COMMONWEALTH ENVIRONMENTAL SERVICES, LLC
"A Native American Woman Owned Company"

Fax Cover Sheet

To:	Hilda Solis & Mr. Bibeault	From:	Gary S. Vander Boegh
Fax:	(904) 357-4704 (202) 693-6111	Date:	11-23-10
Phone:		Pages:	___Pages including the Cover Sheet
Re:	[redacted]	CC:	Attention Jim Bibeault and David Miller

☐ Urgent X For Review ☐ Please Comment X Please Reply ☐ Please Recycle

Comments:

COMMONWEALTH ENVIRONMENTAL SERVICES, LLC
"A Native American - Woman Owned Company"

Gary Vander Boegh, Vice President
Commonwealth Environmental Services, LLC
4645 Village Square Drive, St. F
Paducah, Kentucky 42001
Telephone: (270) 450-0850
Facsimile: (270) 450-0858

November 8, 2010

U. S. Department of Labor,
Frances Perkins Building, 200 Constitution Ave., NW
Room S-2018
Washington, DC 20210

Attention: Madam Secretary Hilda Solis & Jim Bibeault

Employee: [redacted]
File Number: XXXXX

Dear Ms Solis,

As "Authorized Representative" (AR) for claimant [redacted], spouse of [redacted] (deceased), I hereby submit the attached EE-2 form for Chronic Beryllium Disease (CBD) based on statutory requirements 42 USC § 7384l (13) (B) as follows:

(B) For diagnoses before January 1, 1993, the presence of—
(i) occupational or environmental history, or epidemiologic evidence of beryllium exposure; and
(iii) any three of the following criteria:
(I) Characteristic chest radiographic (or computed tomography (CT)) abnormalities.
(II) Restrictive or obstructive lung physiology testing or diffusing lung capacity defect.
(III) Lung pathology consistent with chronic beryllium disease.
(IV) Clinical course consistent with a chronic respiratory disorder.
(V) Immunologic tests showing beryllium sensitivity (skin patch test or beryllium blood test preferred).

The Department of Labor has further stated, "***For beryllium disease prior to January 1, 1993, a specific diagnosis of CBD IS NOT REQUIRED (emphasis added.)"....***

The Paducah Gaseous Diffusion Plant was a DOE facility from 1952 to July 28, 1998 and July 29, 1998 to present (remediation) where radioactive and beryllium material were present, according to the Department of Energy Office of Worker Advocacy Facility List (http://www.hss.energy.gov/HealthSafety/FWSP/Advocacy/faclist/findfacility.cfm). Per Chapter 2-700.4 (September 2004) of the Federal (EEOICPA) Procedure Manual, "***To determine whether to use the Pre or Post 1993 CBD criteria,*** the medical evidence must demonstrate that the employee was **either treated for**, **tested** or **diagnosed with lung cancer**. **If the earliest dated document is prior to January 1, 1993, the pre-1993 CBD criteria may be used**. Once it is established that the employee had a chronic respiratory disorder prior to 1993, **the CE is not limited to use of medical reports prior to 1993 to meet the three of five criteria."**

(Excerpt)

DOCKET NUMBER: 57973-2005
Decision Date: January 7, 2005

NOTICE OF FINAL DECISION

This is the decision of the Final Adjudication Branch concerning your claim for compensation under Part B of the Energy Employees Occupational Illness Compensation Program Act of 2000, as amended, 42 U.S.C. § 7384 *et seq.* (EEOICPA or the Act). This decision affirms the recommended acceptance issued on November 30, 2004.

STATEMENT OF THE CASE

On May 28, 2004, you filed a claim for survivor benefits, as the widow of **[Employee]**, Form EE-2, under Part B of the EEOICPA. **YOU IDENTIFIED 'BREATHING PROBLEMS" AND CHRONIC BERYLLIUM DISEASE (CBD) AS THE CLAIMED CONDITIONS. (emphasis added)...**

...."Based upon the DOE response that F.H. McGraw held a number of contracts from 1951 to 1954 and the security Q clearance notification, the district concluded that the DOE had a business or contractual arrangement with F.H. McGraw. **THE DISTRICT OFFICE FURTHER CONCLUDED THAT YOUR HUSBAND WORKED WITH F.H. MCGRAW AT THE PADUCAH GASEOUS DIFFUSION PLANT FOR AT LEAST ONE DAY ON DECEMBER 17, 1954 (emphasis added)** based upon the reduction in force notice.[2]...."

....."You submitted a medical report dated February 23, 1991, from Lowell F. Roberts, M.D., which indicates a history of chronic obstructive pulmonary disease (COPD), shortness of breath, and dyspnea. A February 23, 1991 X-ray report, from D.R. Hatfield, M.D., indicates a diagnosis of COPD. A February 25, 1991 CT-scan, from Barry F. Riggs, M.D., indicates abnormal nodular densities of the right lower lobe and a diagnosis of COPD. A February 26, 1991 medical report from M.Y. Jarfar, M.D. indicated that pulmonary function tests showed mild obstructive defects and mild diffusing lung capacity defects. You also submitted an X-ray report dated September 6, 1994, from Robert A. Garneau, M.D., that indicated

diagnoses of COPD and Interstitial Fibrosis. A November 27, 1994 medical report from David Saxon, M.D., indicated findings of rales and wheezing. A December 2, 1994 medical report from Dr. Saxon, indicates hypoxemia to the left lower lung. A December 2, 1994 medical report from Lowell F. Roberts, M.D., indicated diagnoses of shortness of breath, congestive heart failure, dyspnea and cough, and rales in the lung base. An August 13, 1995 X-ray report from Charles Bea, M.D., indicates a diagnoses of bibasilar infiltrates. A December 30, 1996 X-ray report from Sharron Butler, M.D., indicates an increase of lung markings since the September 14, 1992 study. In the March 1, 1998 X-ray report from Dr. Butler diagnoses of "advanced chronic lung changes, mild interstitial prominence diffusely, and patch density of the posterior right lung" are indicated. An August 19, 1998 CT-scan from James D. Van Hoose, indicates diagnoses of pleural thickening and pulmonary calcifications. **AN AUGUST 6, 1999 PULMONARY FUNCTION TEST FROM WILLIAM CULBERSON, M.D. INDICATES A DIAGNOSIS OF MODERATELY SEVERE RESTRICTIVE DISEASE(emphasis added).** An October 12, 1999 discharge summary from Eric B. Scowden, M.D. indicates diagnoses of progressive shortness of breath, congestive heart disease, COPD, and history of right-sided empyema complicating pneumonia necessitating prolonged chest tube drainage with a continued open sinus tract." Based upon these reports the district office concluded that you had CBD prior to January 1, 1993.[3]

On November 30, 2004, the district office issued a recommended decision concluding that your husband was a covered beryllium employee, that he was exposed to beryllium, and that he had symptoms and a clinical history similar to CBD prior to January 1, 1993. They further concluded that you are entitled to 30.316(a) of the EEOICPA implementing regulations provides that, "if the claimant does not file a written statement that objects to the recommended decision and/or requests a hearing within the period of time allotted in 20 C.F.R. § 30.310, or if the claimant waives any objection to all or part of the recommended decision, the Final Adjudication Branch (FAB) will issue a decision accepting the recommendation of the district office, either whole or in part." 20 C.F.R. § 30.316(a). On December 1, 2004, the FAB received your signed waiver of any and all objections to the recommended decision. After considering the evidence of record, your waiver of objection, and the NIOSH report, the FAB hereby makes the following:

FINDINGS OF FACT

1. You filed a claim for benefits under Part B of the EEOICPA on May 28, 2004.

2. YOUR HUSBAND WAS EMPLOYED AT THE PADUCAH GASEOUS DIFFUSION PLANT FOR AT LEAST ONE DAY ON DECEMBER 17, 1954.

....."You submitted a medical rep

(III) Lung pathology consistent with chronic beryllium disease.

