLCO 3.3%. All patients all had predominantly pleural changes with minimal to no interstitial disease.*

**Conclusions** *The study demonstrates a progressive loss of pulmonary function in patients exposed to tremolite asbestos.* Am. J. Ind. Med. 46:219–225, 2004. © *2004 Wiley-Liss, Inc.*

*KEY WORDS: tremolite; asbestos; pulmonary function; Libby; vermiculite; environmental; exposure; mining; dust*

## INTRODUCTION

In November 1999, it was reported that the community of Libby, Montana was experiencing an epidemic of pulmonary disease associated with occupational and environmental contamination of asbestiform amphibole materials within the community. Investigations revealed that the asbestos contamination was associated with a vermiculite mining and processing operation. Tremolite is an amphibole which has very little commercial value but is a contaminant of the vermiculite ore source in Libby [McDonald et al., 1986a]. This report will reference the high incidence of asbestos related pleural changes and their progression associated with tremolite exposure from the vermiculite mining and processing activity in Libby. The amphibole of the Libby mine has been characterized by mineralogists as a tremolite–actinolite–richterite–winchite transition fiber and will henceforth be referred to as tremolite [US Geological Survey, Bulletin 2193, 2002].

The vermiculite bed seven miles northeast of Libby was discovered in 1916 and mined initially for asbestos by the Zonolite Corporation and then subsequently for vermiculite. It was mined by W.R. Grace & Co. from 1963 to 1990 and was for a long period of time the world's largest producer of vermiculite.

Vermiculite is a hydrated, laminar, aluminum-non-magnesium micacious silicate, which when heated expands to between 10 and 20 times its original proportions and is excellent as an insulator, soil conditioner, and fertilizer additive [Moatamed et al., 1986].

In the process of mining and processing this material, W.R. Grace Company had multiple sites in proximity to Libby including an expanding and shipping facility. The ore body contained 21–26% tremolite and was initially pro-

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cessed on the mountain. The concentrated unexpanded ore, which contained over 2–6% tremolite [Amandus et al., 1987] was then loaded in railcars and shipped throughout the nation to over 200 regional processing or expanding sites. With the application of heat, the ore expands to an accordion like configuration. The expanded vermiculite had up to 1–3% tremolite [Amandus et al., 1987].

Both expanded and unexpanded forms of vermiculite from the mine were made freely available to the community. Many of the homes in the community were insulated with vermiculite. Vermiculite was placed on the ball fields, school track, and children played in piles of vermiculite, which were near the mining and processing facilities. The vermiculite was also used as insulation for plywood dryers in the local lumber mills and could be found in the rail yards where ore cars were loaded for shipping.

Studies of occupational exposure and disease among former vermiculite mine workers found significantly increased rates of asbestosis and lung cancer [Amandus et al., 1987]. A mortality study of the Libby area by the Agency for Toxic Substances and Disease Registry (ATSDR) found that deaths due to asbestosis were among the highest in the country at 40–60 times the expected national rate [DHHS/ATSDR, 2000].

Medical screening in the year 2000 of approximately 6,200 residents of the Libby area who lived there prior to 1990 found over 14% of all participants had radiographic changes consistent with asbestos related abnormalities. These findings represent a significant public hazard in view of the long term health impact known to be associated with amphibole exposure. Additional medical screening in 2001 added more patients, now estimated at over 1,000 plus the 491 patients in this clinical practice who are not part of the 1,000 and who have been followed for up to 14 years. These 491 patients demonstrate isolated pleural plaques to diffuse pleural or interstitial disease including 40 known deaths from asbestos- related diseases. They were examined and followed by a two physician practice specializing in pulmonary disease. The patients were either referred by internists and family practitioners or were self referred. These patients have not been previously reported. Initially, they were mostly employees of W.R. Grace as well as some family members of employees. More recently, non-occupational exposed residents of the community have been identified with asbestos-related health abnormalities. Because of extensive longitudinal medical data in this clinical practice setting, a study was undertaken to determine if there was accelerated loss of pulmonary function in this group of patients.