(IV) Clinical course consistent with a chronic respiratory disorder.

(V) Immunologic tests showing beryllium sensitivity (skin patch test or beryllium blood test preferred).

The Department of Labor has further stated, "***For beryllium disease prior to January 1, 1993, a specific diagnosis of CBD IS NOT REQUIRED (emphasis added.)"....***

CONCLUSIONS OF LAW

Section 7384s of the Act provides for the payment of benefits to a covered employee, or his survivor, with an "occupational illness," which is defined in § 7384l(15) of the EEOICPA as "a covered beryllium illness, cancer. . .or chronic silicosis, as the case may be." 42 U.S.C. §§ 7384l(15) and 7384s. 42 U.S.C. § 7384l.

PURSUANT TO § 7384L(13)(B) OF THE EEOICPA, TO ESTABLISH A DIAGNOSIS OF CBD BEFORE JANUARY 1, 1993, THE EMPLOYEE MUST HAVE HAD "AN OCCUPATIONAL OR ENVIRONMENTAL HISTORY, OR EPIDEMIOLOGIC EVIDENCE OF BERYLLIUM EXPOSURE; AND (III) ANY THREE OF THE FOLLOWING CRITERIA: (I) CHARACTERISTIC CHEST RADIOGRAPHIC (OR COMPUTED TOMOGRAPHY (CT)) ABNORMALITIES. (II) RESTRICTIVE OR OBSTRUCTIVE LUNG PHYSIOLOGY TESTING OR DIFFUSING LUNG CAPACITY DEFECT. (III) LUNG PATHOLOGY CONSISTENT WITH CHRONIC BERYLLIUM DISEASE. (IV) CLINICAL COURSE CONSISTENT WITH A CHRONIC RESPIRATORY DISORDER. (V) IMMUNOLOGIC TESTS SHOWING BERYLLIUM SENSITIVITY (SKIN PATCH TEST OR BERYLLIUM BLOOD TEST PREFERRED)." 42 U.S.C. § 7384L(13)(B). (emphasis added)

The evidence of record establishes that the employee was a covered beryllium employee who had at least three of the five necessary medical criteria to establish pre-1993 CBD under the EEOICPA. Therefore, you have provided sufficient evidence to establish that your husband was diagnosed with pre-1993 CBD, pursuant to § 7384l(13)(B) of the EEOICPA.

The undersigned has reviewed the facts and the district office's November 30, 2004 recommended decision and finds that you are entitled to $150,000 in compensation.

The decision on the claim that you filed under Part E of the EEOICPA is being deferred until issuance of the Interim Final Regulations.

Washington, DC

Tom Daugherty
Hearing Representative
Final Adjudication Branch

[1] The Paducah Gaseous Diffusion Plant was a DOE facility from 1952 to July 28, 1998 and July 29, 1998 to present (remediation) where radioactive and beryllium material were present, according to the Department of Energy Office of Worker Advocacy Facility List (http://www.hss.energy.gov/HealthSafety/FWSP/Advocacy/faclist/findfacility.cfm).

[2] Per Chapter 2-100.3h (January 2002) of the Federal (EEOICPA) Procedure Manual, "The OWCP may receive evidence from other sources such as other state and federal agencies" to support a claim under the EEOICPA.

[3] Per Chapter 2-700.4 (September 2004) of the Federal (EEOICPA) Procedure Manual, **"To determine whether to use the Pre or Post 1993 CBD criteria, THE MEDICAL EVIDENCE MUST DEMONSTRATE THAT THE EMPLOYEE WAS EITHER TREATED FOR, TESTED OR DIAGNOSED WITH A CHRONIC RESPIRATORY DISORDER. (emphasis added)** If the earliest dated document is prior to January 1, 1993, the pre-1993 CBD criteria may be used. **ONCE IT IS**

ESTABLISHED THAT THE EMPLOYEE HAD A CHRONIC RESPIRATORY DISORDER PRIOR TO 1993, THE CE IS NOT LIMITED TO USE OF MEDICAL REPORTS PRIOR TO 1993 TO MEET THE THREE OF FIVE CRITERIA."
(emphasis added)

[redacted] Lung Disease, Chronic Obstructive Pulmonary Disease (COPD), Bilateral Scarring of Lungs Reflect Compliance With Pre-1993 CBD Criteria

1/ CA-001: Western Baptist Consultation, operation, and x-ray misc. medical reports that includes evidence of X-rays revealing the following, COPD, Bilateral scarring, infiltrate in the right lung base dated February 9, 1988 by Dr. Paul Grumley, Dr. Grumley confirmed in a letter to the family dated January17, 2008 that "the x-ray showed bilateral scarring or possible infiltrates in the right lung base. The newly discovered x-ray evidence obtained from Western Baptist Hospital dated February 13, 1988 x-ray report shows scarring in both lung bases. In addition, the newly obtained X-ray report dated February 15, 1988 shows an infiltrate within the inferior right hilum vs. fibrosis with follow up suggested as stated by William E. Adams.

Conclusion: Compliance pursuit with § 7384L(13)(B), Criteria's I, II, & IV

2/ CA-002, 4 pages, James Bibeault evidence of [redacted] compliance with "Statutory" requirements for Chronic Beryllium Disease (CBD). Factual statements provided by the District Office Director.

Conclusion: Compliance pursuit with § 7384L(13)(B), Criteria's I, II, & IV

3/ CA-003, Dr. Yarborough report dated April 21, 2009 confirming [redacted] COPD.

Conclusion: Compliance pursuit with § 7384L(13)(B), Criteria's I.

4/ CA-004, "Statement of Accepted Facts" (SOAF) that document diagnosed conditions of Emphysema and COPD. Employee was exposed to beryllium in the workplace at PGDP, but not captured in the SOAF.

Conclusion: Compliance pursuit with § 7384L(13)(B), Criteria's I.

5/ CA-005, "Memorandum from DEEOICP Director Peter Turic" dated 8/25/05 regarding casual relationship between respiratory disorders and CBD.

Conclusion: Compliance pursuit with § 7384L(13)(B), Criteria's I.

Request for Approval of Part B & E Compensation for Chronic Beryllium Disease (CBD)

Based on the above medical and x-ray documentation submitted on behalf of claimant Velta Feezor for her deceased husband [redacted], Mrs. [redacted] has met her statutory and regulatory burden of proof that establishes her survivorship benefits and compensation for EEOICPA Part B in the amount of $150,000.00 and Part E CBD claim for $125,000.00 .

Please feel free to contact me at 270-559-1752 or 270-450-0850.

Sincerely,

Gary S. Vander Boegh
"Authorized Representative"
Vice President- Commonwealth Environmental Services, LLC.

Cc. Honorable Secretary of Labor Hilda Solis w/Attachments (202) 693-6111

U.S. Department of Labor
200 Constitution Avenue, NW
Room S-2018
Washington, DC 20210

Malcolm Nelson, EEOICP Ombudsman (by email and facsimile)
David Nolan, Esq. (by email w/attachments)

Claim for Survivor Benefits Under the Energy Employees Occupational Illness Compensation Program Act

U.S. Department of Labor
Employment Standards Administration
Office of Workers' Compensation Programs

Note: Provide all information requested below. Do not write in the shaded areas.