## MATERIALS AND METHODS

Pulmonary function studies including spirometry with bronchodilator, plethysmographic lung volumes, and single breath carbon monoxide diffusion (DLCO) were conducted. The studies prior to 1998 were performed on a Sensormedics model 6200 and subsequently on a Medgraphics model 1085. All studies were done before and after bronchodilator utilizing Albuterol. The same technician was used throughout the entire period. Lung volumes and DLCO were measured after bronchodilator.

Normal values of pulmonary function results used spirometry as described by Knudson et al. [1983], lung volumes established by the Intermountain Thoracic Society [Kanner et al., 1984], and DLCO (non-adjusted values) by Miller et al. [1983]. All studies were reviewed to be certain that height, which was measured to the nearest half inch, and age at test date were correct, and if differences in height were present they were adjusted to match across study dates. American Thoracic Society (ATS) pulmonary function testing guidelines were used throughout [American Thoracic Society, 1995]. In total, 30 patients were removed from the study for the following reasons: chronic obstructive pulmonary disease with elevated residual volumes (14), previous thoracic surgery (1), unacceptable pulmonary function tests because of patient unreliability and inability to meet ATS acceptability criteria (9), and/or the presence of a significant non-asbestos related condition such as sarcoidosis or congestive heart failure (9). Several patients had multiple disqualifying diagnoses. The first and last set of pulmonary function tests were compared for all patients tested (153).

Since the patient values were all age corrected against the normative predicted values, changes in the percentage of predicted over time reflected changes of pulmonary function beyond that accounted for by aging. Differences between the first and last pulmonary function were tabulated and changes per year were calculated. Changes were recorded in percentage change per year because of the wide variation in ages and the usual way of presenting this data in a clinical practice setting.

Repeated measures of analysis of covariance was used to statistically test changes in pulmonary function over time with time modeled linearly. To account for individual differences in the period between assessments, the time between the first and last assessments was entered into the statistical analysis as a covariant.

The initial postero-anterior chest X-ray was graded for extent of pleural changes by the principle investigator and also by a board certified radiologist (Dr. Teel). The extent of pleural changes were graded as follows. The percentage of the lateral chest wall involved with pleural changes was measured and the average of both sides of the chest calculated. All patients were weighed at each visit and body mass index calculated.

## RESULTS

Of the 491 subjects, 220 were employees of the vermiculite facilities, 121 were family members, and 150 were

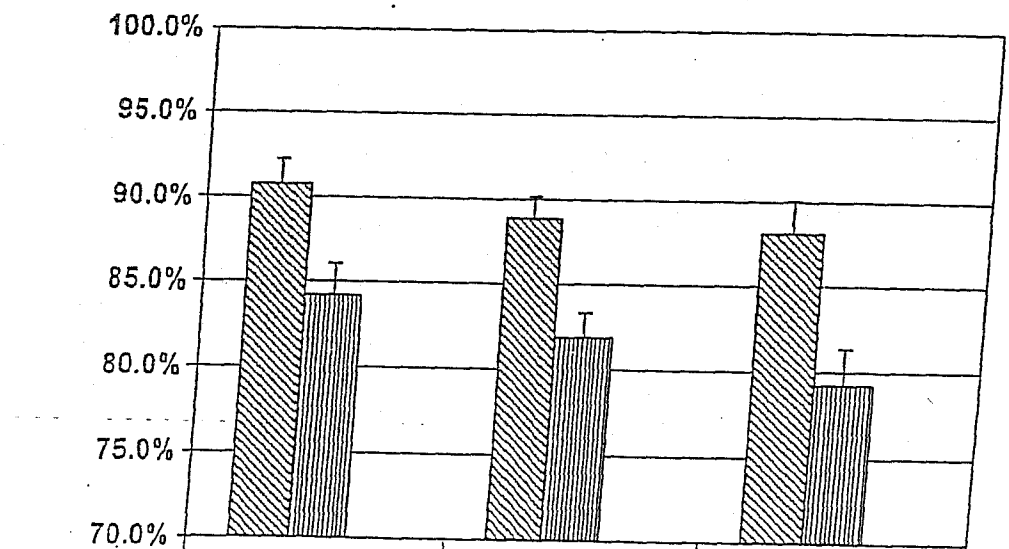
environmental exposures. Two or more sets of pulmonary functions were available on 153 patients. These subjects are representative of the Libby area population and the practice group of 491 patients. All had lived in Libby the majority of their life prior to 1990.