OMB Number: 1215-0197
Expiration Date: 08/31/2010

Deceased Employee Information (Please Print Clearly)

1. Name (Last, First, Middle Initial)	2. Sex	3. Social Security Number
[redacted] [redacted]	☒ Male ☐ Female	[redacted]4992

4. Date of Birth	5. Date of Death	6. Was an autopsy performed on the employee?
11 (Month) 23 (Day) 1916 (Year)	02 (Month) 26 (Day) 1988 (Year)	☐ YES - List Medical Facility: ☒ NO ☐ DON'T KNOW

Survivor Information (Please Print Clearly)

7. Name (Last, First, Middle Initial)	8. Sex	9. Social Security Number
[redacted] [redacted] R	☐ Male ☒ Female	[redacted]-6785

10. Date of Birth	11. Your relationship to the deceased employee
12 (Month) 28 (Day) 1923 (Year)	☒ spouse ☐ child ☐ step-child ☐ parent ☐ grandparent ☐ grandchild ☐ Other:

12. Address (Street, Apt. #, P.O. Box)	13. Telephone Numbers
7560 Benton Road	a. Home: (270) 898 - 2028
(City, State, ZIP Code)	
Paducah KY 42003	b. Other: ( ) -

14. Identify the Diagnosed Condition(s) Being Claimed as Work-Related (check box and list specific diagnosis)

	15. Date of Diagnosis Month	Day	Year
☐ **Cancer** (List Specific Diagnosis Below)			
a.			
b.			
c.			
☐ **Beryllium Sensitivity**			
☒ **Chronic Beryllium Disease** (CBD)	02	09	1988
☐ **Chronic Silicosis**			
☐ **Other Work-Related Condition(s) due to exposure to toxic substances or radiation** (List Specific Diagnosis Below)			
a.			
b.			
c.			

Awards and Other Information

16. Did the employee work at a location designated as a Special Exposure Cohort (SEC)?	☒ YES ☐ NO
17. Have you or the deceased employee filed a lawsuit seeking either money or medical coverage for the claimed condition(s)?	☐ YES ☒ NO
18. Have you or the deceased employee filed any workers' compensation claims in connection with the claimed condition(s)?	☐ YES ☒ NO
19. Have you, the deceased employee, or another person received a settlement or other award in connection with the above claimed condition(s)?	☐ YES ☒ NO
20. Have you either pled guilty or been convicted of any charges connected with an application for or receipt of federal or state workers' compensation?	☐ YES ☒ NO
21. Have you or the employee applied for an award under Section 5 of the Radiation Exposure Compensation Act (RECA)? **If yes, provide RECA Claim #:**	☐ YES ☒ NO
22. Have you or the employee applied for an award under Section 4 of the Radiation Exposure Compensation Act?	☐ YES ☒ NO

Other Potential Survivors

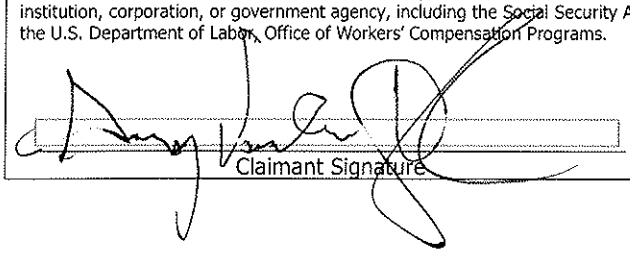
23. Are you aware of any person(s) who may also qualify as a survivor of the deceased employee? ☐YES ☒NO

If YES, please provide the following:

	Name	Relationship to the deceased employee	Address	Phone Number(s)
a.			†	Home: Other:
b.			†	Home: Other:
c.			†	Home: Other:
d.			†	Home: Other:
e.			†	Home: Other:
f.			†	Home: Other:
g.			†	Home: Other:
h.			†	Home: Other:
i.			†	Home: Other:
j.			†	Home: Other:

Survivor Declaration

Any person who knowingly makes any false statement, misrepresentation, concealment of fact, or any other act of fraud to obtain compensation as provided under EEOICPA or who knowingly accepts compensation to which that person is not entitled is subject to civil or administrative remedies as well as felony criminal prosecution and may, under appropriate criminal provisions, be punished by a fine or imprisonment or both. Any change to the information provided on this form once it is submitted must be reported immediately to the District Office responsible for the administration of the claim. I hereby make a claim for benefits under EEOICPA and affirm that the information I have provided on this form is true. If applicable, I authorize the Department of Justice to release any requested information, including information related to my RECA claim, to the U.S. Department of Labor, Office of Workers' Compensation Programs (OWCP). Furthermore, I authorize any physician or hospital (or any other person, institution, corporation, or government agency, including the Social Security Administration) to furnish any desired information to the U.S. Department of Labor, Office of Workers' Compensation Programs.



Claimant Signature

11/23/2010

Date

Resource Center Date Stamp

CES0133

Internal Medicine Group

Professionals Caring For You!

Mae Fischer, M.D.
Ryan Frazine, M.D.
Paul J. Grumley, M.D., F.A.C.P.

Polly J. LeBuhn, M.D.
James H. Long, Jr., M.D.
Joseph M. Pittard, M.D.
Shane Carter, Chief Executive Officer

Richard D. Smith, M.D.
David G. Stricklin, M.D.
Jesse Wallace, M.D., F.A.C.P.

January 17, 2008

RE: [redacted]
DOB: 11/23/1916

To Whom It May Concern:

[redacted] was a patient I took care of about 20 years ago. He died on February 26, 1988. The family asked me to comment on the possibility that he had chronic obstructive pulmonary disease and that this was related to his work at the Atomic Energy Plant.

The note of February 9, 1988 says that he has chronic obstructive pulmonary disease and a chest ray showed bilateral scarring or possible infiltrate in the right lung base. Unfortunately, that particular chest x-ray has been purged and I do not have an official report of that. I do not have any pulmonary function studies to document that chronic obstructive pulmonary disease was present. He was a smoker, but he smoked only in his youth while in the service.

He had multiple other medical problems including diabetes and significant atherosclerosis. He died of complications following a stroke and a carotid endarterectomy in 1988. The note of February 9, 1988 suggests that he had known chronic obstructive pulmonary disease and, of course, chronic obstructive pulmonary disease can be exacerbated by the effects of exposures to industrial toxins.

If you need further information concerning this patient, I would be happy to attempt to help.

Sincerely,

Paul J. Grumley, M.D., F.A.C.P.

PJG:mmw

[redacted]

Transmitted to DOL-DO
FEB 0 1 2008

We Practice Medicine for Individual Reasons. You!

WESTERN BAPTIST HOSPITAL
2501 KENTUCKY AVE.
PADUCAH, KENTUCKY 42001

NAME: [redacted]
CHART: 7594801-04
DOCTOR: Grumley/Jaafar
ADM: 02/09/88
DIS:
ROOM: 492-1
PAGE: 1

CONSULTATION

ADMITTING PHYSICIAN:
Paul J. Grumley, M.D.

HISTORY & PHYSICAL:
This is a 71 year old white gentleman who was admitted to the hospital by Dr. Grumley with the diagnosis of:
1. Severe vascular thrombosis.
2. Atherosclerotic cardiovascular disease.
3. Non-insulin dependent diabetes mellitus.
4. Dementia.

The patient, after admission, had a stroke. He also underwent an emergency carotid surgery to prevent further extension and worsening of his stroke. Postop, the patient has been very congested, coughing. The nurses stated that they had been sucking a lot of phlegm out of him, mostly yellowish, thick and purulent in nature. The patient used to be a heavy smoker in the past.

PHYSICAL EXAMINATION:
Examination revealed an elderly male, poorly responsive except for pain. His pupils were reactive to light.
NECK: Revealed evidence of recent carotid surgery on the right.
CHEST: Exam revealed diminished breath sounds with a lot of rhonchi in both sides.
HEART: Revealed irregular rhythm, showing atrial fib on the monitor. No gallop was present.
ABDOMEN: Very soft.
EXTREMITIES: Unremarkable.