The majority of the 123 patients were ex-smokers with 8 of 123 (7%) being current smokers. Also, 27 (21%) never smoked. In total, 86 (70%) were former employees of W.R. Grace, 27 (22%) were family members of employees, and 10 of 123 (8%) were characterized as Libby environmental exposures only. In total, 99 were males (80%), 24 females (20%), and the average age was 66 years at first pulmonary function study.

Over the course of the study group observation, average BMI increased less than 1 $kg/m^2$ and there was no statistical correlation between increasing BMI and loss of lung function. Bronchial asthma was also evaluated as a confounding variable. Many subjects used a variety of bronchodilators prescribed by their personal physician although none carried a diagnosis of bronchial asthma and there was no evidence of significant changes in $FEV_1$ following bronchodilators.

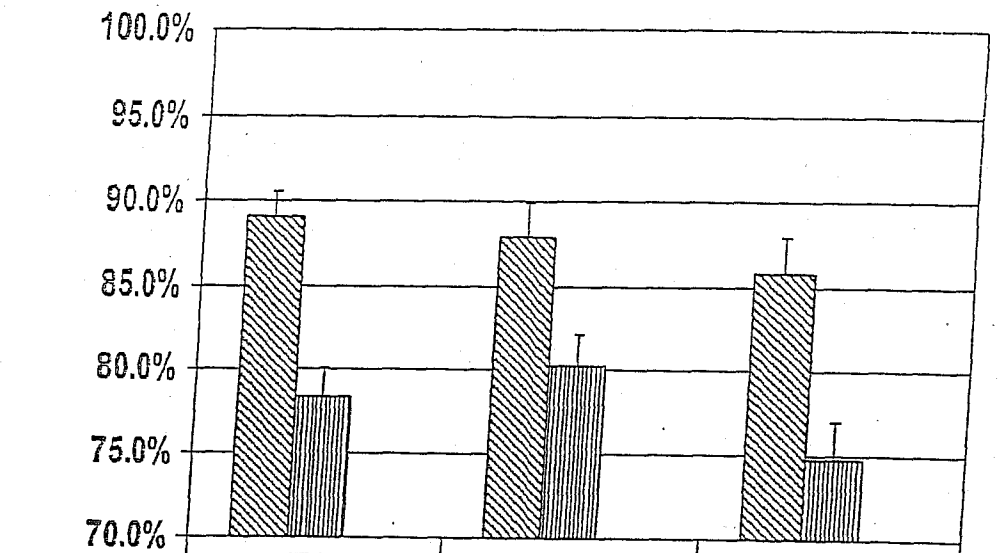
The majority had pleural changes only, consisting of either pleural plaques or diffuse pleural thickening. Because only about half the patients had high resolution computed tomography (HRCT) scans, it was not possible to differentiate this further with any certainty, due to the variations between the plain PA chest film and the HRCT. A total of 67 of 123 (55%) had no evidence on chest X-ray or HRCT of interstitial changes. The remaining patients (56) had minimal radiographic evidence of irregular interstitial changes involving the bases at profusion category 0/1 or 1/0. Of 123 films reviewed, 4 subject films were felt to be normal or equivocal. Of these, all subsequently developed overt pleural changes within a few years and three of four had pleural changes consistent with asbestos exposure on HRCT.

The parameters that were felt to be most valuable for analysis were forced vital capacity (FVC), (taking the best available and valid number from each set), total lung capacity (TLC), and the single breath diffusion capacity (DLCO). In the group of 123 patients (including those with improved FVC), the average yearly loss was 2.2 % for FVC, 2.3% for TLC, and 3.0% for DLCO as calculated over an average of 35 months (Fig. 1). Using FVC as the primary measure of worsening lung function, 94 of the 123 (76%) had an accelerated loss in this parameter. Analyzing the 94 of 123 who had progressive loss of FVC, the loss per year for FVC was 3.2%, TLC 2.3%, DLCO 3.3% (Fig. 2). In total, 79 of 123 patients with greater than 1% loss of FVC per year the average yearly loss was 3.6% for FVC per year, 2.5% for TLC, and 3.5% for DLCO (Fig. 3). The loss rate in this group could not be explained by increases in weight, extent of disease initially or subsequently or other concomitant illness. For the 67 patients with pleural changes alone and with no interstitial changes, the average yearly loss was 2.2% for



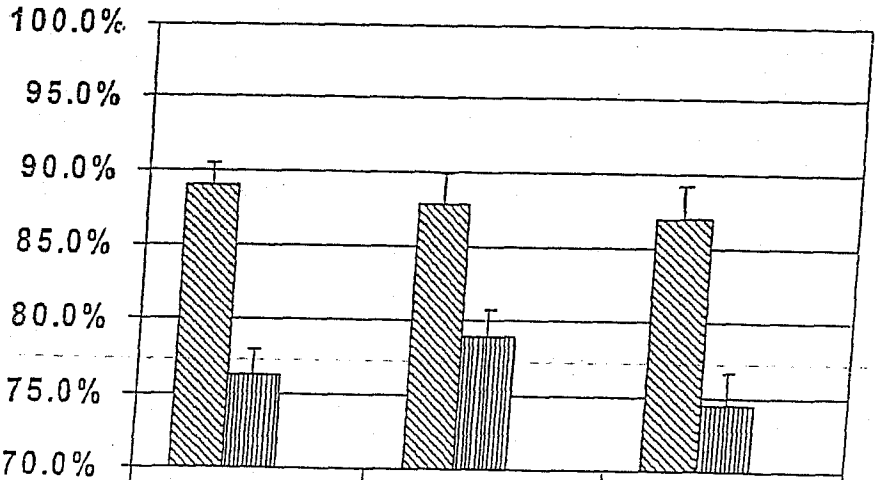

| | FVC | TLC | DLCO |
|---|---|---|---|
| ▨ BEFORE | 90.7% | 88.8% | 88.0% |
| ▥ AFTER | 84.3% | 82.0% | 79.3% |
| ☐ LOSS/YR | 2.2% | 2.3% | 3.0% |

FIGURE 1. Loss of pulmonary function; all 123 patients, average 35 months ($P < 0.001$).



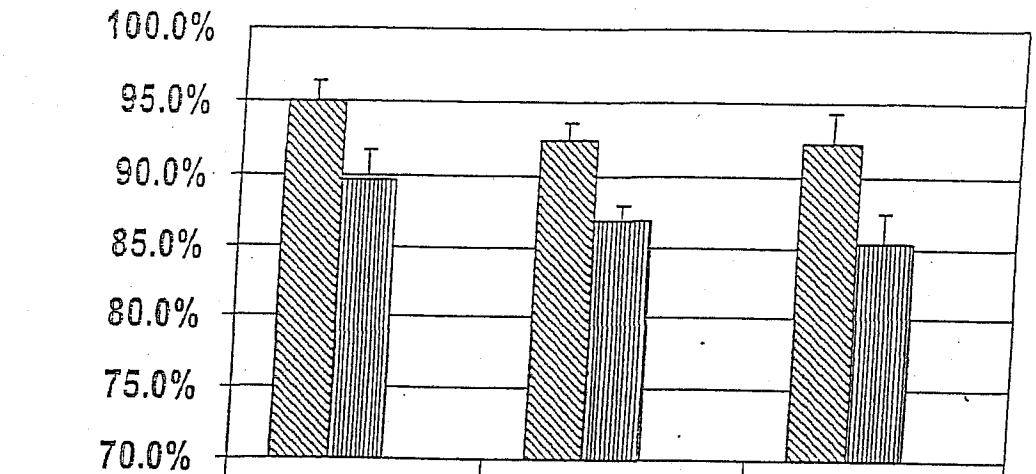

| | FVC | TLC | DLCO |
|---|---|---|---|
| BEFORE | 89.0% | 87.9% | 85.8% |
| AFTER | 78.4% | 80.3% | 74.8% |
| LOSS/YR | 3.2% | 2.3% | 3.3% |

**FIGURE 2.** Loss of pulmonary function 94/123 patients with worse FVC.




| | FVC | TLC | DLCO |
|---|---|---|---|
| BEFORE | 89.0% | 87.8% | 86.9% |
| AFTER | 76.3% | 78.9% | 74.5% |
| %LOSS/YR | 3.6% | 2.5% | 3.5% |