His ABGs revealed a pH of 7.45, pCO2 of 37, pO2 of 111 on oxygen.

Chest x-ray revealed the following:
1. COPD.
2. Bilateral scarring.
3. Possible new infiltrate in the right lung base.

IMPRESSION:
1. Chronic obstructive lung disease.
2. CVA.
3. Atrial fibrillation, new.
4. History of ASHD.

CONTINUED:
(CONSULTATION)

CLAIMANT ATTACHMENT 001
PAGE NO. 286

CHART COPY

WESTERN BAPTIST HOSPITAL
2501 KENTUCKY AVE.
PADUCAH, KENTUCKY 42001

NAME: [redacted]
CHART: 7594801-04
DOCTOR: Grumley/Jaafar
ADM: 02/09/88
DIS:
ROOM: 492-1
PAGE: 2

CONSULTATION (CONTINUED)

RECOMMENDATIONS:
1. Agree with Dr. Luigs and Dr. Grumley to put the patient on Digitalis.
2. Continue IPPB with Proventil.
3. Add Mucomyst twice a day.
4. Cephobid one gram every eight hours to cover for possible aspiration.
5. Consider bronchoscopy if he continues to have a lot of secretions.

Thank you for asking me to see this patient with you.

CLAIMANT ATTACHMENT 001
PAGE NO. 386

M. Y. Jaafar, M.D.

MYJ/021588
(CONSULTATION)

WP43.021588.1

CHART COPY

WESTERN BAPTIST HOSPITAL
WHERE YOUR NEEDS COME FIRST
2501 KENTUCKY AVE
PADUCAH, KENTUCKY 42001

NAME: [redacted]
CHART: 7594801-04
DOCTOR: Jaafar
ADM: 02/09/88
DIS:
ROOM: 350-1

OPERATION

DATE:
02-22-88

ADMITTING PHYSICIAN:
Paul J. Grumley, M.D.

PREOPERATIVE DIAGNOSIS:
Possible aspiration.

POSTOPERATIVE DIAGNOSIS:
SAME.

PROCEDURE:
Bronchoscopy.

The patient was preopted on the floor and he was taken down to the endoscopy room. Local anesthetic was applied to his nose and pharynx. The tracheobronchial tree was inspected carefully. When the scope was passed through the nose there was little cough reflex in the larynx. The trachea and bronchial tree revealed severe inflammatory changes. A lot of secretion suctioned out. A large mucous plug was also removed. Bronchial lavaging was done. More than 200 cc of purulent secretions suctioned out. The patient did well without any complications.

M. Y. Jaafar, M.D.

MYJ/022288
(OPERATION)

WP31.022388.1

CHART COPY

CLAIMANT ATTACHMENT 001
PAGE NO. 486

NAME: [redacted]
CHART: 7594801-04
DOCTOR: Grumley
ROOM: 492-1
XRAY #: 759480

WESTERN BAPTIST HOSPITAL
WHERE YOUR NEEDS COME FIRST
2501 KENTUCKY AVE
PADUCAH, KENTUCKY 42001

X-RAY REPORT

ADMITTING PROBLEM/HISTORY:
CVA.

DATE:
02/15/88

PORTABLE CHEST:
Today's study is compared to the preceeding exam of 02/13. Minimal atelectasis about the inferior right hilum is felt to be present. Some early pneumonia is not excluded in this region. A follow up is suggested.

IMPRESSION:
Minimal early infiltrate within the inferior right hilum vs. fibrosis with follow up suggested.

WEA/021688
(X-RAY REPORT)

WP32.021688.1X

CHART COPY

WALKER

William E. Adams, M.D.

CLAIMANT ATTACHMENT 001
PAGE NO. 5 of 6

NAME: [redacted]
CHART: 7594801-04
DOCTOR: Grumley
ROOM: 492-1
XRAY #: 759480

WESTERN BAPTIST HOSPITAL
WHERE YOUR NEEDS COME FIRST
2501 KENTUCKY AVE.
PADUCAH, KENTUCKY 42001

X-RAY REPORT

ADMITTING PROBLEM/HISTORY:
CVA; stroke

DATE:
02/13/88

PORTABLE AP CHEST, 09:45 HOURS:
Comparison is made to 02/09/88.

Some scarring is seen in the lung bases. The chest is essentially unchanged since 02/09/88.

IMPRESSION:
Minimal scarring both lung bases. No active disease.

GH 1/02

GWH/021388
(X-RAY REPORT)

Gary W. Heath, M.D.

WP36.021388.6X

CHART COPY

CLAIMANT ATTACHMENT 001
PAGE NO. 6 & 6

U.S. Department of Labor

Employment Standards Administration
Office of Workers' Compensation Programs
Division of Energy Employees' Compensation
400 West Bay Street, Suite 722
Jacksonville, FL 32202

May 17, 2009

Employee: [redacted]

[redacted]
Paducah, KY 42003

Dear [redacted]

I am writing in reference to your claim for survivor benefits under Part E of the Energy Employees' Occupational Illness Compensation Program Act (EEOICPA). By letter dated March 13, 2009, you requested that your claim be reopened for the claimed condition of chronic obstructive pulmonary disease (COPD).

20 C.F.R. § 30.320 provides that a claimant may file a written request that the Director of the Division of Energy Employees Occupational Illness Compensation (DEEOIC) reopen his or her claim. The decision whether or not to reopen a claim under this section is solely at the discretion of the Director. The authority to review and deny certain requests has been delegated by the Director, DEEOIC, to the District Director having jurisdictional authority over the case. Pursuant to this authority, I have reviewed your request and have outlined my findings below.

The medical evidence of record used to adjudicate your survivor claim established the employee, [redacted], was diagnosed with COPD on February 9, 1988. The employment evidence of record supports your late husband worked as an electrician for various Department of Energy (DOE) subcontractors for intermittent periods at the Paducah Gaseous Diffusion Plant from 1951 to 1959.

On March 3, 2008, the Final Adjudication Branch (FAB) issued a Final Decision (Docket Number 10051487-2007) denying your survivor claim under Part E of the EEOICPA. The FAB concluded the evidence was insufficient to establish that it is "at least as likely as not" that exposure to a toxic substance at a DOE facility was a significant factor in aggravating, contributing to, or causing the employee's death.

CLAIMANT ATTACHMENT 002
PAGE NO. 184

On January 28, 2008, the Paducah Resource Center received your new Claim for Survivor Benefits, Form EE-2 for emphysema and COPD. You submitted a letter from Dr. Paul J. Grumley dated January 17, 2008, in which he confirms that the employee had COPD and that COPD can be exacerbated by the effects of exposure to industrial toxins. The letter states that the employee died of complications following a stroke and a carotid endarterectomy in 1988.

On May 05, 2008, the Jacksonville District Office issued a Recommended Decision denying your claim for emphysema and COPD under Part E of the Act because the evidence is insufficient to establish that the employee's COPD was a significant factor in aggravating, contributing to, or causing the employee's death.

On August 27, 2008, FAB issued a Final Decision Following A Review of the Written Record (Docket Number 10041469-2008) indicating that on June 13, 2008, they received your letter of objection dated May 12, 2008 and that your objections were taken into consideration however; determined they were not sufficient to modify the Recommended Decision of May 05, 2008. The FAB denied your survivor claim for the condition of emphysema and COPD.