**FIGURE 3.** Loss of pulmonary function 79/123 patients with greater than 1% loss rate per year of FVC.




| | FVC | TLC | DLCO |
|---|---|---|---|
| BEFORE | 95.0% | 92.4% | 92.2% |
| AFTER | 89.6% | 86.8% | 85.2% |
| LOSS/YR | 2.2% | 2.3% | 2.9% |

**FIGURE 4.** Loss of pulmonary function 67/123 patients, pleural disease only.

FVC, 2.3% for TLC, and 2.9% for DLCO (Fig. 4). These results are very similar to those of the entire 123 patients (compare Figs. 1–4).

All values as noted above for decline of pulmonary function were statistically significant at $P \leq .01$. There did not appear to be any difference between the patients with pleural changes who had minor interstitial changes versus no interstitial changes. It is also noted that in the entire group the decline in the diffusion capacity was more rapid than the decline in either the FVC or TLC.

Extent of pleural changes as measured as described on the chest X-ray was evaluated in relation to the loss of lung function. There was no statistical correlation between the extent of pleural changes measured on the chest X-ray and the loss of pulmonary function. The only clearly discernible event leading to accelerated loss of pulmonary function in this entire group was benign asbsestos related effusions (three patients). These were treated vigorously with tube drainage and pleurodysis and the rate of loss equated to the 76% who lost function (2.2–3%).

## DISCUSSION

The progressive loss of pulmonary function in 76% of the 123 patients with pleural changes followed in this group of patients with Libby tremolite exposure is excessive compared to other published reports. Progression of asbestos disease in patients with exposure to chrysotile asbestos is well documented. Jones et al. [1989] demonstrated declines in FVC and FEV1 in men who had progressive pleural thickening. Of this group, 31% demonstrated progression of parenchymal small opacities in patients with pleural thickening and smoking was not a significant determinant of pleural progression. The amphibole crocidolite was present in one of the two plants studied and there was a higher rate of progression with crocidolite present. Miller and Miller [1983] demonstrated that patients with longstanding clinically inconsequential plaques remain at risk for diffuse pleural thickening and associated impairment of pulmonary function, which was the case in three patients with pleural effusions. Furthermore, in this group, there was no evidence of progression of small opacities. Decreases in vital capacity have been described by Lilis et al. [1991] and Schwartz et al. [1994]. Ohlson et al. [1985] described 4 year declines in FVC and $FEV_1$ in a group of asbestos cement workers. The average 4-year decrement of FVC in exposed subjects was 1.9% greater than the reference (control) subjects. Rom [1992] studied 77 asbestos insulators and found that losses of FVC averaged 92 cc per year, $FEV_1$ 66 cc per year, and TLC 14 cc per year. Kouris et al. [1991] found decreased pulmonary function associated with pleural plaques and more significantly with diffuse pleural thickening. Schwartz et al. [1990] demonstrated loss of $FEV_1$ and FVC associated with both plaques and diffuse pleural thickening and they concluded that "pleural fibrosis" among asbestos exposed patients is an independent predictor of spirometric patterns

consistent with restrictive lung function. Brodkin et al. [1996] further correlates loss of pulmonary function associated with increasing respiratory symptoms. Lockey et al. [1984] described changes in weight as a confounding variable measuring pulmonary function in the workplace. There was no evidence of significant weight changes in this group [McKay et al., 1999].

There are fewer articles on exposure to amphiboles. Shepherd et al. [1997] showed progression of pleural and parenchymal abnormalities associated with amosite. Sluiz-Cremer and Hnizdo, 1989] studied crocidolite workers in South Africa, and was able to demonstrate that once a dose of amphibole asbestos sufficient to initiate disease had been retained it was a naturally progressive process. Cookson et al. [1986] studying crocidolite workers demonstrated that asbestosis was actively progressing even after more than three decades. Erlich et al. [1992] demonstrated in amosite exposed workers that there was progression of pleural abnormalities 20 years after exposure. They found exposure of as little as 1 month was sufficient to produce radiologic signs of parenchymal and pleural fibrosis and progression was detectable greater than 20 years after the end of exposure. McDonald et al. [1986b], studying workers exposed to Libby tremolite from the Grace mine in Libby, Montana, has previously demonstrated extensive pleural plaques and pleural thickening on chest radiographs. Previously, Lockey et al. [1984], was first to describe an association between benign pleural effusions as well as pleural plaques on exposure to Libby tremolite that had been processed at an expansion plant in Ohio to be used as a conditioner for fertilizer.