The FAB noted the final decision was based on the evaluation conducted by a District Medical Consultant (DMC) dated November 28, 2007. The report determined that it is not "at least as likely as not" that exposure to a toxic substance at a DOE facility was a significant factor in aggravating, contributing to, or causing the employee's emphysema, COPD, coronary artery disease, and cerebral artery thrombosis or that the employee's emphysema, and scars on lungs were a significant factor in causing, contributing to, or aggravating the employee's death.

On March 13, 2009, the Paducah Resource Center received a letter from you requesting a reopening of your claim, specifically making your request relative to your late husband's condition of COPD. Along with your reopening request you submitted medical research documents that you believe support your position that your late husband's COPD was a significant contributing factor in his death, which was listed as stroke and heart attack on his death certificate. You referenced that you previously submitted medical records from Dr. Grumley that provided evidence for his COPD, based upon an x-ray taken before his death in 1988.

As part of our review of your claim and reopening request, a search of the Department of Labor Site Exposure Matrices (SEM) was again completed in an effort to identify any new evidence not previously identified. This search revealed the employee was potentially exposed to ammonia, asbestos, cadmium, and

CLAIMANT ATTACHMENT 002
PAGE NO. 2 of 4

nitrogen dioxide while working as an electrician at the Paducah Gaseous Diffusion Plant.

On April 3, 2009, your late husband's file was submitted to the DMC for review, along with the newly submitted medical research documents regarding the condition of COPD. In his report dated April 21, 2009, the DMC opined:

> "As pointed out by the DMC in 2007, there is no history of acute overexposures to the lung irritants, ammonia, and nitrogen dioxide. Also there is no history of kidney disease reported that could suggest cadmium toxicity.
>
> ...Dr. Grumley wrote the History and Physical Examination of 02/09/1988 but he did not mention of any lung disease, and Mr. Freezor's medications did not include any pulmonary drugs upon admission. He died 17 days after the diagnosis (actually, diagnostic impression) of COPD. There is no ICD-9 code of COPD disorders on the final hospitalization summary. A diagnosis of COPD requires confirmation by spirometry (ATS ERS, 2004, section 2.3, page 9), but no pulmonary function tests are in the records. A lung CT scan is not found in the records to help delineate the correct diagnosis.
>
> "The diagnosis [of COPD] requires spirometry; post-bronchodilator FEV1/forced vital capacity <0.7 confirms the presence of airflow limitation that is not fully reversible" (ATS ERS, 2004, section 2.3., page 9) Hogg (2008) has described the anatomical changes of lungs and airways in COPD. Emphysema must be distinguished from the honeycomb changes observed in advanced asbestosis (Sporn and Roggli, 2004), but in this case there was minimal bibasilar scarring reported but no emphysematous findings.
>
> COPD was diagnosed in 1988 by a consulting pulmonologist but apparently this diagnosis was not accepted by the attending physician based on his exclusion of any COPD diagnosis on the hospital summary and death certificate.
>
> In my opinion, it is <u>not</u> "at least as likely as not" that exposure to toxins at the Paducah Gaseous Diffusion Plant was a significant factor in causing, contributing to, or aggravating the employee's COPD."

In addressing the question as to whether exposure to toxic substances at a DOE facility was a significant factor in aggravating, contributing, or causing the death of the employee, the DMC opined:

> "The second question about the employee's COPD or its treatment having caused, contributed to, or aggravated his death is moot because of the lack of its association with his employment at Paducah Gaseous Diffusion Plant."

Based upon a thorough review of the evidence of record, medical research documents submitted with your request, a new search of SEM, and a referral for medical opinion, we were unable to establish any new substantive facts that would change the outcome of your claim. Consequently, your reopening request does not contain the requisite evidence to warrant a reopening of your clam for benefits.

Accordingly, I regret that the request to reopen this claim must be denied. If you have any questions about this letter, you may contact me toll free at: 1-877-336-4272.

Sincerely,

JAMES R. BIBEAULT
District Director
Division of Energy Employees'
Occupational Illness Compensation

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Charles M. Yarborough, M.D., M.P.H.
4400 Chalfont Place, Bethesda, MD 20816 (301) 320-3171

MEDICAL CONFIDENTIAL

April 21, 2009

Joyce Perret
Claims Examiner
Office of Workers' Compensation Programs
Division of Energy Employees' Compensation
U.S. Department of Labor
400 West Bay Street, Suite 722
Jacksonville, FL 32202

Re: [redacted] (File Number 4[redacted]992)

Dear Ms. Perret:

This letter is in response to the inquiry regarding Brooks E. Freezor (date of birth: Nov. 23, 1916; date of death: Feb. 26, 1988). I have reviewed the details in the Statement of Accepted Facts (SOAF), read several times all records and documents that were sent (approximately 1½ inches thick), including a DMC opinion in 2007 regarding COPD and claimant's "enclosed medical research documentation," and conducted a relevant, scientific literature search for current full epidemiologic, toxicologic and review articles identified by PubMed search, in references, and of relevant authoritative textbooks and professional society and government agency opinions, reports and guidelines.

I understand that the questions for my expert medical opinion are:

1. In your opinion, is it "at least as likely as not" that exposure to toxins at the Paducah Gaseous Diffusion Plant [PGDP] was a significant factor in causing, contributing to, or aggravating the employee's COPD?

2. If so, do you believe his COPD or treatment thereof also caused, contributed to, or aggravated the employee's death?

For the purposes of my review, I interpret the terms as noted in the following parentheses according to the August 2008 version of the DMC Handbook: *aggravating* (i.e., the worsening of a previously existing disease, condition or physical impairment by a workplace exposure or event.); *contributing to* (i.e., an increased risk of illness, progression or acceleration the adverse outcome); directly *causing* (i.e., clear, linear, one-on-one relationship between the exposure and the illness or death in the absence of other diseases or conditions).

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I certify that I am an expert in the required areas of medical expertise for the issues raised in this case and this is my objective medical opinion provided in accordance with the DEEOIC program procedures and guidelines. I also certify that I neither have now, nor have had in the past, any relationship with the claimant, his/her physicians, their attorneys, representatives or any employee, employer, manufacturer or entity that may be connected with this case that would influence my opinion in any way. I also certify that my opinion was not influenced by any financial consideration that may benefit me, my family or my heirs.

Pertinent Medical History

The death certificate of the employee lists sepsis as the immediate cause preceded by aspiration and cerebrovascular thrombosis, with the underlying cause being coronary artery disease. Dr. Paul Grumley lists no other conditions as significantly contributing to death. No autopsy was performed. The hospital discharge summary, signed by Dr. Grumley, has 3 principal diagnoses without comorbidities. Absent from the hospital discharge summery is a diagnosis code for COPD (491, chronic bronchitis; 492, emphysema; 496, COPD). The principal diagnoses are: atherosclerotic cardiovascular disease (CVD), non-insulin dependent diabetes mellitus and dementia.

Dr. Grumley wrote the last History and Physical Examination on 02/09/1988. In that document he describes hemiparesis of recent onset, atherosclerotic cardiovascular disease with known, inoperable coronary stenoses and occlusive femoral artery disease, and progressive dementia. Past medical history included non-insulin dependent diabetes mellitus. There was no mention of lung disease, and his medications did not include any pulmonary drugs. Dr. Grumley noted a hospitalization one month earlier, but I did not find those records. Neither spirometry nor lung CT scan is found in the records.

A consultation by M.Y. Jaafar, M.D., apparently on 02/15/1988, does not mention any lung symptoms or prior pulmonary disorder. He was consulted for the patient's post-operative lung congestion and coughing. He describes a chest x-ray film (the technique and date are not noted) as revealing COPD, bilateral scarring and possible new infiltrate in the right lung base. (The claimant writes that "emphysema – scars on lungs" was diagnosed in 1978.) Pleural plaques are not noted to be present. Previous films are not reviewed. He recommended continuing Proventil® with the IPPB and adding Mucomyst®.