## CONCLUSIONS

This study demonstrates that pleural changes related to exposure to Libby tremolite are associated with progressive loss of pulmonary function in a group of patients exposed to tremolite from approximately 1950 to 1975. Progressive loss of lung function is continuing 40 years after last exposure in 76% of this group who are representative of the population of Libby, Montana. The studies quoted above document both interstitial disease and pleural disease, both radiographically and functionally, but none document the rapid progression of loss of pulmonary function in such a large group of patients with predominantly pleural disease. McDonald et al. [1999] speculated on tremolite's increased fibrogenicity, and it would appear that tremolite–actinolite–richterite–winchite amphibole found in Libby vermiculite has a propensity for causing pleural changes that result in a progressive restrictive pattern on pulmonary function testing. Pleural changes alone are unlikely to cause a decrease in DLCO. DLCO decreases are likely to be associated with interstitial disease not apparent clinically on either plain chest radiograph or HRCT.

Exposure histories for this group are complex, because for the most part there was continuous exposure throughout this entire period that they lived in Libby, whether they were mine workers, family members of workers, or community members living near the vermiculite processing facilities.

This study demonstrates that the number of patients progressing is much higher than has previously been reported in studies with either chrysotile or amphibole asbestos exposure. Lincoln County, Montana, (where Libby is the county seat) has the highest mortality rate from asbestosis in the nation [DHHS/ATSDR CERCLIS No MT00090883840, 2000].

It is apparent from these data that the majority of the 1,500 persons who have radiologic changes of asbestos exposure are at increased risk for progressive loss of lung function from pleural changes alone or from potential future development of interstitial fibrosis. Assuming a latency period of between 20 and 30 years to significant disease, it is not unreasonable to expect that the people of Libby, Montana will have to be monitored over the next 30–40 years, because of the risk for loss of pulmonary function and other known diseases historically associated with asbestos exposure.

## ACKNOWLEDGMENTS

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Inspiratory Reserve Volume
Tidal Volume
Expiratory Reserve Volume
Residual Volume
Inspiratory Capacity
Functional Residual Capacity
Vital Capacity
Total Lung Capacity
Normal (resting) Inspiration
Normal (resting) Expiration
Maximum Inspiration
Residual Volume

EXHIBIT

3

Case #: ADV-04-176 ACTIONS/JUDGEMENTS SCAN & MAINTENANCE Last Doc 17
Judge 0808

Pltf: EDWARDS, ROBERT M. + Defd: STATE OF MONTANA +

| Doc# | Date (mm/dd/yyyy) | # | Description | Rcpt# | Fees | RL# | MF# |
|---|---|---|---|---|---|---|---|
| 14 | 5/11/2005 | 2 | * RANDY J. COX/ BOX 9199/ MISSOULA, MT 59807 | | | | |
| 14 | 5/11/2005 | 3 | * 406-543-6646 | | | | |
| 15 | 5/11/2005 | 1 | BNSF'S ANSWER TO AMENDED COMPLAINT & DEMAND | | | | |
| 15 | 5/11/2005 | 2 | FOR JURY TRIAL | | | | |
| 16 | 5/19/2005 | 1 | DEFT BNSF'S BRIEF IN SUPPORT OF MOTION FOR | | | | |
| 16 | 5/19/2005 | 2 | CHANGE OF VENUE | | | | |
| 17 | 6/07/2005 | 1 | BRIEF IN OPPOSTION TO MOTION FOR CHANGE | | | | |
| 17 | 6/07/2005 | 2 | OF VENUE (PLTFS) | | | | |

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Case #: ADV-04-176 ACTIONS/JUDGEMENTS SCAN & MAINTENANCE Last Doc 17
Judge 0808