Dr. Health interpreted a portable chest x-ray film done on 02/13/1988 and compared it to one of 02/09/1988. His impression was unchanged minimal scarring of both lung bases without active disease. Dr. Jaarfar performed 2 bronchoscopies and found severe bronchial inflammation and copious purulent secretions, and he recommended antibiotic therapy.

A District Medical Consultant (DMC) opined on 11/28/2007 that the observation of "his scars on the lungs" – but not emphysema or COPD – was at least as likely as not significantly associated with exposure to toxic substances while working at PGDP. Furthermore, the DMC believed the lung scars (called "pulmonary fibrosis" on page 2 of the report) were manifestations of asbestosis, resulting at least in part, from exposures to asbestos fibers for 5½ years at PGDP in the 1950's.

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Smoking History

Dr. Jaafar wrote that the patient had been a heavy smoker. Dr. Grumley indicated that he stopped several years before his final hospitalization, implying he ceased in the early 1980's when he was approximately 65 years old. This recounting is inconsistent with the claimant's history (see SOAF).

Work History

See SOAF for work timeframe at the site for this claim of 1950s at the PGDP. He worked as an electrician/wireman while there. The death certificate has that his usual occupation was as an electrician working in the construction industry.

Potential Job Exposures

Toxic substances are noted in the SOAF as exposures. Asbestos [fibers] is one of the substances. Also listed are ammonia, cadmium and nitrogen dioxide. His use of personal protective equipment to reduce exposures to inhaled particles (specifically respirator protection) was recorded as infrequent or never. An occupational history questionnaire and a Site Exposure Matrix (SEM) are included in the provided documents.

Case Evaluation: COPD

In addressing the specific question on lung cancer for my expert opinion, I consider the following 5 aspects for this case:

1. Exposure. The accepted exposure to asbestos and his job as wireman/electrician in the 1950s suggest the possibility of significant exposures to asbestos fibers whether chrysotile and/or amphiboles. Pleural involvement is a hallmark of asbestos exposure whereas it is unusual in other interstitial lung disorders (Zenz, 1994; Levy and Wegman, 1995). Calcified diaphragmatic pleural plaques are considered pathognomonic of asbestos exposure (see Churg, 1982), but the claimant did not have this finding according to the radiology report and pulmonologist's review in 1988. Even if present, plaques do not necessarily imply asbestos-related interstitial lung disease (asbestosis). The higher the exposure, the more likely there will be extensive calcified pleural plaques (Rom, 2007), yet the claimant had no calcified plaque observed. This fact implies that asbestos exposures were not especially elevated.

The claimant has an appropriate latency for the diagnosis of asbestosis, which becomes evident only after an appreciable time period, in general 20 - 30 years after peak asbestos exposure and diagnosis. Duration and intensity of exposure influence the length of the latency period, with short-term, high-intensity exposures probably having shorter latency periods than prolonged, lower intensity exposures. A significant exposure can be defined as at least several months' exposure to visible dust that began more than 10 years earlier (ATS, 2004; ATSDR, 2007a; O'Reilly et al., 2007; King, 2007). The lack of asbestosis diagnosis by his doctor in this case is therefore not due to insufficient latency in this case.

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As pointed out by the DMC in 2007, there is no history of acute overexposures to the lung irritants, ammonia and nitrogen dioxide. Also there is no history of kidney disease reported that could suggest cadmium toxicity.

2. Health Effect (Outcome). As noted above, Dr. Grumley wrote the History and Physical Examination of 02/09/1988 but he did not mention of any lung disease, and [redacted] medications did not include any pulmonary drugs upon admission. He died 17 days after the diagnosis (actually, diagnostic impression) of COPD. There is no ICD-9 code of COPD disorders on the final hospitalization summary. A diagnosis of COPD requires confirmation by spirometry (ATS ERS, 2004, section 2.3, page 9), but no pulmonary function tests are in the records. A lung CT scan is not found in the records to help delineate the correct diagnosis.

"The diagnosis [of COPD] requires spirometry; post-bronchodilator FEV1/forced vital capacity <0.7 confirms the presence of airflow limitation that is not fully reversible" (ATS ERS, 2004, section 2.3, page 9). Hogg (2008) has described the anatomical changes of lungs and airways in COPD. Emphysema must be distinguished from the honeycomb changes observed in advanced asbestosis (Sporn and Roggli, 2004), but in this case there was minimal bibasilar scarring reported but no emphysematous findings.

COPD was diagnosed in 1988 by a consulting pulmonologist but apparently this diagnosis was not accepted by the attending physician based on his exclusion of any COPD diagnosis on the hospital summary and death certificate.

Plausible Linkage. The claimant's job of electrician has been classified as a construction worker for epidemiologic study (Roggli and Sharma, 2004), and construction workers are potentially at increased risk of COPD (Meldrum et al. 2005). Beckett (2000) published a list of selected common causes of occupational airway disease. From Table 2 of his review, COPD and chronic airflow limitation may arise from overexposures to cadmium fumes (causes emphysema, and is used in electronics, metal plating, and batteries) as listed in the SOAF, as well as crystalline silica (causes chronic airflow obstruction as seen in sandblasting and underground mining), cotton dust, coal dust, and toluene diisocyanate that are not listed in the SOAF.

The construction trades most at risk from asbestos have been electricians, insulators, plumbers and pipefitters, and sheet metal workers (NIOSH, 2004). Mean tissue asbestos body count and uncoated asbestos fibers of lungs of construction workers (including electricians) are 70-fold and 5-fold, respectively, those of the reference population (Roggli, 2004). Amati et al. (2008) observed that electricians (and maintenance workers and pipe fitters) were exposed to a higher cumulative dose of asbestos fibers than other groups.

4. Judgment of Each Causal Element.

Ammonia. The extent of injury produced by exposure to ammonia depends on the duration of the exposure, the concentration of the gas, and the depth of inhalation. Even fairly low airborne concentrations (50 ppm) of ammonia produce rapid onset of eye, nose, and throat irritation; coughing; and narrowing of the bronchi. More severe clinical signs include immediate narrowing of the throat and swelling, causing upper airway obstruction and accumulation of

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fluid in the lungs. Repeated exposure to ammonia may cause chronic irritation of the respiratory tract. Chronic cough, asthma and lung fibrosis have been reported (ATSDR, 2007b). The claimant's records have no evidence to support repeated symptomatic exposures to ammonia gas.

Ammonia and nitrogen dioxide (see below) are pulmonary irritants that can produce permanent residual lung damage and dysfunction. Acute overexposures to these substances result in respiratory distress requiring urgent medical evaluation and treatment (Schwartz, 1994). However there is no record of this event occurring.

Asbestos. The American Thoracic Society issued a Statement on non-malignant disease related to asbestos, noting that the role of asbestos as a cause of airway obstruction has been controversial. In general, the magnitude of the asbestos effect on airway function is small. Short duration and low cumulative exposure, which is the likely situation in this case, are less likely to produce significant obstructive abnormality. Tobacco smoking is the predominate cause of chronic airway obstruction in asbestos-exposed workers who smoke (ATS, 2004).

Asbestosis is a medical condition known to have a threshold that is believed to occur only when cumulative fiber exposures to all asbestos fibers (f) exceed 25–60 f/cc-years (Doll and Peto, 1985; Meldrum, 1996). Mossman and Churg (1998) conclude that "asbestosis does not appear until a threshold exposure has been reached...," and "[e]pidemiological studies indicate very clearly that the development of asbestosis requires heavy exposure to asbestos and provide strong evidence that there is a threshold fibre dose below which asbestosis is not seen..." Writing for the Health & Safety Executive in the United Kingdom, Meldrum (1996) concluded that there would be a threshold below which no radiological or clinical manifestation of asbestosis occurs, and that the level depends on fiber type and size distribution.

Cadmium. COPD and pulmonary fibrosis is found among workers overexposed to cadmium dust and fume by breathing high levels over long time periods (Beckett, 2000, table 2; Kusaka et al., 2001). Furthermore, the kidney is the critical target organ for cadmium toxicity in man; i.e., it is the site of earliest functional disturbances (Zenz, 1994, p. 482). Avoidance of kidney damage is the basis for setting the accepted exposure limit to this substance (Kusaka et al., 2001). It is expected that increased urine excretion of specific proteins would be found before any significant lung dysfunction from cadmium toxicity became evident (Zenz, 1994, p. 483), but this is not found in the claimant's medical records. There is no diagnosis for the claimant of a renal disorder to implicate cadmium-related disease.

Nitrogen dioxide. Nitrogen dioxide is a pulmonary irritant that can produce permanent residual lung damage and dysfunction. Acute overexposure can result in respiratory distress requiring urgent medical evaluation and treatment (OSHA, 2008).

Animal toxicology subchronic studies are available that are applicable. A study assessed the relationship between nitrogen dioxide inhalation and the development of pulmonary emphysema and investigated how the severity of preexisting emphysema may be augmented by a subchronic exposure to a relatively high concentration of nitrogen dioxide. From the data, as well as histologic examinations of lung sections for evidence of emphysema, the authors conclude that (1) a subchronic, moderately high level of NO_2 exposure does not produce an irreversible

emphysematous lesion in the rat model and (2) high exposure of rats (35 ppm, 65.3 mg/m^3) does not potentiate induced emphysema or bring about a progression in preexisting emphysema (Stavert et al., 1986). In another animal study, four weeks of NO_2, cigarette smoke, or their combination was not sufficient to induce significant emphysema, nor did it lead to increased numbers of white blood cells in lung tissue (Brandsma C-A et al, 2008). Thus it is unlikely that there is any association with exposures to low air levels of this chemical over a few years occurring decades ago in this case.

5. Consideration of Alternative Explanations. Chronic obstructive pulmonary disease (COPD) is defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2006 Guidelines as "a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases." COPD is usually caused by cumulative exposure to tobacco smoke, which in the western world is the dominant factor in up to 90% of cases, but occupational dusts and chemicals, and indoor or outdoor air pollution may also play a role (ATS and ERS, 2004; Halpin, 2008).

The claimant was a former smoker, but when he quit and how much he smoked is unclear. The smoking history of the employee implicates long-term inhalation of cigarette smoke as a cause of the COPD if the condition was truly present. Moreover, tobacco smoking is the predominate cause of chronic airway obstruction in asbestos-exposed workers who smoke (ATS, 2004).

The claimant supplied an incomplete printout of a medical research article with the request for another review of the case. I obtained and read the full paper by Sidney et al (2005). As the authors point out there is little prospective research on the risk of CVD among those patients having known COPD. There is considerable debate regarding the strongest predictors of mortality in COPD. Key predictors include advanced age, smoking status, low forced expiratory volume in 1 second (FEV_1), peak expiratory flow, low arterial partial pressure of oxygen (PaO_2), low body mass index and reduced exercise capacity.

The BODE index – a composite index reflecting the multicomponent nature of COPD – has also proved to be a good predictor of mortality as it incorporates systemic as well as pulmonary characteristics of COPD. BODE stands for Body mass index, airflow Obstruction, Dyspnea and Exercise capacity. There is insufficient data in this case to calculate the survival index. Coexisting cardiac disease, such as in this case, is a strong predictor of death within 6 months among COPD patients (Weiss, 2009).

[redacted] would not have qualified to be case for this very large study by Sidney et al. in that he did not have an ICD-9 diagnosis of COPD upon hospital discharge or mortality, was not taking 2 COPD medications within the specified time periods, and did not have 12 months follow-up time (3 years was average). Also he was 72 years old when diagnosed/expired [redacted] Sidney et al. noted the highest risk in patients less than 65 years of age. A major limitation in their study was that subjects' smoking histories were unavailable to analyze. As they state in the paper, cigarette smoking is the most powerful predictor for COPD, and it is the

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most important risk factor in CVD. Not being able to accurately control for smoking might have significantly biased the results and at least makes drawing definitive conclusions problematic.

The other paper supplied by the claimant is from www.swedish.org apparently. It explains symptoms of COPD but does not discuss the risk of CVD from COPD.

COPD frequently coexists with other chronic conditions and the presence of these co-morbidities adversely affects outcome. Some of the co-morbidities share a common etiology with COPD and in the past it has been assumed that this explained their coexistence. Recently, however, it has been recognized that additional systemic effects of COPD could include an increased risk of myocardial infarction (MI) and other cardiovascular manifestations, most of which are likely to be associated with an ongoing low-grade systemic inflammation (Halpin, 2008).

There is some evidence that there may be a "COPD effect" that contributes to CVD in this condition (Maclay et al., 2007). However, accepted major coronary heart disease (CHD) risk factors include age, sex, smoking, blood pressure, lipoproteins, and cholesterol, but COPD is not among them. Future studies will need to determine the absolute risk for developing CHD in patients with COPD (Hunninghake, 2005). Of course, this consideration of possible additional risk from COPD for CVD incidence is moot for the evaluation of this case if the COPD is not judged to be related to his employment at Paducah Gaseous Diffusion Plant.

Individuals with the following clinical characteristics are at very high risk for the development of cardiovascular disease events and do not need risk estimated: established cardiovascular disease; diabetes mellitus (type 1 or 2); chronic kidney disease; hereditary dyslipidemias known to be associated with the premature development of atherosclerotic events. COPD is not identified as a factor for risk estimation of CVD events (Wilson, 2009). The employee had 2 of the known medical conditions, each of which obviate a risk estimation, and a CVD event could be anticipated.

Summary

In my opinion, it is <u>not</u> "at least as likely as not" that exposure to toxins at the Paducah Gaseous Diffusion Plant was a significant factor in causing, contributing to, or aggravating the employee's COPD.

1. COPD was diagnosed 5 decades after verified employment and clinically by smoking history and chest film interpretation only 17 days before his death from sepsis after an operation and post-operative pulmonary aspiration.

2. The "minimum" scarring of the lungs noted by the radiologist and pulmonologist in 1988, the claimant's report of this finding first being noted in 1978, along with potentially significant asbestos exposures at PGDP, suggest the presence of mild, subclinical interstitial lung disease rather than indicative of COPD.

3. Although stated as a diagnostic impression by a consulting pulmonologist, the attending physician apparently disagreed with this new diagnosis (COPD) as evidenced by his exclusion of any COPD diagnosis on the hospital summary and death certificate. He did not include COPD or any lung disease on his hospital admission note.

4. Overexposure to inhaled asbestos fibers, especially amphiboles, can cause interstitial lung disease, but an association of asbestos exposure with COPD remains controversial.

5. There is no history of serious overexposure to the lung irritants in this case, ammonia and nitrogen dioxide, to suggest the presence of long-term adverse effects to the lung airways.

6. There are no reports of any renal damage, the target organ of cadmium toxicity, which might imply an association with work-related exposures to cadmium.

The second question about the employee's COPD or its treatment having caused, contributed to, or aggravated his death is moot because of the lack of its association with his employment at PGDP in my opinion. The employee had 2 of the 4 major known medical conditions (i.e., established cardiovascular disease and diabetes mellitus) for having a very high risk for a CVD event. Furthermore, the hypothesis that COPD is itself an independent risk factor is still a research question that requires more studies.