Pltf: EDWARDS, ROBERT M. + Defd: STATE OF MONTANA +

| | Doc# | mm/dd/yyyy Date | # | Description | Rcpt# | Fees | RL# | MF# |
|---|---|---|---|---|---|---|---|---|
| | 6 | 6/08/2004 | 3 | * RANDY COX/MATTHEW HAYHURST/BOONE KARLBERG | | | | |
| | 6 | 6/08/2004 | 4 | * 201 W MAIN, STE 300, POB 9199, MISSOULA, MT | | | | |
| | 6 | 6/08/2004 | 5 | * 59807-9199, (406) 543-6646 * | | | | |
| | 7 | 6/08/2004 | 1 | JT STIP & MOT TO STAY ACTION AS TO DEFT ST | | | | |
| | 7 | 6/08/2004 | 2 | OF MONTANA | | | | |
| | 8 | 6/11/2004 | 1 | ORD TO STAY ACTION AS TO DEFT ST OF MT | | | | |
| | 9 | 6/28/2004 | 1 | SUMMONS **RETD**(2)(-) | | | | |
| | 10 | 6/28/2004 | 1 | NTC OF DISMISSAL WITHOUT PREJUDICE | | | | |
| | 11 | 8/19/2004 | 1 | STIPULATION AND ORDER (AMENDED COMPLAINT | | | | |
| | 11 | 8/19/2004 | 2 | BE FILED) | | | | |
| | 12 | 8/19/2004 | 1 | AMENDED COMPLAINT AND DEMAND FOR JURY TRIAL | | | | |
| | 12 | 8/19/2004 | 2 | AMENDED SUMMONS **ISSD** | | | | |
| | | 8/31/2004 | 1 | AMENDED SUMMONS **ISSD**(4) | | | | |
| | | 4/20/2005 | 1 | SUMMONS **ISSD** | | | | |
| | 13 | 4/22/2005 | 1 | SUMMONS **ISSD** (B.N. 04/21/4005) | | | | |
| * | 14 | 5/11/2005 | 1 | BNSF'S MOTION FOR CHANGE OF VENUE | | | | + |

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Case #: ADV-04-176 ACTIONS/JUDGEMENTS SCAN & MAINTENANCE Last Doc 17
Judge 0808

Pltf: EDWARDS, ROBERT M. + Defd: STATE OF MONTANA +

| Doc# | mm/dd/yyyy Date | # | Description | Rcpt# | Fees | RL# | MF# |
|---|---|---|---|---|---|---|---|
| | | | * * * * * NEILL ACCEPTS JURISD * * * * * * | | | | |
| | | 1 | | | | | |
| 1 | 2/23/2004 | 1 | COMPLAINT AND DEMAND FOR JURY TRIAL | | | | |
| 1 | 2/23/2004 | 2 | * JON L. HEBERLING | | | | |
| 1 | 2/23/2004 | 3 | * 745 SOUTH MAIN | | | | |
| 1 | 2/23/2004 | 4 | * KALISPELL MT 59901, 752-5566 * | | | | |
| 1 | 2/23/2004 | 5 | * TOM L. LEWIS | | | | |
| 1 | 2/23/2004 | 6 | * P.O. BOX 2325, 761-5595 * | | | | |
| 1 | 2/23/2004 | 7 | SUMMONS **ISSD** (2) | | | | |
| | 4/05/2004 | 1 | SUMMONS **ISSD** (2) | | | | |
| * 2 | 4/27/2004 | 1 | MOTION FOR SUBSTITUTION OF DISTRICT JUDGE | | | | |
| 3 | 4/30/2004 | 1 | SUMMONS RETD (ST MT 4-20-04) | | | | |
| 4 | 5/03/2004 | 1 | REQUEST (NEILL ACCEPTS JURISD FOR MCKITTRICK) | | | | |
| 5 | 5/05/2004 | 1 | SUMMONS **RETD**(JOHN SWING 04-16-04) | | | | |
| 6 | 6/08/2004 | 1 | JOHN SWING'S ANSWER TO COMPLAINT & DEMAND FOR | | | | |
| 6 | 6/08/2004 | 2 | JURY TRIAL | | | | + |

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