If you have any questions regarding my comments and opinion on this case please contact me, and I will be glad to elaborate.

Sincerely,

Charles M. Yarborough, M.D., M.P.H.

(DEEOIC Provider ID 610994200)

References

Amati M, et al. 2008. Assessment of biomarkers in asbestos-exposed workers as indicators of cancer risk. Mutat Res 655:52-8.

[illegible]an Thoracic Society (ATS). 2004. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med 170:691-715.

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PAGE NO. 888

Statement of Accepted Facts (SOAF)
Revised Format 3/2008

Employee's Name: [redacted]
Claimant Name: [redacted]
File: [redacted]4992
Jacksonville, FL

1. **Employee Information**

   a. Name: [redacted] (deceased)
   b. Claimant: [redacted]
   c. Case File Number: [redacted]4992
   d. Date of Birth: November 23, 1916
   e. Date of Death: February 26, 1988
      i. If deceased, list Cause(s) of Death from Death Certificate
         1. Cause of Death: sepsis
         2. Underlying Cause of Death: aspiration pneumonia
         3. Underlying Cause of Death: cardiovascular thrombosis
         4. Underlying Cause of Death: coronary artery disease
      ii. If deceased, list the claimant: [redacted]
   f. Is the claimant part of an identified Special Exposure Cohort (SEC)? No

2. **Claim History**

Claimant filed a claim October 19, 2005, claiming emphysema (scars on lungs), stroke and heart attack. The file was forwarded to the DMC to determine whether exposure to a toxic substances at the PGDP was at least as likely as not a significant factor in causing, aggravating, or contributing to the employee's death. The DMC opined there was insufficient evidence of exposure meeting the "at least as likely as not" criteria that toxic exposure at a DOE facility was a significant factor in aggravating, contributing to, or causing the employee's death. A final decision was issued denying the claimant for survivor's benefits stating the evidence of record does not establish that it is "at least as likely as not that exposure to a toxic substance at a Department of Energy facility was a significant factor in aggravating, contributing to or causing the employee's death.

On March 3, 2008, a Final Decision was issued denying the claimant for survivor's benefits for the claimed conditions of emphysema (scars on the lungs/COPD), stroke, and heart attack under Part E of the Act.

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PAGE NO. 1 of 3

Statement of Accepted Facts (SOAF)
Revised Format 3/2008

Employee's Name: [redacted]
Claimant Name: [redacted]
File: [redacted]4992
Jacksonville, FL

b. Diagnosed Condition
(provide date of diagnosis for each; if diagnosed condition is skin cancer, provide body location)

i. Diagnosed Condition A (include date of diagnosis and location if applicable): COPD, 2/9/1988
ii. Diagnosed Condition B (include date of diagnosis and location if applicable): Heart Attack (atrial fibrillation, 2/1/1988
iii. Diagnosed Condition C (include date of diagnosis and location if applicable): Stroke, 2/1/1988
iv. Diagnosed Condition D: emphysema-scars on lungs (COPD), 1/1/1978
v. Diagnosed Condition E: atherosclerotic cardiovascular disease
vi. Diagnosed Condition F: cerebral artery thrombosis
vii. Diagnosed Condition G: peripheral vascular disease

The file contains a medical consultation, dated 2/9/1988, giving a diagnosis of severe vascular thrombosis and atherosclerotic cardiovascular disease. The medical documentation also reveals chest x-ray results showing COPD, bilateral scarring, and possible new infiltrate in the right lung base. The medical report (dated 2/9/1988) gives an impression of chronic obstructive lung disease, CVA, atrial fibrillation, and a history of ASHD. A medical consult, dated 2/9/1988, from John M. Colby, M.D. indicates the employee suffered from a stroke. Medical documentation from Western Baptist Hospital in Paducah, KY, dated 2/11/1988, gives a diagnosis of atherosclerotic cardiovascular disease, status post right middle cerebral artery thrombosis with left hemiparesis, coronary artery disease and peripheral vascular disease.

Smoking History: In the occupational history questionnaire, the claimant stated the employee smoked 4-5 packs of cigarettes/cigars per day since the age of 24 years. She also indicated the employee stopped smoking at the age of 43 years.

In the Occupational History questionnaire, the claimant indicated the employee was exposed to asbestos. She did not know of any other chemicals/toxins the employee was exposed to at PGDP. The claimant also indicated the employee worked in all buildings at the PGDP.

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Statement of Accepted Facts (SOAF)
Revised Format 3/2008

Employee's Name: [redacted]
Claimant Name: [redacted]
File: 4[redacted]4992
Jacksonville, FL

5. **Site Exposure Matrices (SEM) Search Results**

No toxic substances were listed for the labor category of journeyman, wireman. Toxic substance information was listed for electrician. For the health condition of COPD, the employee was exposed to ammonia, asbestos, cadmium and nitrogen dioxide while working as an electrician at the Paducah GDP.

6. **Claims Examiner Information**

a. Submitting District Office: Jacksonville
b. Claims Manager: Patti Purcell
c. Unit designation: H
d. Telephone Number: (904) 357-4795, ext. 74428
e. E-mail address: Perret.Joyce@dol.gov
f. Date of referral:

7. **Other Information.**

Please see DMC's prior opinion regarding COPD. It is marked with a red tab.

8. **Verification of Review**

The referral should be signed by the Claims Examiner's supervisor (a Senior CE or a Claims Manager) indicating that the referral information has been reviewed and meets minimum criteria for submittal.

Joyce Perret, Claims Examiner — **Date** 4/3/2009

Jean Lafontant, Senior Claims Examiner — **Date** APR 0 3 2009

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PAGE NO. 383

Judy Vander Boegh

From: "Saved by Windows Internet Explorer 7"
Sent: Wednesday, November 25, 2009 8:02 AM
Subject: Chapter 2-1000 Exhibit 1

Memorandum from DEEOIC Medical Director
Regarding Causal Relationship Between
Established CBD and Other Respiratory Disorders

Memorandum

Date: 08/25/2005

To: Peter Turcic, Director of DEEOIC, Department of Labor

From: Sylvie I Cohen, MD, MPH

RE: Chronic Pulmonary Diseases

This memo is to address the rationale between the accepted medical condition under part B of the program for Chronic Beryllium Disease (CBD) and its contribution and aggravation of other chronic pulmonary diseases

CBD is considered to be a disease that is involved with the destruction of viable pulmonary tissue that normally aides an individual in the process of gas exchange and blood oxygenation

There are other chronic pulmonary diseases that are involved with lung tissue destruction or replacement that for the purpose of this memo we shall call "Other Chronic Pulmonary Diseases." Diseases that should be considered as members of this set are: asbestosis, silicosis, Chronic Obstructive Pulmonary Disease (COPD), emphysema, and pulmonary fibrosis

Since both CBD and Other Chronic Pulmonary Diseases share in the destruction and or replacement of viable lung tissue, it can be concluded that the presence of CBD contributed or aggravated one of the illnesses named in the list of Other Chronic Pulmonary Diseases which led to an individual's death

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*** FAX TX REPORT ***
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TRANSMISSION OK

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RESULT	OK

COMMONWEALTH ENVIRONMENTAL SERVICES, LLC
A Small, Service-Disabled Veteran Owned Company

Fax Cover Sheet

To:	Hilda Solis & Mr. Bibeault	From:	Gary S. Vander Boegh
Fax:	(904) 357-4704 (202) 693-6111	Date:	11-23-10
Phone:		Pages:	___Pages including the Cover Sheet
Re:	[redacted]	CC:	Attention Jim Bibeault and David Miller

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TRANSACTION OK	912026936111
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PGS.	33
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